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Review

Mechanisms and Clinical Applications of Cooling Interventions for Sleep

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Abstract

Thermoregulation is essential for sleep, with the nocturnal core body temperature decline triggering sleep onset and modulating sleep architecture. Disruptions to this thermal balance cause fragmented sleep and reduced sleep quality. Preliminary evidence suggests cooling may reduce hyperarousal, shorten sleep onset latency, and improve sleep continuity through multiple mechanisms, including enhanced distal-proximal skin temperature gradient, parasympathetic activation, and hypothalamic-pituitary-adrenal axis modulation. However, most studies are small (n<30), short-term (1-4 weeks), and lack standardized protocols or long-term follow-up. This narrative review evaluates evidence on cooling mechanisms (thermoregulatory and stress-related) and evaluates environmental and targeted cooling approaches (mattress systems, forehead devices) for insomnia, anxiety, post-traumatic stress disorder, and migraine. Across conditions, cooling shows promise for reducing hyperarousal and improving sleep outcomes, though evidence quality varies substantially. Critical research gaps include the absence of trials combining cooling with behavioral treatments such as Cognitive Behavioral Therapy for Insomnia, no trials in some clinical populations, limited biomarker-guided personalization, and uncertain long-term effects. Rigorous trials with active controls, objective endpoints, phenotypic stratification, and long-term follow-up are needed to establish cooling as an evidence-based intervention.

Keywords: cooling interventions; thermoregulation; sleep onset latency; stress responses; cognitive behavioral therapy of insomnia (CBT-I)

1. Introduction

Thermoregulation plays a central role in both sleep initiation and maintenance. As part of the circadian rhythm, core body temperature naturally declines in the evening, enabling the transition into slow-wave sleep (SWS) that supports memory consolidation, physical recovery, and immune function [1]. This temperature drop is facilitated by peripheral vasodilation, which increases heat loss through the skin and signals the brain that it is time to initiate sleep. The distal-proximal skin temperature gradient (DPG)—the difference between distal extremity temperatures (hands, feet) and proximal body temperatures (trunk)—is a strong predictor of sleep onset latency, with higher DPG values (reflecting greater peripheral heat dissipation) associated with shorter sleep latencies [2]. Disturbances in these intrinsic thermoregulatory mechanisms, along with external environmental mismatches such as excessively warm bedroom temperatures, are associated with fragmented sleep, insomnia, and overall reduced sleep quality [3]. People with insomnia and older adults who have less efficient temperature regulation are especially sensitive to these disturbances [4]. Therefore, approaches that address these thermoregulatory challenges and correct environmental mismatches are essential for improving sleep onset, maintaining deeper stages of sleep, and ensuring more stable, restorative sleep.

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Recent research highlights that cooling interventions, including environmental adjustments and device-based cooling, may improve thermoregulatory efficiency and can enhance sleep onset and overall sleep quality while helping reduce comorbid symptoms such as insomnia, anxiety, and migraines [5]. By potentially reducing cerebral cortical metabolism, lowering peripheral and core body temperature, and reducing hyperarousal through parasympathetic activation [6], sleep-cooling interventions may support the thermoregulatory processes necessary for both initiating and sustaining sleep. Evidence suggests that tools such as forehead coolers or cooling mattresses can enhance heat dissipation and may improve sleep onset and sleep maintenance in some individuals with insomnia [7,8]. Additionally, cooling is also beneficial for individuals with anxiety, as it activates the parasympathetic pathways to counteract anxiety-related sympathetic arousal and cortisol elevation, thereby reducing physiological activation and supporting both improved sleep and decreased anxiety symptoms [9]. Moreover, because sleep deprivation is strongly associated with increased migraine frequency and severity, forehead cooling has also been shown to reduce migraine intensity and enhance sleep quality [10]. Cooling devices therefore have the potential to provide dual benefits by optimizing thermoregulation and modulating neurovascular and autonomic activity, leading to better sleep quality and reductions in associated symptoms such as pain, anxiety, and hyperarousal.

While early clinical studies suggest that cooling interventions such as environmental cooling and forehead-based devices may improve sleep, the existing evidence is limited by wide variability in study designs, outcome measures, and participant characteristics. This variability makes it difficult to draw clear conclusions about who benefits most, which mechanisms are most relevant, and how cooling should be implemented in practice. Most existing evidence comes from short-term studies (typically 1-4 weeks) in controlled settings, with limited data on long-term effects, optimal dosing, or comparisons between different cooling modalities. In this narrative review, we synthesize current knowledge on the physiological mechanisms through which cooling influences sleep, examine its role in regulating stress and autonomic activity, and evaluate its emerging clinical applications across insomnia, anxiety, post-traumatic stress disorder, and migraine populations. By consolidating the fragmented evidence on cooling, this review aims to clarify the underlying mechanisms, highlight areas of clinical promise, and outline priorities for future research and implementation.

2. Mechanisms of Cooling on Sleep

2.1. Roles of Thermoregulation on Sleep Architecture

Thermoregulation is an essential biological system supporting sleep, driven by circadian timing, core body temperature rhythms, and environmental conditions that shape sleep initiation, depth, and continuity [11]. Under normal circadian regulation, the core body temperature peaks in the late afternoon and gradually declines by about 1 °C in the evening, reducing metabolic rate and neuronal activity, promoting drowsiness, and preparing for sleep onset [11]. This decline in core body temperature is mediated by the suprachiasmatic nucleus (SCN), the master regulator for circadian rhythms; melatonin release, which itself exerts a direct hypothermic effect; and peripheral vasodilation that increases the DPG, a strong predictor of sleep onset latency [2,12]. Ambient temperature also influences the quality and architecture of sleep. The thermoneutral zone refers to the ambient temperature range in which the body can maintain thermal balance without activating heat-gain (e.g., shivering) or heat-loss (e.g., sweating) mechanisms. Because the thermoneutral zone is narrower during sleep than wakefulness, even modest deviations are sufficient to provoke arousal and fragment sleep [11,13,14]. Studies have shown that moderately cool ambient temperatures (approximately 18-22 °C for most adults, though optimal ranges vary by age, bedding, and individual factors) support the nocturnal decline in core body temperature, enhance heat dispersion, and stabilize both slow-wave sleep (SWS) and rapid eye movement (REM) sleep—stages critical for memory consolidation, emotional regulation, and physiological restoration [15]. In contrast, warmer environments (> 32 °C) decrease total sleep time, supress SWS, and increase night awakenings [5,16].

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REM sleep is particularly sensitive to ambient temperature, because thermoregulatory responses such as sweating and shivering are disabled during this stage of sleep, forcing the body to rely almost solely on external conditions for thermal stability [17]. Together, disruptions in these thermoregulatory processes are associated with delayed sleep onset and heightened hyperarousal, a core feature of insomnia [18]. Therefore, targeted cooling strategies may restore the heat-loss mechanisms and reduce hyperarousal, as discussed in the sections below.

2.2. Mechanisms of Cooling on Stress and Hyperarousal

2.2.1. Vagus Nerve Activation and Parasympathetic Regulation

The relationship between thermoregulation and sleep extends into the core physiology of stress and arousal regulation. Stress responses are mediated through tightly linked autonomic pathways, where sympathetic overactivation and reduced parasympathetic tone contribute to the heightened arousal seen in insomnia and other stress-related sleep disturbances [19]. Cooling interventions modulate autonomic stress pathways by stimulating peripheral thermoreceptors (cold-sensitive nerve fibres), that send signals to brain regions controlling autonomic balance. These inputs can then activate reflex circuits that increase vagal efferent activity and shift the body toward parasympathetic dominance, helping counteract hyperarousal [20]. Cooling-induced vagal activation manifests as increased high-frequency heart rate variability (HF-HRV), ain indirect index of parasympathetic tone, reduced heart rate, decreased blood pressure, and lower subjective stress levels [20,21]. Clinical studies have shown that targeted forehead or face cooling increases HF-HRV and reduces sympathetic drive, creating a physiological state conducive to sleep initiation and counteracting the hyperarousal common in insomnia and stress-related disorders [21,22]. The trigeminal nerve, which densely innervates facial and forehead regions, plays a particularly important role in mediating these autonomic responses to targeted cooling of the face or forehead [21].

2.2.2. Cortisol Modulation and Hyperarousal Control

Cooling influences neuroendocrine stress regulation through its effects on the hypothalamic-pituitary-adrenal (HPA) axis. Elevated evening cortisol—a downstream marker of heightened HPA-axis activation and a hallmark of physiological hyperarousal—has been consistently associated with delayed sleep onset, fragmented sleep, and diminished slow-wave sleep, particularly in individuals with insomnia [23]. The relationship between cooling and cortisol is dose-dependent and context-specific. Mild, targeted cooling delivered at sleep-promoting temperatures (e.g., forehead cooling at 14-16 °C) may reduce evening cortisol in individuals with insomnia and hyperarousal [7,24], likely through parasympathetic activation and reduced sympathetic drive [9]. In contrast, intense cold exposures (e.g., cold-pressor tests, whole-body cold-water immersion below 15 °C) trigger acute stress responses with increased cortisol secretion [25]. Whole-body cryotherapy at -110 °C to -160 °C represents a distinct intervention that, despite its extreme temperatures, has been associated with reductions in depression and anxiety symptoms in RCTs. However, its effects on cortisol are complex and may involve adaptive stress responses and anti-inflammatory pathways distinct from thermal comfort mechanisms [26,27]. In contrast to these cold-stress conditions, bedtime cooling that supports normal thermoregulation tends to lower evening cortisol in individuals with hyperarousal or insomnia, although these findings largely come from small clinical studies and mechanistic physiology research rather than large RCTs [24]. Thus, the impact of cooling on HPA-axis activity depends critically on intensity, duration, and individual state: therapeutic cooling interventions designed to promote sleep may help normalize elevated evening cortisol in individuals with insomnia and support smoother transitions into restorative sleep stages, whereas cold exposures sufficiently intense to be perceived as stressful may acutely elevate cortisol through stress response pathways [28]. Together, these autonomic and endocrine pathways provide a mechanistic basis for the therapeutic potential of cooling in reducing stress-driven sleep disturbances.

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Table 1. Mechanisms of cooling on sleep, arousal, and stress response.

Mechanism	Physiological Pathway	Effect on Sleep	Effect on Stress / Cortisol	Key Supporting Evidence
Nocturnal Core Body Temperature Decline	Circadian SCN-driven thermoregulation; evening drop ~1 °C lowers metabolic activity	Facilitates sleep initiation and reduces sleep latency	Indirectly reduces HPA activation by lowering arousal load	Natural CBT decline linked to sleep onset timing; impaired drops delay sleep [11,12]
Distal-Proximal Skin Temperature Gradient (DPG) Increase	Peripheral vasodilation disperses heat via hands/feet	Higher DPG strongly predicts faster sleep onset [2]	Thermal unloading reduces sympathetic tone	DPG is one of the strongest predictors of sleep initiation speed [12]
Autonomic Rebalancing (↑ Parasympathetic / ↓ Sympathetic)	Cooling activates thermoreceptors → vagal efferent pathways → parasympathetic dominance	Lowers sleep onset hyperarousal, increases sleep continuity	Associated with HRV increases and reduced sympathetic output	Forehead/facial cooling improves HF-HRV and decreases arousal (20-22)
HPA-Axis Modulation	Reduced sympathetic drive → dampened CRH/ACTH release	Improves transition into early sleep stages; reduces arousal-driven awakenings	May lower elevated evening cortisol in hyperarousal	Dose-dependent: mild cooling may reduce cortisol in hyperarousal; intense cold stress increases cortisol (23-25, 28)
Reduction of Cognitive-Somatic Hyperarousal	Thermal comfort reduces sensory load; stress vigilance	Quicker sleep transition; fewer cortical micro-arousals	Lower subjective stress, cortisol, HR spikes	Cooling associated with reduced stress markers across insomnia/anxiety groups (23-25, 28)

3. Cooling Intervention Approaches

Cooling interventions target thermoregulatory processes that facilitate heat loss before and during sleep. A range of methods have been developed to enhance these physiological pathways, such as passive heating followed by rapid cooling (the 'warm bath effect', which a meta-analysis found reduced sleep onset latency by approximately 10 minutes [29]), cooling mattresses, and local cooling approaches, including forehead devices [8,11,30]. These techniques are increasingly being explored as adjunctive options for treatment of insomnia, anxiety-related sleep disturbances, and conditions characterized by impaired vasodilation, where thermoregulatory dysfunction contributes to difficulties initiating and maintaining sleep [31]. The following sections outline evidence for the range of current cooling approaches.

3.1. Environmental Cooling

Environmental temperature is one of the most fundamental determinants of sleep quality, with extensive evidence showing that keeping the sleep environment within a thermoneutral range supports normal nocturnal declines in core body temperature, promotes stable sleep architecture, and minimizes circadian and autonomic disruption during sleep [5,11]. Experimental studies support 18-22 °C as the optimal ambient temperature range for sleep in most healthy adults, though individual preferences and optimal temperatures vary with age, sex, bedding insulation, sleepwear, and acclimatization [15]. Cool temperatures within this range are associated with higher delta wave

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activity (deepest sleep stage), shorter sleep onset latency, and longer SWS (for restorative sleep) [13,15]. Randomized and crossover trials provide more granular evidence that even subtle skin warming of merely 0.4 °C, while not altering core temperature, significantly increased SWS and reduced nocturnal awakenings, especially in older adults with age-related sleep fragmentation [32]. A recent systematic review demonstrated that higher ambient temperatures are consistently associated with shorter sleep duration, reduced sleep efficiency, and more awakenings across diverse populations and climates [13]. Additionally, insufficient airflow has been identified as a major driver of heat strain and thermal discomfort, whereas adequate ventilation improves proper oxygen exchange, stabilizes thermal load, and reduces perceived heat stress during sleep [13,33]. Together, these findings indicate that both appropriate ambient temperature and sufficient ventilation are essential for maintaining thermal balance and reducing perceived heat stress during sleep.

Bedding design has become a growing focus in optimizing the sleep environment. A systematic review [34] found that sleepwear and bedding fiber types that more effectively regulate skin temperature and manage moisture are associated with shorter sleep onset latency and improved overall sleep quality. Recent randomized crossover studies similarly showed that sleeping on a temperature-controlled mattress cover for one week significantly enhanced both subjective sleep quality and increased time spent in both deep NREM and REM sleep, although biometric outcomes such as heart rate (HR) and HRV showed variable results [8,35]. Additionally, phase-change materials (PCMs) represent a novel bedding strategy that specifically target thermal buffering, due to their ability to store and release heat as they transition between solid and liquid states, allowing the sleep surface to absorb excess body warmth and release it gradually to help stabilize temperature fluctuations throughout the night [36]. Controlled laboratory studies comparing PCM mattresses to conventional designs have shown that PCM layers can improve thermal comfort and modestly affect sleep microarchitecture [37], though evidence for clinically meaningful sleep improvements remains limited and requires larger trials with standardized protocols. Taken together, these findings demonstrate that environmental cooling—either achieved through optimized ambient temperature, enhanced ventilation, or thermoregulating bedding materials—can meaningfully stabilize nocturnal thermoregulation and support deeper, more consolidated sleep. However, the evidence base remains heterogeneous, and larger, well-controlled trials comparing different cooling strategies, temperature targets, and bedding technologies are needed to establish clear, standardized recommendations and to identify which populations benefit most from tailored thermal interventions.

3.2. Targeted Cooling Devices

Beyond optimizing the overall sleep environment, targeted cooling devices, including wearables, can deliver cooling to specific body regions, modulate arousal, and create localized microclimates that support thermoregulation. For example, an RCT demonstrated that local body cooling to the back and neck improved sleep efficiency, time in deep sleep (N3), subjective thermal comfort, and overall sleep quality [30].

Among targeted cooling approaches, forehead cooling has emerged as a promising modality for insomnia and thermoregulation-related sleep disturbance. The scalp and forehead have dense trigeminal and autonomic-linked sensory innervation, and selective cooling in this region can reduce cortical metabolism and shift autonomic balance toward parasympathetic dominance [38]. In a double-blind RCT of 106 adults with insomnia, forehead cooling at 14-16 °C produced significant improvements versus sham in several secondary sleep onset measures: relative change from baseline in latency to persistent sleep, absolute latency to stage 1 and stage 2 NREM sleep, and minutes of sleep in the first hour [39]. However, the pre-specified primary endpoints—absolute latency to persistent sleep and sleep efficiency—did not reach statistical significance [39], highlighting the importance of standardized outcome measures in future trials. In another crossover RCT in young women during the luteal menstrual phase, head cooling at 25 °C reduced arousal time, increased deep NREM sleep, and enhanced delta power, while also improving subjective sleep comfort, compared to the 35 °C control [40]. Such physiological effects align with pilot neuroimaging evidence

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that forehead cooling reduces frontal cortical metabolism during sleep in individuals with insomnia, addressing the heightened prefrontal activation that characterizes the hyperarousal state [6,39]. A four-week pilot study tested nightly use of a forehead cooling device (open-label) in veterans with chronic insomnia and comorbid medical and psychiatric conditions. Findings showed that 71% of participants reported clinically meaningful improvements in insomnia severity and 42% experienced remission, with concurrent reductions in anxiety and depressive symptoms, and sleep onset latency and wake after sleep onset were also significantly reduced after treatment [7]. However, as an open-label pilot study without a control group, these findings require confirmation in larger, placebo-controlled trials.

Therefore, targeted cooling strategies show potential for reducing hyperarousal and improving sleep parameters. However, larger adequately powered RCTs with active control conditions, standardized devices and protocols (including optimal temperature, duration, and timing of cooling), objective polysomnographic endpoints, long-term follow-up, and assessment of moderators (e.g., baseline DPG, chronotype, age, sex) are needed to define who benefits most and how best to implement these tools in clinical practice.

Table 2. Cooling interventions and their effects on sleep outcomes.

Cooling Category	Implementations	Key Outcomes	Summary of Evidence	Refs
Optimal Ambient Cooling (Thermoneutral Zone Range)	18-22 °C bedroom conditions (varies by age, bedding, individual factors); climate control	Sleep latency, SWS continuity, REM stability	Optimal range per recent systematic reviews; improves delta power, reduces awakenings, supports stable architecture; individual variation exists	[13,15]
Airflow-Based Cooling	Fans, natural ventilation, microclimate air circulation	Thermal comfort, awakenings, perceived rest	Ventilation reduces heat strain and nocturnal disruption; enhances subjective rest even without temperature change	[13,33]
Bedding-Based Thermal Regulation	Thermoregulating fiber bedding; sleepwear; PCM materials; temperature-controlled mattress covers	Sleep latency, SWS/REM depth, continuity	Regulates skin temperature; moisture → ↓ SOL, ↑ deep NREM/REM; PCM shows thermal effects with variable sleep benefits; controlled covers enhance continuity	(8, 34-37)
Forehead/Scalp Cooling (Autonomic-Linked)	Thermal frontal device (14-16 °C); head cooling during luteal phase; multi-week clinical nightly treatment	Sleep latency (secondary measures), nighttime awakenings, delta power	Secondary sleep onset improvements in RCT (primary endpoints NS); delta power increased; open-label trial showed symptom reduction	[7,39,40]

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3.3. Integration of Cooling as Adjunct Therapy with CBT-I

Cooling-based interventions are compatible with established treatments like Cognitive Behavioral Therapy for Insomnia (CBT-I) [41,42]. While CBT-I remains the gold-standard treatment for chronic insomnia, many individuals continue to experience residual symptoms (in 30-40% of individuals with insomnia) related to hyperarousal and impaired thermoregulation after completing treatment [43]. Cooling interventions may address these physiological barriers by facilitating peripheral heat dissipation (increasing the DPG), potentially reducing elevated core body temperature in hyperaroused individuals, and promoting parasympathetic activation, aligning the mechanisms with CBT-I components such as relaxation training and stimulus control [42,44]. Integrating cooling devices (such as forehead coolers or cooling mattresses) into CBT-I protocols may help shorten sleep onset latency, reduce nocturnal awakenings, and support adherence to behavioral recommendations. Populations with impaired thermoregulation, such as older adults, women experiencing menopause, shift workers, and individuals with cardiometabolic disease, may be particularly responsive to such strategies, as they address underlying physiological vulnerabilities that sustain insomnia [45–47]. Overall, cooling interventions act on mechanisms of hyperarousal and disrupted thermoregulation, positioning them as promising non-pharmacological tools that can be integrated with CBT-I to enhance treatment outcomes. Larger, long-term RCTs will be crucial for defining efficacy across patient subgroups and clinical settings who are most likely to benefit from cooling-based adjuncts to CBT-I. Despite this mechanistic promise, RCT evidence for cooling as an adjunct to CBT-I is currently lacking. No published RCTs have evaluated the combination of cooling interventions with CBT-I compared to CBT-I alone. The rationale for integration is based on complementary mechanisms rather than empirical evidence of synergistic effects. Future research should prioritize such combination trials, particularly in treatment-resistant insomnia where residual hyperarousal persists despite CBT-I.

4. Clinical Applications and Therapeutic Relevance

4.1. Insomnia

Insomnia, among the most prevalent sleep disorders globally, involves physiological hyperarousal (elevated core body temperature, sympathetic activation, heightened evening cortisol), and thermoregulatory impairments (reduced distal vasodilation, blunted evening temperature decline) that collectively delay sleep onset and fragment sleep [48,49]. This dysregulation forms a self-perpetuating cycle in which impaired heat loss reinforces hyperarousal, while ongoing sleep disruption further destabilizes circadian and thermoregulatory balance [5].

Cooling interventions target these thermoregulatory and arousal abnormalities, though clinical evidence remains preliminary. As described above, forehead cooling has been associated with faster transitions into stages 1 and 2 NREM sleep and greater early-sleep time in a double-blind RCT of adults with insomnia [39], as well as reduced insomnia severity, nocturnal hyperarousal, and mood symptoms in a 4-week trial of veterans [7]. Additionally, forehead cooling was associated with lowered frontal cortical metabolism in individuals with primary insomnia in preliminary neuroimaging studies, consistent with reduced cognitive/emotional overactivation that impedes sleep onset [6].

Taken together, these findings indicate that cooling interventions may modulate the neurophysiological substrates that maintain insomnia, making them a biologically targeted, non-pharmacological tool for insomnia. However, critical gaps remain: (1) lack of standardized protocols for temperature, duration, timing, and device specifications; (2) absence of long-term efficacy and safety data; (3) uncertainty about which patient subgroups benefit most; and (4) no comparisons of different cooling modalities. Future research should prioritize adequately powered RCTs with objective sleep outcomes, phenotypic stratification based on biomarkers (e.g., baseline DPG, evening cortisol, HRV), and pragmatic trials assessing real-world effectiveness and cost-effectiveness.

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4.2. Generalized Anxiety Disorder (GAD)

Autonomic dysregulation is a core physiological feature of GAD, characterized by excessive sympathetic activation, heightened HPA-axis activity, and elevated cortisol levels, amplifying stress responses and contributing to persistent physiological tension [50]. Individuals with GAD frequently show reduced HRV and impaired vagal tone, mirroring the physiological hyperarousal seen in insomnia, which could explain the strong comorbidity between anxiety and sleep disturbance [51]. Given this shared pathophysiology, cooling-based interventions may counteract these processes by reducing peripheral and central temperature and shifting autonomic balance toward parasympathetic dominance. Direct evidence for cooling interventions in primary GAD is currently lacking, as no published RCTs have evaluated cooling specifically in GAD populations. In the absence of GAD-specific trials, indirect evidence from cold stimulation studies in healthy volunteers and mixed psychiatric populations suggests potential anxiolytic effects. Studies of cold stimulation applied to the face or neck (the "Cold Face Test") showed robust activation of vagal pathways, with reductions in heart rate, increases in HRV, and attenuated cortisol responses to acute psychosocial stress [9,20]. Whole-body cryotherapy, the brief exposure to extremely cold air in a chamber cooled with liquid nitrogen, has also been evaluated as an adjunct treatment in mood and anxiety disorders. RCTs in individuals with depression and anxiety disorders reported significant reductions in anxiety when cryotherapy was added to standard pharmacotherapy [26], and a systematic review and meta-analysis also suggested medium-to-large effects of whole-body cryotherapy on improving depression and anxiety symptoms [27]. However, these whole-body cryotherapy studies (involving exposure to -110 °C to -160 °C for 2-3 minutes) represent a distinct intervention from the mild cooling approaches (18-22 °C environmental cooling or forehead cooling at 14-16 °C) discussed elsewhere in this review, operating through different physiological mechanisms and requiring specialized facilities. Cooling may address both anxiety symptoms and sleep disturbance through autonomic modulation. While the mechanism-based rationale is compelling, GAD-specific RCTs are needed before cooling can be recommended for anxiety management, with particular attention to whether mild cooling approaches (e.g., forehead devices) or intense cold exposures (e.g., whole-body cryotherapy) are more appropriate for different clinical presentations.

4.3. Post-Traumatic Stress Disorder (PTSD)

PTSD is characterized by persistent physiological hyperarousal, marked sympathetic dominance, and impaired autonomic regulation, contributing to severe sleep disturbances, including nightmares, frequent awakenings, and fragmented sleep [52]. This chronic elevation in sympathetic activity disrupts normal thermoregulation and increases cortical hyperexcitability, making it difficult for individuals to initiate and maintain restorative sleep [53]. Like other stress-related sleep disorders, the interplay between hyperarousal and dysregulated temperature control forms a self-reinforcing cycle that worsens both sleep quality and trauma-related symptoms [54].

Forehead or scalp cooling may reduce sympathetic outflow, enhance parasympathetic tone, and attenuate cortical hyperactivation via trigeminal and brainstem pathways [21]. Evidence for cooling interventions in PTSD is extremely limited, with no published RCTs evaluating cooling devices in PTSD populations. Early clinical findings support cooling as a biologically informed adjunct to established PTSD treatments such as CBT-I and trauma-focused psychotherapies. In a 4-week open-label pilot study (n=24) of veterans with chronic insomnia and comorbid psychiatric conditions (including but not limited to PTSD), nightly use of a forehead cooling device produced large reductions in insomnia severity, shorter sleep onset latency and wake after sleep onset, and concurrent improvements in anxiety and depressive symptoms [7]. However, this pilot study lacked a control group and did not separately analyze individuals with PTSD, limiting conclusions about PTSD-specific effects. Nevertheless, these findings support the feasibility of cranial cooling in trauma-exposed populations. As research advances, PTSD-specific RCTs with standardized cooling protocols and polysomnographic and symptom outcomes will be essential for clarifying how cooling

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interventions can be integrated into comprehensive treatment strategies for PTSD and co-occurring insomnia.

4.4. Migraine

Migraine is a neurological disorder closely linked to sleep disturbances and impaired thermoregulatory function [55]. Clinical and epidemiological studies show bidirectional associations, where poor sleep can trigger migraines, and migraines disrupt sleep continuity and aggravate sleep-related pathology [56]. These interactions appear to stem from shared physiological mechanisms involving disruptions in thermal homeostasis, heightened stress reactivity, and abnormal central and peripheral neuronal excitability [57]. This overlap highlights the potential value of cooling interventions, which may simultaneously improve sleep and attenuate migraine symptoms [58].

Preliminary evidence supports cooling for acute migraine relief. A systematic review and meta-analysis of cold interventions for acute migraine (14 studies, n=427) found that various cold applications (cold-gel headband, cold-gel cap, intraoral cooling, and cold wraps) significantly reduced pain intensity in the short-term (typically within 30-60 minutes), though effects on long-term migraine frequency and disability were less consistent and quality of evidence was limited by small sample sizes and methodological heterogeneity [58]. In a crossover RCT, targeted neck cooling using a neoprene wrap with ice packs was associated with a greater reduction in pain intensity at 30 minutes compared with control placement, demonstrating that localized cooling of the carotid and vertebral regions can attenuate migraine pain [59]. A prospective trial also reported that intranasal evaporative cooling, which delivers coolant-conditioned air through a nasal catheter, was associated with improvements in headache severity and symptoms associated with migraine that were sustained for 24 hours [60]. A more recent randomized, double-blind multicenter study found that lower-flow transnasal cooling was associated with significant pain relief at 2 hours and was well tolerated for acute migraine treatment [61]. Another RCT study found that combining cold application with progressive muscle relaxation reduced pain intensity, migraine frequency, disability, and quality of life at 4-weeks compared to baseline, suggesting cold intervention as a useful adjunct strategy for migraine management [10]. Notably, most migraine-cooling trials focus on acute pain relief rather than sleep outcomes, and no studies have specifically examined whether cooling interventions improve sleep architecture or reduce nocturnal migraine-related awakenings in individuals with comorbid migraine and insomnia. This represents a critical gap, as the sleep-migraine link suggests potential for dual benefits if cooling were studied with sleep as a primary endpoint.

Table 3. Clinical applications of cooling-based interventions across disorders.

Condition	Targeted Mechanism	Intervention Type	Evidence Strength; Key Findings	Refs
Insomnia	↓ Hyperarousal (↓ core temp & ↓ cortical metabolism) → ↑ heat loss, ↓ latency → improved continuity	Forehead/scalp cooling devices; nocturnal targeted cooling	**Limited evidence** –RCT shows improved secondary latency (N1/N2 latency, first-hour sleep), primary endpoints NS; open-label pilot shows symptom improvements; preliminary neuroimaging supports metabolic mechanism but short-term data only	[5–7,39,48,49]
Generalized Anxiety Disorder (GAD)	↓ Sympathetic tone, ↑ vagal activation (↑ HRV), ↓ cortisol stress	Cold-face/neck stimulation; whole-body cryotherapy (-110 to -160 °C)	**Limited/indirect evidence**—No GAD-specific trials; RCTs show increased HRV; reduced cortisol; reduced anxiety symptoms; sleep outcomes not directly tested yet	[9,20,26,27,50,51]

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Post-Traumatic Stress Disorder (PTSD)	↓ Sympathetic dominance → ↓ cortical hyperactivation → improved sleep initiation and maintenance	Forehead cooling, scalp/head devices	**Very limited**—One open-label pilot (n=24, mixed psychiatric conditions) showed improvements; no PTSD-specific controlled trials	[7,21,52–54]
Migraine	↓ Neuronal excitability and thermal stabilization → relief of acute pain	Cold wraps, head-cooling bands, transnasal evaporative cooling	**Moderate for pain, minimal for sleep**—Meta-analysis shows acute pain reduction; sleep effects unstudied	[10,55–61]

5. Conclusions

Cooling interventions show mechanistic plausibility through multiple pathways—core temperature reduction, enhanced peripheral heat dissipation, and parasympathetic activation—that target hyperarousal and thermoregulatory dysfunction in insomnia, anxiety, PTSD, and migraine. Preliminary evidence suggests that cooling may improve sleep initiation in some populations and modulate stress physiology by lowering cortisol and restoring autonomic balance. As research and device technology advance, cooling may move from an experimental supplement to a practical component of comprehensive sleep and stress-management strategies. Combining cooling with behavioral approaches such as CBT-I with optimized bedroom environments or with targeted devices like forehead coolers may offer synergistic benefits and provide an environmentally responsive, non-pharmacological option for addressing sleep disturbances.

Despite promising preliminary findings, substantial evidence gaps limit clinical translation. Most trials are short-term (1-4 weeks), small (n<30), and lack real-world validation. Critical gaps, including the absence of standardized protocols, direct comparisons of cooling modalities, combination trials with CBT-I, and phenotypic stratification to identify responders (e.g., based on baseline DPG, cortisol levels), should inform future studies. Future priorities include: (1) adequately powered, multi-site RCTs with active controls and objective endpoints; (2) long-term follow-up (≥6 months); (3) head-to-head trials comparing modalities; (4) combination trials (cooling plus CBT-I versus CBT-I alone); (5) biomarker-guided stratification; (6) cost-effectiveness analyses; and (7) studies across diverse populations and settings to establish generalizability and guide evidence-based implementation. Addressing these gaps will clarify whether cooling can serve as a scalable, non-pharmacological adjunct within comprehensive sleep and stress management strategies.

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