Review

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Review

Sleep Onset Anxiety: Mechanisms and Evidence-Based Approaches in Clinical Practice

Running Title: Evidence-Based Interventions for Sleep Onset Anxiety

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Abstract

Sleep-onset anxiety is a transdiagnostic contributor to difficulty initiating sleep that is maintained through interconnected cognitive, physiological, and neurobiological processes. This narrative review synthesizes current evidence on mechanisms and behavioral interventions for sleep-onset anxiety, highlighting pre-sleep worry, rumination, conditioned arousal, autonomic dysregulation, and brain network alterations as key mechanisms. Evidence-based interventions are reviewed, including cognitive behavioral therapy for insomnia (CBT-I), mindfulness and acceptance-based approaches, relaxation strategies, and environmental optimization. Clinical considerations are examined for individuals with comorbid anxiety disorders, across developmental stages, and across diverse cultural contexts. Digital delivery formats are reviewed as scalable approaches to extend treatment access. Much existing evidence derives from broader insomnia populations rather than sleep-onset anxiety as a distinct construct, limiting mechanistic specificity. Future research directions include mechanistic trials integrating objective biomarkers with clinical outcomes, component optimization studies, and scalable implementation approaches. This review provides an integrative framework linking mechanisms to evidence-based interventions for sleep-onset anxiety across diverse clinical populations and settings.

Keywords: sleep onset anxiety; cognitive behavioral therapy for insomnia (CBT-I); mindfulness; somatic hyperarousal; relaxation techniques; environmental modifications

1. Introduction

Anxiety-related sleep disturbances are common across the lifespan [1], with insomnia disorder affecting approximately 12-16% of the general population [2]. Sleep onset anxiety, a heightened state of worry and cognitive arousal occurring during the transition to sleep, functions as a transdiagnostic symptom cluster that increases vulnerability to insomnia rather than constituting a standalone disorder [3]. Cognitive models of insomnia and emerging data linking circadian factors to affective dysregulation suggest how pre-sleep anxiety amplifies threat monitoring and hyperarousal, thereby delaying sleep initiation [3,4]. This heightened alertness disrupts normal sleep initiation, contributing to sleep onset insomnia. The condition is often accompanied by mental distress and physical tension, further compounding sleep difficulty [3].

Sleep onset anxiety is challenging to operationalize given its overlap with general insomnia symptoms. Moreover, sleep disturbances caused by pre-sleep worry extend beyond nighttime distress to broader health consequences, such as mood disorders, emotional dysregulation, and cognitive impairment [5]. Emerging evidence supports insomnia symptoms, including sleep-onset difficulties, as transdiagnostic phenomena maintaining psychiatric disorders through shared cognitive and physiological mechanisms [6,7]. Moreover, insomnia is associated with weakened

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immunity, elevated stress, and reduced quality of life [8], with individuals with chronic anxiety disorders being particularly vulnerable to sleep initiation difficulties. Addressing sleep onset anxiety through integrated psychological and physiological assessment is therefore warranted to optimize insomnia treatment outcomes.

Behavioral sleep medicine interventions target specific cognitive, behavioral, and physiological contributors to pre-sleep arousal. These include cognitive behavioral therapy, relaxation-based strategies, mindfulness-based interventions, and environmental modifications that reduce arousal and support more efficient sleep initiation [9]. However, despite the substantial evidence for behavioral approaches, mechanistic understanding of how individual components exert their effects, such as stimulus control, sleep restriction, or mindfulness, remains incomplete. In particular, the pathways linking intervention components to neurobiological targets, including autonomic regulation, brain network dynamics, and hypothalamic-pituitary-adrenal (HPA) axis function, require further elucidation. This narrative review synthesizes current evidence on mechanisms underlying sleep-onset anxiety and corresponding evidence-based interventions. By integrating cognitive, physiological, and neurobiological perspectives with intervention research, this review provides a mechanistic framework to guide assessment and treatment selection across diverse clinical populations. The following section outlines cognitive, physiological and neurobiological mechanisms that maintain sleep onset anxiety and serve as targets for behavioral interventions.

2. Mechanisms of Sleep Onset Anxiety

2.1. Cognitive Mechanisms

Cognitive processes play a major role in the onset and maintenance of sleep onset anxiety, particularly through patterns of rumination and worry [10]. Although both involve repetitive thought, rumination and worry represent distinct cognitive processes in insomnia: worry is future-oriented and characterized by verbal-linguistic processes about anticipated threats, whereas rumination is past-oriented and involves repetitive focus on negative emotions, their causes, and consequences [11]. Many individuals experience sleep onset anxiety as a result of worry or rumination, which creates a barrier to relaxation, preventing the natural transition into sleep [12]. Catastrophic thinking, where one envisions the worst-case scenario for sleep problems, further contributes to anxiety and frustration, perpetuating sleeplessness [4]. Collectively, these maladaptive cognitive patterns exacerbate pre-sleep arousal, making it harder to initiate sleep. Furthermore, anxiety is aggravated by fears related to sleep – worries about insufficient sleep and its consequences (e.g., weak work performance or health-related problems). This fear of insomnia may initiate a self-perpetuating cycle wherein greater difficulty initiating sleep increases the likelihood that the individual will worry about the consequences of sleep deprivation.

Additionally, metacognitive processes, defined as beliefs about thoughts and strategies for controlling mental activity, play an important role in maintaining sleep onset anxiety [13,14]. These include beliefs about the uncontrollability and danger of sleep-related thoughts, the perceived utility of worry (e.g., “worrying helps me prepare”), and maladaptive thought-control strategies such as thought suppression. Metacognitive beliefs can amplify the emotional salience of primary sleep-related cognitions, increase attentional bias toward sleep-related threats, and paradoxically intensify pre-sleep arousal when individuals attempt to suppress unwanted thoughts [15,16]. These metacognitive processes represent a secondary arousal system that perpetuates insomnia by influencing how individuals relate to their sleep-related thoughts, beyond the content of the thoughts themselves.

Sleep-related safety behaviors are overt or covert strategies employed to prevent feared outcomes related to poor sleep, such as spending excessive time in bed trying to force sleep, using electronic devices for distraction, or napping during the day to compensate for lost rest [17]. Although these behaviors are intended to reduce distress or promote sleep, they maintain insomnia by preventing disconfirmation of exaggerated worries about sleep and often disrupt sleep drive and circadian rhythms [18]. Importantly, research indicates that the perceived utility (i.e., the belief that

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these behaviors are necessary) rather than just the frequency of safety behaviors, predicts insomnia severity, suggesting that functional relationship individuals have with these behaviors is critical to understanding their maintaining role [18]. These maladaptive coping strategies maintain both pre-sleep arousal and irregular sleep patterns, thereby exacerbating sleep onset anxiety.

2.2. Somatic Hyperarousal and Neurobiological Dysregulation

Physiological responses also contribute to sleep onset anxiety. Somatic hyperarousal is characterized by excessive physical tension and heightened autonomic activation, leading to symptoms such as increased heart rate, muscle contraction, and elevated cortisol release, a hormone associated with stress [19]. These physiological responses reflect a "fight-or-flight" response activated by the sympathetic nervous system, which, although adaptive in life threatening situations, is counter-productive for sleep. Heightened sympathetic activity delays the parasympathetic dominance required for sleep initiation, contributing to restlessness and delayed sleep onset [20,21].

The HPA axis is a major constituent of the body's stress response and plays a critical role in this process. HPA axis dysregulation has been associated with elevated nighttime cortisol secretion, increasing alertness and interfering with the sleep cycle [22]. Meta-analytic evidence confirms that individuals with insomnia show moderately increased cortisol levels, with the greatest elevations observed in the evening and first half of the night, precisely when sleep initiation should occur [23,24]. Notably, this elevation relates positively to the degree of objective sleep disturbance, consistent with a disorder of 24-hour hyperarousal rather than simply sleep loss [25]. Recent evidence also demonstrates elevated levels of corticotropin-releasing hormone (CRH) and copeptin (a marker of arginine vasopressin activity) in individuals with chronic insomnia, reflecting both hypothalamic activation and sympathetic nerve excitation [26].

Cognitive arousal can further exacerbate HPA dysfunction, impeding the body's ability to relax prior to sleep [27]. The interaction between HPA axis activity and cognitive arousal is bidirectional: rising cortisol amplifies cognitive arousal, and heightened cognitive arousal in turn increases cortisol levels, creating a self-perpetuating cycle of anxiety and sleep disturbance. This bidirectional relationship has been demonstrated in multiple studies, with perseverative cognitive processes such as worry and rumination linked to extended cortisol activation [28]. Targeting this cycle represents a key therapeutic opportunity for interventions aimed at reducing sleep onset anxiety.

2.3. Interaction between Cognitive and Physiological Arousal

Cognitive and physiological arousal are tightly interconnected, forming a self-reinforcing cycle that contributes to sleep onset anxiety. Cognitive arousal, including worry and rumination, elevates heart rate, increases muscle tension, and activates stress pathways, thereby triggering physiological hyperarousal [29]. In turn, heightened physiological sensations (e.g., racing heart, restlessness) can amplify anxious thoughts, prompting further rumination and reinforcing the perceived inability to fall asleep [4]. This bidirectional relationship sustains wakefulness through mutually reinforcing mental and bodily tension.

Effective treatment therefore requires approaches that target both systems. Cognitive-behavioral strategies such as cognitive restructuring reduce maladaptive thinking patterns and pre-sleep worry, while relaxation-based interventions, such as progressive muscle relaxation, diaphragmatic breathing, and mindfulness, help downregulate sympathetic activity and promote parasympathetic dominance [30]. By calming both mind and body, these approaches disrupt the cycle and facilitate smoother sleep initiation.

Emerging research suggests biomarkers that may guide more precise interventions. Autonomic indicators such as reduced heart-rate variability (HRV) have also been found to relate to impaired stress regulation during bedtime, where lower pre-sleep HRV is associated with greater cognitive and somatic arousal, longer sleep-onset latency, and poorer overall sleep quality in both insomnia and high-stress populations [31,32]. Functional connectivity between the amygdala and prefrontal cortex has been associated with heightened emotional reactivity and rumination during bedtime [33].

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Recent neuroimaging studies also implicate altered default mode network (DMN)-salience network coupling and limbic-prefrontal hyperconnectivity during periods of sleep anticipation and rumination [34,35]. Specifically, individuals with insomnia demonstrate increased connectivity within the DMN, a network associated with self-referential processing and rumination, which may prevent the deactivation necessary for sleep onset [36]. Studies suggest associations between DMN connectivity and subsequent sleep quality as measured by polysomnography, with greater DMN-cortical connectivity associated with better sleep efficiency and total sleep time [37]. Additionally, altered effective connectivity between the DMN, executive control network, and salience network has been identified as a neural correlate of the hyperarousal state in chronic insomnia, with disrupted "switching" between these networks preventing successful sleep initiation [38].

These mechanistic markers help explain why cognitive and physiological arousal are tightly linked and highlight novel therapeutic targets. Interventions such as HRV biofeedback, real-time neurofeedback targeting DMN activity, and pharmacologic modulation of stress-response pathways are being investigated to alter brain connectivity, autonomic balance, and endocrine rhythms [39]. Preliminary studies using transcranial magnetic stimulation targeting the DMN prior to sleep onset to reduce cortical hyperarousal and facilitate sleep, but clinical efficacy for insomnia has not been established [40]. By intervening directly on these biological systems, future therapies may more effectively interrupt the anxiety-arousal cycle and promote timely transition into sleep. **Table 1** summarizes cognitive and physiological processes implicated in sleep onset anxiety.

Table 1. Summary of Cognitive and Physiological Mechanisms.

Mechanism		Description	Impact on Sleep	**Interventions/Targets**	Refs
Cognitive	Rumination	Repetitive negative thinking about past events or emotions.	Sustains cognitive arousal and prevents relaxation, delaying sleep initiation.	Cognitive restructuring, mindfulness, scheduling "worry time."	[10,12]
	Worry/ Catastrophizing	Anticipation of negative outcomes or excessive concern about sleep and its consequences.	Creates anticipatory arousal and heightens anxiety, preventing sleep onset.	CBT-I cognitive restructuring, paradoxical intention, psychoeducation.	[3,10]
	Metacognitive Processes	Beliefs about thoughts (e.g., uncontrollability of worry) and maladaptive thought-control strategies.	Amplifies emotional valence of sleep-related thoughts; increases attentional bias and arousal.	Mindfulness, metacognitive therapy, acceptance-based approaches	[13,14]

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	Safety Behaviors	Overt/covert strategies to prevent feared sleep outcomes (e.g., excessive time in bed, device use).	Prevents disconfirmation of sleep-related fears; disrupts sleep drive and circadian rhythms.	Stimulus control, behavioral experiments, reducing perceived utility of behaviors.	[4,18]
Somatic	Somatic Hyperarousal	Heightened sympathetic activation (increased heart rate, muscle tension).	Activates the fight-or-flight response and prevents parasympathetic dominance for sleep onset.	Progressive muscle relaxation, diaphragmatic breathing, biofeedback, mindfulness.	[20]
	HPA Axis Dysregulation	Abnormal cortisol rhythms with inappropriate nighttime secretion and heightened stress reactivity.	Increases alertness and impairs the natural sleep-wake transition.	Stress reduction techniques, behavioral interventions, cortisol regulation strategies.	[24,26]
	Autonomic Dysregulation	Reduced heart-rate variability (HRV) reflecting impaired vagal tone and stress regulation.	Promotes sustained physiological arousal and delays sleep initiation.	HRV biofeedback, slow breathing, mindfulness-based interventions.	(17, 18)
	Neural Network Dysregulation	Altered DMN-salience network coupling; increased amygdala-prefrontal connectivity; failed DMN deactivation.	Sustains rumination and emotional reactivity; prevents cognitive disengagement needed for sleep.	Neurofeedback targeting DMN, mindfulness, CBT-I, transcranial magnetic stimulation.	[34,37,38]

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3. Evidence-Based Interventions

To translate this understanding into practice, the following sections highlight interventions with strong empirical support in clinical populations. Each targets modifiable drivers of pre-sleep anxiety, offering structured strategies to enhance relaxation and improve sleep outcomes.

3.1. Cognitive-Behavioral Therapy for Insomnia (CBT-I)

CBT-I is the established first-line treatment for chronic insomnia and has been applied to address sleep onset anxiety [41,42]. It integrates multiple components, including cognitive restructuring, sleep restriction, stimulus control, and relaxation-based strategies, to target both the cognitive worry loops and physiological hyperarousal that maintain insomnia. Multiple randomized controlled trials (RCTs) across clinical and psychiatric populations demonstrate that CBT-I improves sleep latency and pre-sleep anxiety, with therapeutic gains maintaining beyond 6 months. In meta-analyses, CBT-I yields comparable short-term improvements and more durable long-term outcomes than pharmacotherapy for chronic insomnia [41–43].

3.1.1. Cognitive Restructuring

Cognitive restructuring targets the identification and modification of dysfunctional beliefs about sleep (e.g., catastrophizing consequences of insufficient sleep), replacing them with more balanced, evidence-informed expectations [44]. By reducing maladaptive threat appraisals, cognitive restructuring attenuates anticipatory anxiety and rumination, thereby reducing cognitive arousal and facilitating sleep initiation. Paradoxical intention – a strategy reduced performance pressure around sleep – has shown significant reduction in sleep-onset latency in a meta-analysis [45].

3.1.2. Sleep Restriction

Sleep restriction increases homeostatic sleep pressure by limiting time spent in bed to align with actual sleep duration, thereby increasing sleep efficiency [46]. Over time, this strengthens homeostatic sleep pressure and helps regularize sleep schedule [47]. Meta-analytic evidence supports that standalone sleep restriction therapy improved sleep efficiency and depressive symptoms in adults with insomnia [46], whereas combining sleep restriction with other CBT-I elements such as stimulus control and relaxation strategies increased its effectiveness and reduced both nighttime hyperarousal and conditioned wakefulness in bed [47].

3.1.3. Stimulus Control

Stimulus control directly targets conditioned arousal, a key mechanism of insomnia in which the bed and bedroom environment become cues for wakefulness rather than sleep [41]. By restructuring behavioral contingencies to restrict the bed to sleep and sex, initiate sleep only when drowsy, and maintain consistent wake times, stimulus control re-establishes the bedroom as a discriminative cue for parasympathetic activation and sleep initiation. Environmental cue optimization (light, sound, temperature) complements stimulus control but targets separate circadian and sensory pathways [48]. By aligning sensory inputs with physiological sleep readiness, stimulus control interventions may reduce conditioned hyperarousal and support the neurobiological conducive to sleep onset [48].

Light Intervention

The circadian system is regulated by the intensity and timing of light exposure, which modulates sleep-wake cycle. Systematic reviews and meta-analyses demonstrate that evening exposure to bright or blue-enriched light suppresses melatonin production, delays sleep onset, and reduces overall sleep efficiency by signaling the brain to remain alert, whereas warm, dim lighting and blue-light filters on electronic devices during evening hours minimized circadian disruption [49]. Dimming lights at least

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one hour before bedtime can promote relaxation and facilitate faster sleep onset [50]. Conversely, bright morning light exposure, particularly within the first hour of waking, suppresses melatonin, advances circadian timing, and strengthens a stable sleep-wake rhythm [51]. Light therapy has been supported in clinical settings, where structured light therapy improves sleep alignment in circadian rhythm sleep-wake disorders such as delayed sleep phase, and has demonstrated efficacy in alleviating both mood and sleep symptoms in seasonal affective disorder, especially during months with limited sunlight [52]. Together, these findings underscore light management as a key behavioral consideration to optimize circadian health and sleep quality.

Auditory Intervention

The auditory environment significantly influences sleep quality, with noise exposure linked to delayed sleep onset, more frequent nighttime awakenings, and reduced sleep depth [53]. Individual sensitivity to sound varies considerably: while some individuals are highly sensitive to even minor sounds, others benefit from gentle background noise that masks disturbances [54,55]. Practical strategies such as earplugs, noise-cancelling headphones, and soundproofing can reduce environmental disruptions, especially for those with heightened noise sensitivity [56]. Controlled studies suggest white noise or relaxing soundscapes may improve sleep continuity in some individuals [57,58]. A systematic review has shown that incorporating soothing music into a pre-sleep routine reduced anxiety, slowed heart rate, and shortened sleep latency, leading to deeper and more restorative rest [59]. Collectively, these findings suggest that optimizing the auditory environment can play meaningful, albeit variably supported, roles in supporting healthy sleep.

Thermoregulation

Temperature regulation plays an important role in sleep initiation and maintenance because the body's core temperature naturally decreases in preparation for sleep [60]. Excessive warmth can disrupt this process, causing restlessness, sweating, and difficulty maintaining sleep throughout the night. In contrast, a cool, well-ventilated environment supports efficient thermoregulation and overall sleep quality [61]. Research suggests that many adults experience optimal sleep at bedroom temperatures between 15-20 °C, a range that facilitates the body's natural cooling signals and promotes faster sleep onset [62]. Practical adjustments, such as using fans, air conditioning, or heaters, can help maintain this ideal range across seasons and household environments [63]. Additionally, cooling bedding materials, moisture-wicking sleepwear, and breathable fabrics have also been shown to improve airflow and thermal comfort, reducing nighttime awakenings and enhancing sleep quality in a systematic review [64]. Collectively, these strategies highlight the importance of creating a temperature environment that aligns with the body's physiological sleep cues, which may contribute to deeper, more restorative sleep.

3.1.4. Relaxation Training

Relaxation training in CBT-I includes methods such as progressive muscle relaxation, diaphragmatic breathing, and guided imagery, all of which reduce physical tension and anxiety that interfere with sleep onset [65]. Progressive muscle relaxation targets physical muscle tension, whereas diaphragmatic breathing activates the parasympathetic nervous system for a calming effect, and guided imagery promotes relaxation by focusing the mind on soothing mental imagery [66–68]. Together with cognitive restructuring, sleep restriction and stimulus control, these methods address both physiological and cognitive components of sleep onset anxiety to improve sleep quality.

3.2. Psychoeducation

In addition to cognitive-behavioral methods, psychoeducation plays a crucial role in managing anxiety-induced chronic insomnia [69], by helping individuals understand how anxiety affects their sleep and the sleep-anxiety cycle, whereby anxiety triggers hyperarousal that further disrupts sleep.

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Education on the biological mechanisms underlying sleep onset anxiety, including both cognitive and physiological aspects, empowers individuals to identify symptoms, apply coping strategies, and reduce maladaptive responses to pre-sleep worry [70]. Psychoeducation commonly incorporates guidance on sleep hygiene principles and behavioral modification, such as maintaining a consistent sleep-wake schedule to stabilize circadian and arousal systems. Instruction on how to create an optimal sleep environment (e.g., dark, quiet, and cool) and the benefits of a regular sleep schedule reinforces these positive habits, promoting sleep quality in the long term [71]. Additionally, guidance on modifying sleep-affecting behaviors, such as limiting screen time before bed or reducing caffeine intake, further supports anxiety reduction and promotes restorative sleep [72]. Overall, psychoeducational interventions strengthen self-efficacy and treatment adherence, translating mechanistic understanding into sustained behavioral change that supports long-term sleep restoration.

3.3. Mindfulness-Based Interventions

Mindfulness-based interventions can help individuals focus on the present moment and reduce the cognitive hyperarousal that often accompanies sleep onset anxiety [73,74]. Recent systematic reviews and meta-analyses have shown that mindfulness-based interventions typically yield small-to-moderate improvements in insomnia severity and sleep quality, particularly among individuals with depression or anxiety disorders [75]. Mechanistically, mindfulness meditation cultivates nonjudgmental awareness of thoughts, diminishing the impact of intrusive and worry-based cognitions that interfere with sleep initiation [76]. RCTs further indicate that mindfulness-based interventions significantly alleviated insomnia severity and depression, reduced pre-sleep rumination, and decreased physiological arousal, thereby improving sleep outcomes [77]. Converging evidence supports mindfulness-based interventions as effective strategies that modulate cognitive-emotional reactivity and autonomic activity to promote sleep initiation.

3.4. Acceptance and Commitment Therapy (ACT)

ACT extends the principles of mindfulness by emphasizing psychological flexibility – one's capacity to tolerate aversive internal experiences, such as anxiety or wakefulness, without engaging in maladaptive control efforts [78]. Rather than suppressing distressing cognitions, ACT promotes acceptance and value-consistent action, thereby reducing the struggle that sustains insomnia-related arousal [78]. Preliminary studies suggest ACT may influence neural and autonomic markers of emotional regulation, though mechanisms remain under investigation [79]. These neurobiological effects collectively reduce both cognitive hyperarousal and autonomic dysregulation, facilitating sleep initiation and maintenance. A systematic review has shown that ACT significantly improved both primary and comorbid insomnia and sleep quality across clinical populations [80]. Emerging evidence from RCTs further demonstrates sustained improvements in sleep efficiency, daytime functioning, and affective regulation, positioning ACT as a viable adjunct or alternative to traditional CBT-I for patients with high anxiety sensitivity or emotional dysregulation [81]. These approaches acceptance-based approaches have demonstrated efficacy as adjunctive treatments for chronic insomnia, supporting sustained improvements in sleep quality and emotional well-being.

3.5. Engagement Strategies

Engagement strategies are crucial for attaining success in long-term treatment of sleep onset anxiety. Motivational interviewing (MI) and behavioral contracts are evidence-based models used to maximize adherence to treatment plans and encourage motivation toward lasting changes [82,83]. MI is a client-oriented, empathic approach that assists individuals in exploring and overcoming ambivalence toward behavioral change. In the context of sleep onset anxiety, MI guides individuals to identify their personal motivations for improving sleep, as well as the obstacles that impede engagement in healthier sleep habits. Through open-ended questions, reflective listening, and

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affirmations, MI helps to clarify goals, recognize the value of restorative sleep, and build readiness for change [82]. MI is particularly useful for those who are skeptical about treatment or struggle with maintaining long-term commitment to behavioral interventions [84]. Behavioral contracts are structured agreements between therapist and client that outline treatment goals, expectations, and responsibilities [83]. In the case of sleep onset anxiety, such contracts clarify the individual's role in their own therapeutic process, facilitating adherence to interventions, and enhancing treatment engagement. When collaboratively developed, behavioral contracts promote patient accountability and shared decision-making, which are associated with improved therapeutic outcomes [85]. Furthermore, consistent positive reinforcement when goals are achieved has been demonstrated to strengthen motivation and sustain behavioral change [86]. Together, these engagement strategies translate motivational and behavioral science into actionable frameworks that improve adherence, optimize treatment response, and consolidate the long-term efficacy of sleep-focused interventions.

3.6. Practical Tools

Various practical tools have been implemented as adjunctive interventions between therapy sessions to support progress and reduce anxiety. These instruments aim to help individuals gain insights into their sleeping habits and anxiety-inducing factors and provide them with evidence-based approaches for managing distress and improving sleep outcomes.

Worry journals represent a simple and evidence-based approach to manage sleep onset anxiety [87]. They provide a structured method for individuals to record anxious thoughts and worries throughout the day and at bedtime. By externalizing anxiety through writing, clients can reduce internal tension that interferes with sleep [87]. Journaling also helps identify recurring patterns of worry, such as those related to work or family, that often precede heightened nighttime anxiety [87]. Once such patterns are established, cognitive restructuring techniques can be implemented to challenge maladaptive thoughts and replace them with more balanced, reality-based perspectives, thereby reducing pre-sleep arousal and improving sleep quality [88].

Additionally, guided audio sessions have been found to be useful adjunctive tools for reducing sleep onset anxiety and promoting relaxation. These sessions often incorporate evidence-based techniques such as relaxation training, diaphragmatic breathing, progressive muscle relaxation, and guided visualization [89–92]. These approaches have been found to lower physiological arousal by reducing muscle tension, heart rate, and overall anxiety, which can facilitate the body's transition into sleep readiness [93]. Regular audio guidance is thought to condition both body and mind to associate bedtime with calmness, by serving as a controlled distraction from intrusive thoughts, allowing individuals to relax and adopt a more restful state conducive to sleep [93].

Recent advances in digital therapeutics have expanded the accessibility of behavioral sleep interventions, translating evidence-based methods such as CBT-I, ACT, and mindfulness into mobile and web-based formats [94]. Widely studied programs such as Sleepio, an automated digital CBT-I platform (dCBT-I), have demonstrated reductions in insomnia severity and improvements in sleep efficiency, with effect sizes typically in the small-to-moderate range [95]. Similarly, SHUTi (Sleep Healthy Using the Internet) and the CBT-I Coach app show improvements in sleep onset latency, total sleep time, and daytime functioning; however, outcomes are variable and are generally smaller than those observed with therapist-delivered CBT-I [96,97]. Other digital tools extending beyond CBT frameworks include ACT-based programs and mindfulness applications (e.g., Headspace, Calm), which provide structured modules for acceptance, relaxation, and anxiety reduction [98,99]. Furthermore, digital platforms that integrate guided meditation, breathing exercises, white noise, or biofeedback sensors serve as adjuncts to behavioral therapy by facilitating autonomic downregulation and relaxation prior to sleep [58,100–102]. These digital tools represent promising adjuncts for facilitating behavior change by providing readily available options for convenient access to relaxation methods and sleep tracking via smartphone, and by encouraging self-management and integration of evidence-based practices into daily life.

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Despite promising efficacy data, several implementation challenges persist. Data privacy and security remain concerns, as many commercial sleep apps collect sensitive information with variable transparency and oversight. Adherence can decline over time due to limited personalization or user fatigue, and many tools lack integration into formal clinical workflows, reducing opportunities for monitoring and follow-up. Additionally, equitable access remains a growing concern, especially for underserved populations that lack access to smartphones or reliable internet connectivity.

It is important to note that most efficacy data for these digital programs derive from broader chronic insomnia populations, and sleep-onset anxiety is typically addressed as one component of an overall insomnia presentation rather than as a primary, isolated outcome [97]. Nevertheless, digital interventions represent promising, scalable tools for addressing insomnia and sleep onset anxiety. Optimizing their clinical utility will require addressing privacy, engagement, interoperability, and accessibility gaps to ensure these technologies complement rather than replace clinician-guided care.

In summary, the integration of sleep onset anxiety interventions into clinical practice should follow a holistic, multimodal framework that combines CBT-I with psychoeducation, engagement strategies, and practical tools, to empower individuals for self-management, and achieve both immediate therapeutic benefits and sustained long-term improvements in sleep outcomes. This comprehensive approach allows clinicians to address both the cognitive and physiological components of anxiety while supporting adherence to treatment. Despite substantial empirical support, current evidence is constrained by methodological limitations, including small sample sizes, heterogeneous intervention designs, and potential publication bias. Moreover, most studies emphasize short-term efficacy, with comparatively limited data on long-term sustainability, scalability, and population-level generalizability. Future research should prioritize rigorous, longitudinal trials to evaluate maintenance of treatment gains across diverse demographic and clinical contexts. **Table 2** summarizes key evidence-based interventions for sleep onset anxiety.

Table 2. Summary of Evidence-Based Interventions for Sleep Onset Anxiety.

Evidence-Based Intervention	**Goals**	**Practices**	**Refs**
Cognitive-Behavioral Therapy for Insomnia (CBT-I)	Target maladaptive cognitions and behaviors that maintain sleep-onset difficulty	Cognitive restructuring, sleep restriction, stimulus control, paradoxical intention	[44,45]
Environmental Modifications (Stimulus Control)	Optimize sleep environment to reduce arousal and support circadian regulation	Light management, temperature optimization, auditory interventions (white noise, music therapy)	[52,58,63]
Relaxation Techniques	Reduce physical tension and promote parasympathetic activation	Progressive muscle relaxation, diaphragmatic breathing, guided imagery	[65–68]
Mindfulness and Acceptance-Based Strategies	Cultivate non-judgmental awareness and acceptance of anxious thoughts and arousal	Mindfulness meditation, Acceptance and Commitment Therapy (ACT)	[75,80]

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4. Clinical Considerations

Treatment responses for sleep onset anxiety vary considerably across individuals, shaped by comorbidities, developmental stage, and cultural context, which may influence both symptom presentations and treatment effectiveness. Personalized clinical management should account for each individual's cognitive, physiological, and psychosocial profile, ensuring that treatment goals and methods are tailored to optimize therapeutic outcomes.

4.1. Comorbid Anxiety Disorders

Sleep onset anxiety commonly occurs with generalized anxiety disorder (GAD), panic disorder (PD), and post-traumatic stress disorder (PTSD) [3]. Each disorder is associated with mechanisms that exacerbate pre-sleep arousal and perpetuate insomnia symptoms. Management requires disorder-specific therapeutic strategies to address both the primary anxiety condition and its impact on sleep [3]. The following sections outline how these comorbid disorders influence sleep patterns and the corresponding evidence-based treatment approaches.

4.1.1. Generalized Anxiety Disorder

GAD is characterized by persistent and excessive worry across multiple life domains. The intrusive, uncontrollable thoughts often persist into the pre-sleep period, and individuals with GAD frequently experience heightened fear and tension even in non-threatening contexts, perpetuating sleep onset anxiety and insomnia symptoms [1]. CBT remains one of the most widely studied and effective evidence-based interventions for GAD-related sleep difficulties, supported by multiple meta-analyses. CBT helps individuals identify and modify maladaptive or catastrophic thinking patterns, promotes more balanced cognitive appraisals, and reduces anxiety severity [103,104]. Complementary relaxation techniques, such as diaphragmatic breathing, progressive muscle relaxation, and guided imagery, further reduce somatic tension and pre-sleep arousal in individuals with GAD [65]. Meta-analyses support that reinforcing sleep hygiene behaviors, including maintaining consistent sleep-wake schedules and limiting caffeine or stimulating activities before bed, enhances treatment efficacy and supports long-term sleep-wake regulation [105,106]. These multimodal approaches highlight the need to address both cognitive and physiological contributors to pre-sleep arousal among individuals with GAD.

4.1.2. Panic Disorder

PD is characterized by recurrent, unexpected panic attacks that can emerge during sleep, manifesting as sudden episodes of intense fear or physiological arousal that often occur without identifiable triggers, contributing to significant sleep disturbances [107]. Fear of experiencing another attack reinforces hyperarousal, impairing sleep initiation and quality [107]. CBT directly targets the cognitive distortions and catastrophic thinking that sustain panic symptoms, and remains one of the most extensively validated interventions for PD, supported by multiple RCTs and meta-analyses demonstrating efficacy in reducing both panic frequency and sleep-related anxiety [108–110]. Cognitive restructuring enables individuals to identify and reframe irrational fears, reducing anxiety intensity. Exposure-based strategies, such as gradual exposure to sleep-related cues, have shown efficacy in reducing panic-related sleep disturbance in controlled trials [111,112]. Additionally, breathing retraining techniques, such as controlled diaphragmatic breathing, have been shown to regulate autonomic arousal by stabilizing heart rate and respiration, with RCT evidence supporting reductions in physiological hyperactivation and improved relaxation before sleep [113]. These interventions collectively highlight the value of combining cognitive, behavioral, and physiological regulation strategies to break the cycle of hyperarousal and fear conditioning in PD.

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4.1.3. Post-Traumatic Stress Disorder

PTSD is characterized by trauma-related nightmares, intrusive thoughts, and heightened physiological arousal, which contribute to sleep disturbances. Individuals with PTSD often experience persistent hypervigilance and difficulty disengaging from threat-related cognitions, making it difficult to relax and initiate sleep [114]. CBT-I has demonstrated efficacy in trauma-exposed populations, reducing pre-sleep arousal and improving sleep continuity by integrating conventional behavioral sleep strategies with trauma-informed adaptations in multiple RCTs [115,116], especially when combined with trauma-focused interventions, such as trauma-focused cognitive behavioral therapy (TF-CBT) and eye movement desensitization and reprocessing (EMDR) [117,118]. TF-CBT combines cognitive restructuring with trauma-sensitive exposure and coping strategies to alleviate the emotional and physiological sequelae of trauma and reduces trauma-related distress, hyperarousal, and nightmares while enhancing overall sleep quality [116], while EMDR facilitates adaptive reprocessing of distressing memories and decreases associated physiological activation and nightmares, thereby improving sleep [118]. Both TF-CBT and EMDR have demonstrated efficacy in reducing trauma-related distress and may reduce sleep onset anxiety among individuals with PTSD by addressing the underlying anxiety and emotional dysregulation driving sleep disturbances [119,120]. Systematic reviews of RCTs support that incorporating complementary relaxation and mindfulness-based practices provide additional benefits in regulating emotional reactivity and promoting parasympathetic recovery before bedtime [121]. Maintaining a safe and supportive therapeutic environment is essential, as fostering security and trust enables effective trauma processing and the reestablishment of restorative sleep [122]. These findings emphasize that effective management of sleep onset anxiety in PTSD requires an integrated, trauma-informed approach that simultaneously targets cognitive hyperarousal, physiological dysregulation, and emotional safety to restore psychological stability and sleep continuity.

4.2. Age-Related Considerations

Developmental stage influences the manifestation and management of sleep onset anxiety across the lifespan [123]. In children, sleep onset anxiety often arises from developmental factors such as emotional vulnerability, limited coping skills, and social pressures at school or within the family. Children may struggle to fall asleep due to fears of the dark, nightmares, or worries about academic or social performance [124]. Child-focused interventions typically integrate relaxation techniques (such as progressive muscle relaxation or guided imagery), and engage parents in establishing consistent bedtime routines that reinforce sleep-promoting behaviors [125].

Among adolescents, academic, social, and developmental stressors can intensify pre-sleep worry, leading to somatic symptoms such as muscle tension and elevated heart rate [126,127]. The developmental delay in sleep phase observed in adolescents shifts their circadian rhythms toward later sleep timing, that often conflicts with early school schedules, thereby amplifying anxiety around sleep initiation and perpetuating insomnia-like symptoms, which further exacerbates pre-sleep cognitive and physiological arousal [128,129]. Psychoeducation on sleep hygiene and coping techniques such as mindfulness meditation or sleep journals is essential for supporting emotional regulation and circadian alignment [130,131]. Parental involvement is critical in fostering a favorable sleep environment, promoting sleep consistency and modeling adaptive sleep behaviors [132].

In midlife, among women, hormonal transitions such as perimenopause and menopause are associated with fluctuations in estrogen and progesterone that influence thermoregulation, vasomotor symptoms, and anxiety, all of which can exacerbate difficulties with sleep initiation [133]. Vasomotor symptoms and nighttime awakenings may heighten anticipatory worry about sleep, reinforcing sleep onset anxiety. Behavioral interventions tailored for menopausal women, including CBT-I combined with relaxation training and environmental cooling strategies, show emerging efficacy in improving both insomnia symptoms and mood [134].

Among older adults, sleep onset anxiety often stems from chronic health conditions such as chronic pain, medication side effects, cognitive decline, or emotional stressors including loneliness

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and fear of loss or mortality [135–137], which heighten anxiety and interfere with relaxation before sleep. Interventions for this group address both physical and psychological dimensions, with considerations of comorbidities and medication effects that may influence sleep physiology. Environmental modifications, including optimizing room temperature, noise reduction, and light adjustment, help create a calm, safe, and comfortable sleep environment that supports management of sleep onset anxiety in this population [138]. Relaxation techniques such as progressive muscle relaxation or breathing exercises relieve physical tension, while cognitive restructuring reduces irrational fears related to aging [139,140]. Social engagement and physical activity further support emotional well-being and sleep quality in older adults [141].

4.3. Cultural Influences

Despite limited cross-cultural data, existing evidence and systematic reviews suggest that cultural context influences the perception, expression, and treatment of sleep onset anxiety and related sleep problems, by shaping how individuals interpret symptoms, attribute causes, and engage with therapeutic interventions [142]. In some cultures, insomnia and anxiety carry social stigma, discouraging disclosure or help-seeking [143], whereas cultures emphasizing collectivism or holistic wellness may show greater openness to integrative approaches such as CBT or mindfulness that align with traditional healing values [144]. Cultural norms can shape the expression of anxiety, with some populations displaying predominantly somatic symptoms such as headaches or stomach discomfort, whereas others primarily verbalize worry or fear [145,146]. Culturally informed assessment frameworks and adaptations are important to enhance accessibility and engagement while maintaining fidelity of core treatment principles.

Culturally tailored interventions for sleep onset anxiety require adaptation of therapeutic content, delivery format, and communication style to align with patients' cultural values. Clinicians may assess how cultural beliefs shape the meaning of anxiety, sleep, and help-seeking, and flexibly adjust techniques to enhance engagement and reduce stigma. For example, in collectivist, harmony-oriented cultures, interventions may focus on alleviating interpersonal stress through family therapy or community-based support to strengthen motivation, accountability, and adherence [147]. In spiritually oriented populations, mindfulness practices may be reframed through culturally familiar forms such as prayer, reflection, or gratitude rituals to enhance resonance and therapeutic engagement [148]. For individuals from achievement-oriented cultures, strategies incorporating goal setting and self-affirmation may help mitigate performance-related anxiety and facilitate sleep initiation [149]. In cultures where emotional experiences are primarily expressed somatically, cognitive restructuring can be adapted to focus on reframing the physical manifestations of anxiety, while group-based mindfulness interventions may foster collective participation and normalize sleep-related distress within a supportive social context [150].

Incorporating culturally familiar sensory elements, such as traditional music or nature sounds, may enhance relaxation and emotional safety, potentially increasing engagement and reducing stigma [151]. Systematic reviews of cross-cultural RCTs support the use of music therapy as an adjunctive intervention for anxiety and sleep disorders, demonstrating benefits in reducing physiological arousal, promoting relaxation, and enhancing emotional regulation across diverse cultural settings [59,152]. Integrating culturally resonant music therapy within CBT or mindfulness may be a promising approach to augment therapeutic outcomes for individuals with sleep onset anxiety.

5. Conclusions

Sleep onset anxiety arises from the interplay of cognitive hyperarousal, physiological activation, and neurobiological dysregulation, which disrupt the natural sleep initiation process. Intrusive thoughts, heightened vigilance, and somatic tension perpetuate insomnia and impair sleep quality, underscoring the need for integrative approaches that target both cognitive and physiological processes. Evidence supports multimodal approaches combining CBT-I components (cognitive

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restructuring, stimulus control, sleep restriction, relaxation training) with psychoeducation and mindfulness-based techniques. Digital tools extend therapeutic reach beyond clinical sessions, while engagement strategies such as motivational interviewing and behavioral contracts support adherence and self-efficacy. Given that sleep onset anxiety affects individuals across diverse developmental stages and cultural contexts, tailored interventions are essential.

Despite growing evidence, current interventions rely primarily on short-term studies, with limited data on long-term efficacy, cross-cultural applicability, and integration into routine clinical care. Future research would benefit from mechanistic, longitudinal RCTs that integrate objective physiological measures (e.g., heart rate variability, actigraphy, endocrine markers, neuroimaging) with clinical outcomes to clarify causal pathways and inform personalized, scalable interventions. Continued innovation and clinical collaboration will be essential in translating evidence-based practices into accessible, individualized care, addressing sleep onset anxiety comprehensively across populations. Longitudinal and comparative effectiveness studies are especially needed to establish sustained benefit and clarify mechanisms across populations.

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