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# Who Is a Candidate for Cognitive–Behavioral Therapy for Insomnia?

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Chronic insomnia impacts 1 in 10 adults and is linked to accidents, decreased quality of life, diminished work productivity, and increased long-term risk for medical and psychiatric diseases such as diabetes and depression. Recent National Institutes of Health consensus statements and the American Academy of Sleep Medicine's Practice Parameters recommend that cognitive–behavioral therapy for insomnia (CBT-I) be considered the 1st line treatment for chronic primary insomnia. Growing research also supports the extension of CBT-I for patients with persistent insomnia occurring within the context of medical and psychiatric comorbidity. In the emerging field of behavioral sleep medicine, there has yet to be a consensus point of view about who is an appropriate candidate for CBT-I and how this determination is made. This report briefly summarizes these issues, including a discussion of potential contraindications, and provides a schematic decision-to-treat algorithm.

*Keywords:* insomnia, cognitive–behavioral therapy, stimulus control, relaxation, sleep restriction

There is now decades of solid evidence that convincingly demonstrates that cognitive–behavioral therapy for insomnia (CBT-I) is efficacious (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995), as efficacious as sedative hypnotics during acute treatment (4–8 weeks, Smith et al., 2002), and more potent over the long term (Morin, Colecchi, Stone, Sood, & Brink, 1999). This, along with mounting evidence that chronic insomnia is a significant risk factor for psychiatric (Ford & Kamerow, 1989) and medical (Mallon, Broman, & Hetta, 2002) morbidity, clearly indicates that insomnia, in and of itself, is an appropriate target for treatment and that CBT-I should be considered a first line approach. Unfortunately, as with many chronic health conditions, the translation of outcomes research to clinical practice can be difficult and uncertain territory. In the field of behavioral sleep medicine, there is a paucity of pragmatic information elaborating some of the issues fundamental to the practice of CBT-I. The aim of this article is to discuss one such neglected issue by identifying a perspective for determining who is a candidate for CBT-I. In so doing, we will also briefly highlight some of the factors that might contraindicate the implementation of specific cognitive–behavioral components with particular patients.

## What Is CBT-I?

The most commonly used and empirically validated cognitive–behavioral interventions for insomnia include stimulus control therapy (SCT; Bootzin, 1972), sleep restriction therapy (SRT; Spielman, Saskin, & Thorpy, 1987), relaxation therapies (RT, Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001), and multicomponent approaches (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). Although extensive descriptions of each of these interventions may be found in several sources (Morin, Hauri, et al., 1999), the various therapies that constitute CBT-I are briefly described as follows. SCT serves to reassociate the bedroom environment as a primary stimulus for sleep rather than as a cue for sleep-incompatible behaviors and states of arousal. SRT consolidates fragmented sleep by initially curtailing sleep opportunity (increasing sleep deprivation) and then systematically extending sleep opportunity contingent upon the maintenance of adequate sleep efficiency (total sleep time/time in bed). Relaxation therapies aim to directly reduce sleep-incompatible states of arousal. Multicomponent CBT-I typically integrates one or more behavioral treatments with cognitive therapy aimed at modifying maladaptive sleep-related beliefs or managing intrusive presleep cognitions.

## Is CBT-I Only Indicated for Primary Insomnia?

Although there is an extensive outcomes literature for many of the CBT-I interventions and multicomponent treatment, this literature is based largely on highly selected samples of medically and psychiatrically healthy patients diagnosed with "primary insomnia." This is especially problematic when one considers that the vast majority of insomnia complaints are associated with medical and psychiatric disturbances (Buysse et al., 1994; Ohayon, 1997). Therefore, an important practical question faced by most clinicians caring for patients with insomnia is: "Can CBT-I be effectively implemented within the context of medical or psychiatric comorbidity?"

The issue of whether CBT-I can be used effectively in clinical samples can be answered both on theoretical grounds and increasingly on an empirical basis. With respect to the former, cognitive–behavioral models of chronic insomnia posit that acute medical and psychiatric illnesses are common precipitants of acute insomnia. Chronic insomnia, however, is often perpetuated, at least in

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part, by a circumscribed set of factors related to how the patient copes with acute insomnia symptoms (Spielman, Caruso, & Glovinsky, 1987; Perlis, Smith, & Pigeon, 2005). Some of these factors include extending sleep opportunity (going to bed early, sleeping in, and napping), engaging in nonsleep behaviors in the bedroom (worrying, working), and using alcohol and stimulants inappropriately. Often exacerbated by maladaptive beliefs about sleep and sleep loss, each of these behavioral responses to insomnia or fatigue can dysregulate chronobiologic mechanisms, enhance arousal level, and disrupt homeostatic processes necessary for properly timed and consolidated sleep. From a theoretical perspective, CBT-I targets these maintaining factors, and therefore, the extent to which these factors are judged to drive the insomnia complaint is a primary determinant of CBT-I candidacy.

Empirically, two kinds of data suggest that CBT-I can be extended to patients with medical or psychiatric comorbidity. First, three studies have demonstrated that CBT-I is effective in heterogeneous clinical samples and that psychiatric or medical comorbidity does not appear to attenuate outcome (Perlis, Sharpe, Smith, Greenblatt, & Giles, 2001; Morin, Stone, McDonald, & Jones, 1994; Lichstein, Wilson, & Johnson, 2000). Second, a growing number of clinical trials demonstrate that patients with chronic insomnia who carry specific medical or psychiatric diagnoses exhibit significant improvement in sleep when treated via CBT-I. To date, this has been shown for patients with chronic pain disorders, cancer, HIV, depression, posttraumatic stress disorder, and alcohol dependency (Smith, Huang, & Manber, 2005).

Taken together, the theory and available data suggest that CBT-I may be appropriate for both primary insomnia and insomnia occurring in the context of medical and psychiatric comorbidity. In fact, in the final analysis, the issue of "When is CBT-I indicated" is perhaps best framed as "When is CBT-I contraindicated." This perspective is elaborated below.

## When Is CBT-I Indicated or Contraindicated?

Figure 1 provides a "decision-to-treat algorithm" that takes into account many of the factors involved in determining whether CBT-I is indicated. Fundamental to the algorithm are several issues: (a) The candidate must exhibit difficulty initiating or maintaining sleep (DIMS) to a degree that treatment can exert an effect; (b) the DIMS problem is not primarily due to a circadian rhythm disorder; (c) the insomnia is not largely explained by an unstable illness that would interfere with, or be worsened by, the conduct of CBT-I; and (d) the patient exhibits some of the behavioral or psychological factors thought to perpetuate chronic insomnia.

### *The DIMS Complaint*

The patient must complain of trouble initiating or maintaining sleep that causes significant distress and/or impacts daytime functioning. Consistent with prevailing clinical and research criteria, a sleep initiation or maintenance problem is operationalized as reporting an average sleep latency, middle-of-the-night awakening time (wake time after sleep onset), or early morning awakening with a duration of 30 min or longer (Morin, 1993). Specifically excluded is the complaint of nonrestorative sleep in the absence of a sleep initiation or maintenance disturbance. While such a complaint of "primary nonrestorative sleep" may be defined as insomnia for either clinical (American Sleep Disorders Association, 1997) or research purposes (Edinger et al., 2004), there is very little, if any, evidence supporting the efficacy of CBT-I for these patients. Finally, we have deliberately omitted a symptom duration criteria for candidacy. Although most clinical outcome studies have typically included patients with an insomnia complaint of 3 months or longer, the presence of insomnia symptoms and maladaptive behaviors/conditioned arousal, in our opinion, may be sufficient cause to initiate treatment. In cases in which symptoms have been present for less than 3 months, treatment might best be construed as prophylaxis.

### *Circadian Rhythm Disorders Versus Insomnia*

When determining candidacy for CBT-I, it is important to evaluate whether the patient has a primary chronobiologic disorder, such as a phase delay or phase advance syndrome. Often such individuals report normal restorative sleep on weekends when they are able to go to bed and wake up as desired. In such cases, or in patients in whom the problem is limited to a severe (>3 hr) sleep onset or early morning awakening problem, further evaluation of a circadian rhythm disorder should be conducted, because phototherapy or other chronobiologic interventions would likely be the treatment of choice. Many patients with primary insomnia, however, may have some degree of chronobiologic dysregulation and therefore, combining chronobiologic treatment with CBT-I is likely to be the best approach for some (Lack, Wright, & Paynter, 1995).

### *Deferring CBT-I in Cases of Undiagnosed or Unstable Illness*

When it is clear that the patient has an undiagnosed or unstable medical or psychiatric illness, CBT-I should most likely be deferred if at least one or more of the following three common scenarios is present. First, it is suspected that the insomnia will entirely resolve with the acute/unstable illness, and there is no evidence that the insomnia is being maintained by maladaptive behaviors or intrusive presleep arousal. If this is the case, the patient should be counseled to avoid engaging in maladaptive compensatory strategies and should be reevaluated if symptoms persist after adequate treatment for the primary medical or psychiatric disorder. Short-term pharmacotherapy might be appropriate in these cases. Second, it is suspected that the insomnia will persist despite the resolution or stabilization of the illness, but the acute illness is likely to prevent the patient from effectively engaging in CBT-I. For example, an individual with severe major depression may not have the resources necessary to fully engage in SCT instructions or sleep diary monitoring or to implement the considerable practice necessary to learn relaxation skills. Initiating treatment at this time would, therefore, be ill advised and might lead to further reductions in self-efficacy. Third, it is suspected that CBT-I, or more specifically, some of its components, would aggravate the acute illness. Although many might assume CBT-I to be a completely benign set of therapies with minimal side effects, certain CBT-I components may be contraindicated with particular patients as highlighted below.

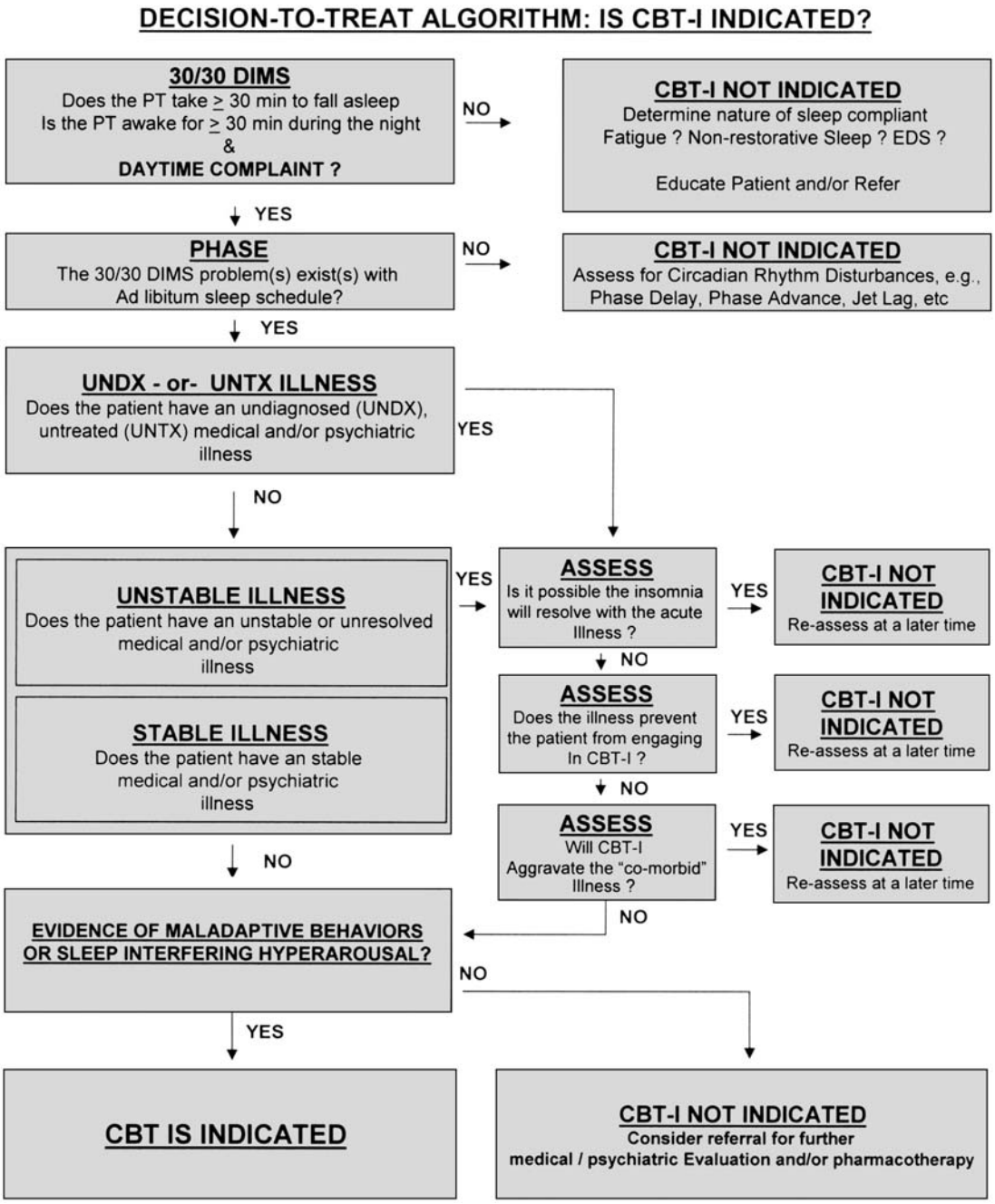


*Figure 1.* Decision-to-treat algorithm. Not enough data are available to recommend cognitive–behavioral therapy for insomnia when the primary complaint is nonrestorative sleep in the absence of other insomnia symptoms. This algorithm assumes that an assessment of other possible sleep disorders has been conducted. DIMS = Difficulty initiating or maintaining sleep, EDS = Excessive daytime sleepiness. Figure is adapted with permission from M. L. Perlis, C. Junquist, M. T. Smith, & D. Posner (2005), *Cognitive–Behaviorial Therapy for Insomnia*, p. 37, New York: Springer.

### *Contraindications of CBT-I Components*

*SCT.* SCT requires the patient to get out of bed and move to another room if not asleep within 15–20 min. These instructions, therefore, can be difficult and even potentially dangerous for frail patients, patients with restricted mobility, or those with medical conditions bearing increased risk of falls, such as orthostatic hypotension.

*SRT.* SRT may aggravate several preexisting conditions, notably: epilepsy, bipolar illness, parasomnias or other illnesses

(including in some rare cases, insomnia) that have excessive daytime sleepiness as a feature of the parent disorder. Sleep loss via sleep restriction may lower seizure threshold in epileptic patients (Fountain, Kim, & Lee, 1998), precipitate mania in bipolar patients (Colombo, Benedetti, Barbini, Campori, & Smeraldi, 1999), and exacerbate parasomnias related to nonrapid eye movement sleep transitions (e.g., sleep walking, night terrors; Mahowald & Bornemann, 2005). SRT might also simply increase daytime somnolence to a point at which it is no longer safe for the patient to drive, operate machinery, or make judgments that adequately promote their safety or the safety of others.

*RT.* RTs are widely assumed to have no side effects, but some studies have found that relaxation based approaches may be associated with paradoxical anxiety reactions in as many as 15% of cases (Heide & Borkovec, 1983). It is unclear which patients are prone to such reactions, but this phenomenon should be borne in mind, particularly with patients who appear overly reluctant to try such approaches.

Although some of the CBT-I components, as noted, may be contraindicated for some patients, it would be unusual that all of the various CBT-I components would be deemed inappropriate for any particular patient. This is encouraging, given recent evidence suggesting that none of the mainstream CBT-I components (SCT, SRT) or multicomponent approaches appear to have major differential efficacy on subjective measures of sleep for both younger and older adults alike (Irwin, Cole, & Nicassio, 2006). A notable exception however, may be that relaxation-based approaches, while efficacious, are not as helpful as SCT, SRT, and multicomponent approaches for some sleep parameters (Irwin, Cole, & Nicassio, 2006).

### *The Identification of "Perpetuating Factors"*

The final step in determining CBT-I candidacy is whether the patient displays evidence of maladaptive behaviors or sleep-interfering states of arousal thought to perpetuate chronic insomnia. As noted above, patients tend to cope with insomnia by extending sleep opportunity (going to bed early, sleeping in, and napping), engaging in nonsleep behaviors in the bedroom, and so forth. In addition, patients with chronic insomnia often exhibit sleep incompatible states of worry or somatized tension about their insomnia. Because each of these factors represent primary targets for CBT-I interventions, we argue that one or more must be evident for CBT-I to be indicated. In cases in which all of the candidacy criteria, except the presence of maladaptive behaviors or presleep hyperarousal, have been satisfied, it is likely that an unidentified medical (including sleep disorders) or psychiatric condition could cause the complaint and further evaluation is necessary. In such cases, pharmacotherapy might be an appropriate treatment option, pending appropriate evaluation.

## Summary

The various therapies that constitute CBT-I have been well-validated and their translation into clinical practice is now rapidly expanding. Theoretical models of insomnia and preliminary empirical data suggest that CBT-I is applicable for both primary insomnia and insomnia occurring within the context of medical or psychiatric comorbidity. Several factors are crucial for determining who is an appropriate candidate for treatment. These include: (a) determining the presence of sufficiently severe trouble initiating or maintaining sleep, (b) determining that symptoms are not exclusively due to a chronobiologic sleep disorder, (c) establishing that an unstable primary medical or psychiatric condition would not contraindicate treatment, and (d) determining the presence of maladaptive coping mechanisms or presleep hyperarousal.

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