**REVIEW OF RELATED LITERATURE:**

**BEHAVIORAL TREATMENT OF INSOMNIA DISORDER**

Introduction  
The annual prevalence of insomnia symptoms in the general adult population ranges from 35% to 50% (Walsh, Coulouvrat & Hajak et al, 2011).  To distinguish chronic from acute insomnia, which may occur in anyone at one time or another, varied definitions for chronic insomnia have been utilized from study to study, with minimum durations ranging from 30 days to as long as 6 months (National Institutes of Health, 2005).  The prevalence of insomnia disorder ranges from 12% to 20% (Morin, LeBlanc, Belanger, Ivers, Merette, & Savard J. 2011; Roth, Coulouvrat, & Hajak et al. 2011).

Insomnia Disorder involves a predominant complaint of dissatisfaction with sleep quantity or quality, associated with one or more of the following symptoms: (1) difficulty initiating sleep, (2) difficulty maintaining sleep characterized by frequent awakenings or problems returning to sleep after awakenings; (3) early morning awakening with inability to return to sleep (American Psychiatric Association, 2013).

Sedative hypnotics or antidepressant drugs are the most common treatments offered patients who meet clinical criteria for Insomnia Disorder (Walsh & Schweitzer, 1999).  Numerous negative side effects accompany traditional hypnotics, e.g., benzodiazepines.  In addition, these drugs provide only symptomatic relief because they do not address underlying mechanisms which sustain primary insomnia. As a result, upon termination of the sleep medications, patients very commonly experience a full return of their insomnia symptoms.

Given the prevalence of insomnia and the limited effectiveness of medication for the treatment of insomnia disorder, there is a need for non-pharmaceutical behavioral treatment approaches.

Cognitive Behavioral Therapy for Insomnia

Cognitive behavioral therapy for insomnia (CBT-I) is the most prominent non-pharmacologic treatment for insomnia disorder (Wu, Appleman, Salazar & Ong, 2015). Early in the development of what is now “CBT-I”, first generation behavioral interventions were designed to target presumed perpetuating mechanisms involved in primary insomnia. The approach aimed to correct sleep disrupting habits using stimulus control and to reduce bedtime arousal using relaxation training, for example. Over time cognitive behavior therapy for insomnia has adopted a multi-component approach to treatment.

As noted by Perlis et al (2008), the clinical practices which constitute the primary or first line components are to some extent debated. However, most clinical practices and clinical trial protocols now include (1) stimulus control therapy and (2) sleep restriction therapy along with (3) sleep hygiene education, (4) cognitive therapy to identify and reduce negative sleep thoughts, and often (5) relaxation strategies such as progressive muscle relaxation and bedtime rituals that facilitate calming, especially as bedtime approaches. (1) Stimulus control is a set of instructions originally developed by Richard Bootzin that address conditioned arousal. These instructions strengthen the bed and bedroom as a cue for sleep and weaken the bed and bedroom as a cue for wakefulness. (2) Sleep restriction therapy or “sleep consolidation training” is a procedure originally designed by Arthur Spielman to eliminate prolonged awakenings in the middle of the night. The procedure can also help with difficulty initiating asleep at the beginning of the night. This step-wise procedure aims to first improve sleep quality and later worry about sleep quantity. The time spent in bed is initially restricted to the currently feasible amount of sleep. Time spent in bed is gradually increased in subsequent steps. (3) Sleep hygiene education commonly lists best practices in sleep hygiene (get up at the same time each day, cut down on all caffeine products, make sure your bedroom is at a comfortable temperature during the night, etc.). Typically this involves education, then identifying specific sleep hygiene practices for improvement, identifying goals and follow up. (4) Cognitive therapy and (5) relaxation therapy are often included on an as needed basis to bolster incomplete treatment responses and address predisposing and precipitating factors thought to still be substantially contributory (Perlis et al, p. 17). The cognitive therapy component identifies negative sleep talk such as “I can’t stand it that I’m not getting to sleep” or “If I don’t get 8 hours of sleep tonight, I’ll do poorly at work and I could get fired”. Some cognitive therapists have a didactic focus. Others use a variety of cognitive restructuring procedures. While the therapies differ in approach, all are based on the observation that persons with insomnia have negative thoughts and beliefs about their condition and its consequences (Perlis, p. 18). Relaxation therapy focuses on managing stress, muscle relaxation and bedtime rituals that facilitate calming [https:// stanfordhealthcare.org/medical-treatments/c/cognitive-behavioral-therapy-insomnia.html].

Effectiveness of CBT-I

The results using CBT-I among subjects with primary insomnia reporting sleep onset problems have been moderately effective for treating sleep onset problems. In a meta-analysis of 37 studies, Wu, Appleman, Salazar and Ong (2015) reported 36% of CBT-I patients were in remission from insomnia and no longer met diagnostic criteria for Insomnia Disorder at post-treatment evaluation compared to 16.9% in comparison conditions.

Key quantitative measures include sleep latency, the amount of time it takes person to initiate sleep upon retiring to bed; wake time after sleep onset (WASO); total sleep time (TST); and sleep efficiency, the percentage of sleep period during which person is actually asleep. In their review of the results of five randomized clinical trials which compared CBT-I to medications among patients with primary insomnia, Mitchell, Gehman, Perlis and Umscheid (2012) report sleep efficiency improved 8 to 16 percent with CBT-I. CBT-I generally led to improvement of 30 to 45 minutes in sleep latency and 30 to 60 minutes in total sleep time. Note that TST is not only an outcome variable but is manipulated as part of CBT-I using the restriction of time spent in bed. Sleep restriction therapy requires that time in bed be reduced to a time interval equal to the patient’s ‘sleep ability’ by measuring average TST with a sleep log during a baseline period. As noted by Mitchell, Gehrman, Perlis and Umscheid, “The net result of this, after completion of CBT-I, is that many patients do not recover their baseline TST but are nevertheless substantially improved with respect to other aspects of sleep” (p. 6).

Limitations of Cognitive Behavior Therapy for Insomnia

According to Mitchell et al “There are two main disadvantages to CBT-I. First, during the first few week of treatment there is often an acute reduction in total sleep time that can lead to the side effect of increased daytime sleepiness which, for some, is enough to lead them to drop out of treatment. Second, improvements from CBT-I are typically not seen until 3-4 weeks into treatment” (p. 3 of 18). Also noteworthy is the emotional response of patients upon learning their sleep will be restricted, commonly with a markedly later bedtime during the implementation of sleep restriction. Although apparently no research to date has assessed patient satisfaction and/or distress during treatment implementation, this may be a factor related to dropout rate with CBT-I. Among ten randomized controlled trials of CBT-I for primary insomnia reporting dropout rate and involving sleep restriction, dropout rates ranged from 0% to 33%. Specifically, 4 studies reported 0% dropout; 1 study reported 7% dropout; 2 studies reported 8% dropout; and dropout rates of 11%, 18% and 33% were each reported in one study (Table 1, Okajima, Komada & Inque, 2011).

In addition, while 36% remission of insomnia disorder is better than 16.9% (comparison group), this means 64% of those completing CBT-I continued to meet diagnostic criteria for Insomnia Disorder (Wu, Appleman, Salazar and Ong, 2015). With all due respect for the major contribution of CBT-I in assisting those with insomnia, there is room for improvement as evidenced by 64% still having Insomnia Disorder upon completion of treatment.

Another potential limitation of CBT-I relates to the mechanism of change and may lead to a means to improve upon the efficacy of behavioral treatment(s) for insomnia. “Research by Gregg D. Jacobs, Ph.D. and colleagues has established that insomniacs have faster brain wave patterns in bed than good sleepers which is consonant with heightened mental activity.” (Jacobs, 2000, p. 22). The faster brain wave pattern is only partially addressed in CBT-I which focuses on negative sleep talk. However, the emphasis in CBT-I is upon reality testing and/or challenging negative sleep talk, not upon slowing the faster brain wave pattern itself. A behavioral treatment approach which targets the actual here-and-now brain wave frequency with the goal of slowing the brain wave frequency in the moment at the time when the person wants to go to sleep may further improve their ability to do so.

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Brain Wave Frequency and Sleep

The human brain is composed of neurons that are interconnected to each other in networks. These neurons receive inputs from other areas of the brain. Electrical activity in the form of nerve impulses is always present in the form of nerve impulses being sent and received, even during sleep. The electrical activity reflects both intrinsic activity of neurons in the cerebral cortex (an internal process, i.e., thinking) and information sent to it by the body and sensory receptors (input from sight, sound, smell, touch).

This composite activity is measured with an electroencephalogram or EEG. An EEG mainly detects the activity of the brain region just under it, the cerebral cortex. Nevertheless, the electrodes receive the activity from thousands of neurons. In fact, one square millimeter of cortex has more than 100,000 neurons.

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| **Frequency range** | **Name** | **Usually associated with:** |
| 13–39 Hz | Beta waves | Active, busy or anxious thinking and active concentration, arousal, cognition, and or paranoia |
| 7–13 Hz | Alpha waves | Relaxation (while awake), pre-sleep and pre-wake drowsiness, REM sleep, [Dreams](https://en.wikipedia.org/wiki/Dream) |
| 4–7 Hz | Theta waves | Deep [meditation](https://en.wikipedia.org/wiki/Meditation)/relaxation, [NREM sleep](https://en.wikipedia.org/wiki/NREM_sleep) |
| < 4 Hz | Delta waves | Deep dreamless [sleep](https://en.wikipedia.org/wiki/Sleep), loss of body awareness |

(Precise boundaries between ranges vary among definitions; there is no universally accepted standard.)

The dominant frequency determines one's current state. For example, if alpha waves are dominating, the person is in the alpha state. This happens when one is relaxed but awake. However, other frequencies will also be present, although with smaller amplitudes.

Falling asleep for a waking state begins with Stage 1 (non-REM) which is the stage between wakefulness and sleep, sometimes referred to as somnolence or drowsy sleep, in which the muscles are still quite active and the eyes roll around slowly and may open and close from time to time. In more scientific terms, stage 1 is the period of transition from relatively unsynchronized beta and gamma brain waves (with a frequency of 12-30 Hz and 25-100 Hz respectively), which is the normal range for the awake state, to more synchronized but slower alpha waves with a frequency of 8-13 Hz, and then to theta waves with a frequency of 4-7 Hz. It is difficult to pinpoint the actual point of sleep onset (falling asleep), as the process is a continuum as brain wave activity gradually slows down (<http://www.howsleepworks.com/types_nonrem.html>).

In short, a predominant lower brain wave frequency is associated with sleep, and a predominant higher brain wave frequency is associated with busy or anxious thinking and arousal. Therefore shifting the predominant brain wave frequency to a lower frequency should be associated with increased ability to initiate sleep.

How can lower brain wave frequencies be increased when a person wants to sleep? A potential answer to this question and an associated treatment approach for insomnia comes primarily from the literature on meditation with ancillary guidance from the literature on brain entrainment.

Meditation

Meditation’s effect on the brain can be put into two categories: state changes and trait changes, respectively alterations in brain activities during the act of meditating and changes that are the outcome of long-term practice. The former is of interest as relates to lowering the predominant brain wave frequency to increase ability to initiate sleep. Assessed in a review by Cahn and Polich (2006), many studies on meditation have linked frequency alpha (8-13 Hz) and theta (4-7 Hz) waves to meditation (Cahn & Polich, 2006). A much older study reports more specific findings, such as decreased alpha blocking and increased frontal lobe specific theta activity. Alpha blocking is a phenomenon where the active brain, normally presenting beta wave activity, cannot as easily switch to alpha wave activity (Kasamatsu & Hirai, 1966).

Envision a person is in bed, head on the pillow, sheet and blanket covering them, eyes closed, and wanting to go to sleep. If the person does not go to sleep, they are very likely thinking about things. For example, they might think about what a family member said or something they have to do tomorrow. In any event, thinking about things activates the brain, and the brain activation maintains a higher predominant brain wave frequency which is associated with being awake. In essence, that upon which the person focuses their attention stimulates a certain brain wave frequency. Imagine falling out a window several stories above street level, for example, and notice how anxiety immediately increases. Think of the possibility a loved one’s flight tomorrow will crash, and anxiety level will again immediately increase. These examples illustrate how focus of attention relates to brain wave frequency: anxiety reflects a higher predominant brain wave frequency at the moment. In short, a person’s focus of attention relates to their predominant brain wave frequency.

When in bed and wanting to initiate sleep, “thinking about things” reflects the mind focused on activating thoughts. These activating thoughts relate to higher brain wave frequency. Thus the person has difficulty initiating sleep, a phenomenon associated with a lower brain wave frequency. With the focus on something less activating, it is reasonable to believe the predominant brain wave frequency will be lower.

Meditation eliminates background mental noise, commonly by focusing attention on the breathe (yoga, for example), a mantra (transcendental meditation), a calming image (guided imagery), or non-judgmental observation of one’s own thinking (mindfulness). As relates to facilitating sleep, the sound of the breath and a mantra are of particular interest as steady, unmoving auditory processes. Imagery involves the visual brain, and it is the auditory brain which is activated while “thinking about things”. Mindfulness meditation focuses on observing one’s own thinking process, which is itself an ever shifting phenomenon and therefore at least mildly activating. While in bed trying to sleep, the act of thinking “Oh, I have to remember to pay the rent tomorrow” involves auditory activation. A much less activating auditory stimulus is the sound of one’s breathing. Shifting and then maintaining the focus of attention on listening to the sound of here-and-now breathing shifts the auditory brain to a less activating auditory stimulus, thereby lowering the predominant brain wave frequency. One tends to become bored with continued listening to the sound of one’s own breath. Boredom reflects less activation. Similarly, repeating a non-stimulating mantra again and again in one’s mind shifts the focus to a less activating auditory stimulus, thereby lowering the predominant brain wave frequency.

Lower brain wave frequency is associated with relaxation, deep meditation and sleep. Therefore, meditative practices which increase the predominance of lower brain waves appear fitting as a means of increasing the ability to initiate sleep.

## The first author has been using specific meditative strategies with outpatient clients whose therapy goals include increased ability to initiate sleep. The meditation strategies are (1) listening to the sound of one’s own breathing; (2) using a mantra to slow and quell thinking; and (3) guided imagery linking breathing with focus on calming during exhalation. With an explanation of the approaches and specific directions for focusing attention on listening to the sound of their own breathing, clients whose overall anxiety level is low to moderate often report the next week, “It works.” However, clients whose overall anxiety level is moderate to high often report the next week, “It works a little.” “Some nights it worked. Some nights I just couldn’t stop thinking about things.” As follow up, when these more anxious clients are then given an explanation and specific directions for using a mantra, these clients often report the next week, “It works.” As follow up with more anxious clients reporting considerable body activation, specific directions are given for guided imagery to link the calming body sensation with breathing out.

## Listening in the moment to the sound of the breath is a more passive activity, presumably with less brain activation than actively repeating a mantra. If in fact manta-based meditation is more effective in slowing the brain of the more anxious person, brain entrainment provides one possible explanation. Here is a synopsis on brain entrainment, followed by a working hypothesis regarding the differential effectiveness of the two meditative strategies.

## Brain Entrainment

## Human subjects rarely hear frequencies below 20 Hz, which is exactly the range of [Delta](https://en.wikipedia.org/wiki/Delta_wave), [Theta](https://en.wikipedia.org/wiki/Theta_rhythm), [Alpha](https://en.wikipedia.org/wiki/Alpha_wave), and low to mid [Beta](https://en.wikipedia.org/wiki/Beta_wave) [brainwaves](https://en.wikipedia.org/wiki/Neural_oscillation). Among the methods by which some investigations have sought to induce lower brain wave frequencies is to have subjects listen to binaural beats.

A binaural beat is an [auditory illusion](https://en.wikipedia.org/wiki/Auditory_illusion) [perceived](https://en.wikipedia.org/wiki/Perception) when two different pure-tone [sine waves](https://en.wikipedia.org/wiki/Sine_wave), both with [frequencies](https://en.wikipedia.org/wiki/Frequency) lower than 1500 Hz, with less than a 40 Hz difference between them, are presented to a [listener](https://en.wikipedia.org/wiki/Hearing), one through each [ear](https://en.wikipedia.org/wiki/Ear). For example, if a 530 Hz pure [tone](https://en.wikipedia.org/wiki/Pitch_(music)) is presented to a subject's right ear, while a 520 Hz pure tone is presented to the subject's left ear, the listener will [perceive](https://en.wikipedia.org/wiki/Perception) the [auditory illusion](https://en.wikipedia.org/wiki/Auditory_illusion) of a third tone, in addition to the two pure-tones presented to each ear. The third sound is called a binaural beat, and in this example would have a [perceived](https://en.wikipedia.org/wiki/Perception) [pitch](https://en.wikipedia.org/wiki/Pitch_(music)) correlating to a frequency of 10 Hz, that being the difference between the 530 Hz and 520 Hz pure tones presented to each ear (Draganova, Ross, Wollbrink & Pantev, 2008).

## Listening to binaural beats has been shown to precipitate auditory driving by which ensembles of cortical neurons entrain their [frequencies](https://en.wikipedia.org/wiki/Frequency) to that of the binaural beat, with associated changes in self-reported subjective experience of emotional and cognitive state (Becher, Höhne, Axmacher, Chaieb, Elger & Fell, 2015; Pratt, Starr, Michalewski, Dimitrijevic, Bleich & Mittelman, 2009; Karino, Yumoto, Itoh, Uno, Yamakawa, Sekimoto & Kaga, 2006; McConnell, Froeliger, Garland, Ives & Sforzo, 2014).

## The brain entraining is more effective if the entraining frequency is close to the user's starting dominant frequency. Therefore, it is suggested to start with a frequency near to one's current dominant frequency (likely to be about 20 Hz or less for a waking person) and then slowly decrease or increase it towards the desired frequency.

## Implications for Meditation Practices for Insomnia

## While brain entrainment provides a means of stimulating a particular brain wave frequency, the approach requires expensive equipment not commonly available and to date untested in the treatment of insomnia. It nonetheless provides guidance in the process of shifting the brain wave frequency. Specifically, a strategy that increases the frequency slightly lower than the predominant brain wave frequency at hour of sleep may be more effective than a strategy that increases a frequency markedly lower than the predominant frequency at that moment.

## Summary

The prevalence of insomnia disorder ranges from 12% to 20%. The most prominent non-pharmacologic treatment for insomnia disorder is cognitive behavioral therapy for insomnia (CBT-I). Meta-analysis indicates 36% remission of insomnia disorder upon completion of CBT-I vs. 16.9% in the comparison group(s). This means 64% of those completing CBT-I continued to meet diagnostic criteria for Insomnia Disorder. While CB-I has made a major contribution in assisting those with insomnia, there is room for improvement as evidenced by 64% still having Insomnia Disorder upon completion of treatment with CBT-I.

## Research has established that insomniacs have faster brain wave patterns in bed than good sleepers which is consonant with heightened mental activity. The mechanisms of action for CBT-I do not directly address the faster brain wave patterns. Assessed in a review by Cahn and Polich (2006), many studies on meditation have linked frequency alpha (8-13 Hz) and theta (4-7 Hz) waves to meditation (Cahn & Polich, 2006). A predominance of these lower brain wave frequencies has been found to be directly related to relaxation and sleep. The first author’s clinical use of meditation techniques to increase clients’ ability to intitiate sleep has met with frequent client feedback that “It works”. This approach, called “focus of attention” appears promising. However, “It works” is not sufficient. A clinical trial is needed in order to assess the relative efficacy of (a) cognitive behavior therapy for insomnia, (b) focus of attention [FOA], (c) a combination of CBT-I and FOA and (d) a comparison group.

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