CHAPTER

1

Standard cognitive behavioral therapy for insomnia (CBT-I)

When is the adaptation of CBT-I warranted?

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Introduction

Cognitive Behavioral Therapy for insomnia (CBT-I) is a multicomponent treatment that specifically targets sleep continuity disturbance[a] (difficulties initiating and/or maintaining sleep). CBT-I is typically comprised of

[a]Sleep continuity refers to the class or set of variables that represent "sleep performance." That is, it is a class term (vs. sleep architecture or sleep microarchitecture) for variables that represent latency to, and duration and efficiency of, the sleep that occurs during the sleep period, including Sleep Latency (SL), Number of Awakenings (NWAK), Wake After Sleep Onset (WASO), Early Morning Awakening (EMA), Total Sleep Time (TST), and Sleep efficiency (SE%). When one or more of these variables are pathologic, this may be referred to as *sleep continuity disturbance*. More, the use of this class term promotes a level of specificity that is unconfounded with the many denotations and connotations of the vernacular term "insomnia."

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Sleep Restriction Therapy, Stimulus Control procedures, and Sleep Hygiene instructions. Classical CBT-I included some form of relaxation training, though this is less true of contemporary protocols. With respect to the acronym "CBT-I," this was adopted (the hyphenated or "rodomized" form of the acronym) to elicit the response "I know what CBT is, but what the heck is CBT-I?!" (i.e., the abbreviation was and is used so that patients and clinicians may distinguish the insomnia form of CBT from other modalities [e.g., CBT-D, CBT-A, etc.]). The purpose of this chapter is to review the components of standard CBT-I, discuss its efficacy and effectiveness as a multicomponent therapy, and review common alternative approaches, rules, and therapies. The chapter closes with a discussion about the need to adapt CBT-I for specific clinical conditions or demographic groups.

Components of therapy

Standard CBT-I is delivered over the course of six to eight sessions (session length may vary between 15 and 90 min) (Perlis, Jungquist, Smith, & Posner, 2006). Each session typically has a specific agenda (e.g., evaluation, rationale, and Rx delivery of single therapies; adherence management; relapse prevention; etc.) (Perlis et al., 2006). Sessions most often occur in person on a weekly or biweekly schedule as individual or group encounters (Bastien, Morin, Ouellet, Blais, & Bouchard, 2004; Perlis et al., 2006; Verbeek, Konings, Aldenkamp, Declerck, & Klip, 2006; Yamadera et al., 2013; Zwart & Lisman, 1979a). Which prescriptive components are thought to be essential or to account for the most clinical outcome variance is a matter of debate and continues to be a subject of empirical evaluation (Epstein, Sidani, Bootzin, & Belyea, 2012; Harvey et al., 2014). Absent definitive data, most treatment protocols, and/or published treatment manuals focus on Sleep Restriction and Stimulus Control as the core therapies (Edinger & Carney, 2014; Manber & Carney, 2015; Morin & Espie, 2007; Perlis et al., 2006). More, the two treatment modalities are often used (and explained to patients) as complementary therapies. SRT's primary indication is to induce sleep quickly and to allow for consolidated sleep (Spielman, Saskin, & Thorpy, 1987). SCT's primary indication is to manage nocturnal wakefulness (Bootzin & Perlis, 2011). In the instances where these treatments are applied without specific cognitive therapy exercises (i.e., to debunk dysfunctional beliefs or to address catastrophization), treatment still includes a fair amount of cognitive work in the form of psychoeducation, the delivery of paradoxical instructions (i.e., stay awake longer and when awake at night get out of bed and be awake), and the management of nonadherence.

Sleep restriction therapy (SRT)

According to the Behavioral model of insomnia, the most important perpetuating factor for chronic insomnia is sleep extension. Sleep extension is the tendency for individuals to compensate for sleep loss by extending their time in bed, either by going to sleep earlier or waking up later (or by napping) (Spielman et al., 1987). A consequence of sleep extension, however, is the mismatch between sleep opportunity (i.e., how much time the patient spends in bed) and sleep ability (i.e., how much time the person actually sleeps). Therefore, the primary goal of SRT is to address this mismatch by limiting the amount of time a patient spends in bed to the amount of time that they are actually sleeping. SRT is thought to be effective owing to its effects on "Process S" (sleep restriction increases the homeostatic pressure for sleep and consequently reduces the time taken to fall asleep (SL) and/or the amount of time spent awake at night (WASO).

While the procedures for SRT vary from investigation to investigation (Kyle et al., 2015) and from treatment manual to manual (see Table 1), the following represents the original formulation (Spielman et al., 1987). The first step is to determine the patient's basal sleep ability in terms of average total sleep time (TST [as assessed with daily sleep diaries gathered over a period of two weeks]). Note that TST may be based on self-reported TST or by a derived measure of TST (TST-C = TIB + [SL + WASO + EMA]). The latter has the value of being internally valid. The next step is to determine a morning wake-up time that the patient can closely adhere to on a daily basis. Given these data, the clinician then sets the prescribed time in bed (PTIB, i.e., the patients' sleep window). This is done by subtracting PTIB from the desired wake-up time. For example, if a patient has a 6-h sleep ability and chooses to get up at 7:00 am every morning, their designated bedtime is 1:00 am. This sleep schedule is maintained or altered based upon how consolidated the individual's sleep is, as assessed with sleep efficiency SE% [sleep time/time in bed $\times$ 100]) once every 7 days. If SE% is less than 85%, PTIB is reduced by 15 min. If SE% is between 85% and 90%, PTIB is remains as prescribed. If SE% is greater than 90%, PTIB is increased by 15 min. Adjustments to the sleep schedule are usually accomplished by delaying, advancing, or leaving unchanged the individual's time to bed (TTB).

In theory, this titration process continues for as long as needed to recover or exceed baseline TST. In practice, titration is limited by the number of sessions allowed by the protocol (or third-party payers). It should be noted that most patients do not recover baseline TST within 8 sessions (83% based on one study (Perlis et al., 2019; Scott et al., 2021)). This has prompted some researchers and clinicians to modify SRT to use: (1) PTIB's that are in excess of average total sleep times; (2) shorter assessment

TABLE 1 Sleep restriction therapy (SRT) rules based on the first four published treatment manuals.

		Manual			
SRT elements/rules	**Issue**	Morin and Espie (2007)	Perlis et al. (2006)	Edinger and Carney (2014)	Manber and Carney (2015)
Session for SRT Rx	When is SRT implemented ?	Session 2 or 3	Session 2	Session 2	Session 2 (1)
# days baseline	How much baseline data is required ?	14 days	7–14 days	At least 14 days	14 days (2)
PTIB calculation	How is the TIB Rx calculated ?	Mean TST; PTIB = mean TST (1)	Mean TST; PTIB = mean TST (1)	Mean TST +30 min (1)	Mean TST; PTIB = Mean TST
PTIB minimum	What is the minimum allowable PTIB ?	5 h	4.5 h (2)	Not specified (2)	5 h
Initial Rx for PTIB	How is the sleep schedule set ? (1)	PTIB from earliest TOB (2)	PTIB from earliest TOB	PTIB from earliest TOB	PTIB from earliest TOB
Point of titration	What time point is used for SSE ? (2)	TTB or TOB	TTB (titrate back toward baseline)	Not specified	TTB or TOB (3)
Number of sessions	How many sessions are recommended ?	6–10 sessions	8 sessions	4 sessions (3)	6 sessions
Cycle length	How many days are required between RXs ?	7 days	7 days	Not specified (4)	7–14 days
Titration rule	What is the threshold for change in PTIB ?	> 90% (increase)	< 85 (decrease) 85–90 (stick) > 90 (increase) (3)	< 80 (decrease) 80–85 (stick) > 85 (increase) (5)	< 80 (decrease) 80–85 (stick) > 85 (increase) (4)
Titration increment	How much time is added/substracted from PTIB ?	15 min	15 min	15 min	15 or 30 (5)
	TIB, time in bed; *PTIB*, prescribed time in bed; *SSE*, systematic sleep extension; *TST*, total sleep time; *TTB*, time in bed; *TOB*, time out of bed				
	(1) Given PTIB, the procedure for setting the sleep schedule or sleep window? For example, if PTIB is 6 hours and the desired TOB is 7am, then TTB = 1am.	(1) 10 day sample of 14 is allowed. This may be to accommodate extreme values. For example, the first 4 days after d/c medication.	(1) It is curently reccomended that TST-C be used in liu of TST where TST-C = (TIB + [SL+WASO+EMA])	(1) The rationale for this accomodation is that +30 accounts for normal duration SL, WASO, and/or EMA and for subjective overestimates in these parameters	(1) Six biweekly sessions are reccomended. But less or more sessions are allowed based on patint needs
	(2) Systematic sleep extension (sleep period or sleep window expansion or time in bed expansion).This rule pertains to where time is added or subtracted during titration (e.g., TTB or TOB)	(2) The selection of TOB is generally based on patient preference, but is biased toward whatever time is earlest.	(2) 4.5 h is based on the orginal Speilman formulation. Less experienced clinicians should not go below 5 h	(2) (Personal Communication). PTIB is generally not set to less than 6 h	(2) 14 days is reccomended with the caveat that more may be needed with patients with highly variable sleep schedules
			(3) Using a lower threshold (e.g., 85% [increase]) may allow for more opportunities for increased TST and exposure opportunities for setbacks.	(3) Two sessions are clearly delineated but there appears to be an allowence for more sessions as needed.	(3) Rounded to the nearest quarter hour
				(4) (Personal Communication). The initial Rx is fixed for 1 month.	(4) This element is not specified in the text (delination of the procedures) but examples are given for both TTB and TOB
				(5) (Personal Communication). Generally acccomplished using SE% (< 85 (decrease) 85–90 (stick) > 90 (increase)). But this may vary by patient.	(5) 30 min is allowed based on elevated ESS scores.

intervals; (3) lower SE% criteria; and/or (4) greater than 15 min increases when conducting upward titration (e.g., see Table 1). Finally, please bear in mind that the above-described procedures are only half of what it takes to deliver SRT. The other half pertains to how the prescription is delivered (i.e., how it is explained to the patient to garner their buy-in). The metaphors, examples, exercises, and dialogs are what make the delivery vehicle as important as the prescription itself. For examples of therapist "deliveries," the reader is referred to the various treatment manuals (Edinger & Carney, 2014; Manber & Carney, 2015; Morin & Espie, 2007; Perlis et al., 2006).

Stimulus control therapy (SCT)

Stimulus Control procedures, while a standard behavioral method, were first applied to the problem of insomnia by Richard Bootzin in 1972 (Bootzin, 1972). The goal of the treatment is to reverse the patient's tendency to stay in bed and "rest" and/or to engage in nonsleep behaviors in the bedroom or during the sleep period. Such practices are thought to lead to stimulus dyscontrol (the reduced probability that sleep will occur when and where the patient wants). Put differently, SCT is based on learning principles and the idea that one stimulus may elicit a variety of responses depending on the conditioning history. In good sleepers, the environmental stimuli typically associated with sleep (e.g., bed, bedroom, etc.) are paired with the response of sleep. In patients with insomnia, sleep-related stimuli become paired with activities other than sleep, such as reading, watching television, and most importantly, lying awake in bed. Engaging in these other behaviors while in bed contributes to maladaptive conditioning and further strengthens the association between one's bed and *wakefulness*. In other words, the bed and the bedroom become cues for wakefulness and frustration (which likely exacerbates sleep onset and maintenance problems). Accordingly, the treatment is to limit the time spent awake and the activities engaged in when the individual is awake at night. This is done by following a simple set of rules (as below).

1. Lie down intending to go to sleep only when you are *sleepy*.
2. Do not read or watch television in the bedroom.
3. If you find yourself unable to fall asleep, get up and go into another room. Stay up *as long as you wish* and then return to the bedroom to sleep.
4. If you still cannot fall asleep, repeat Step 3. Do this as often as is necessary throughout the night. The above rules were to form the basis for the development of permanent sleeping habits. Thus, the same rules were to be observed even after success was achieved.

Please note the above rules are the original version of Stimulus Control instructions. As with SRT, these rules have been modified over time. The rationale for such modifications, while not based on evidentiary criteria, have been altered based on logistical and/or theoretical considerations. Logistically speaking, Rule 1, in the context of CBT-I (vs. SCT as a monotherapy) is no longer relevant because *Time to Bed* (TTB) is a prescribed element of SRT. Theoretically speaking, the admonishments to (1) adopt a 15-min rule for when to implement SCT and (2) self-monitor for "sleepiness" as a que for when to return to bed may be less than optimal approaches. The former may prompt "clock-watching," and this may be anxiogenic. The latter may fuel sleep effort and cause the patient to "under dose" SCT. Finally, it has been suggested that SCT, while unquestionably efficacious, may work via mechanisms not related to, but compatible with, correcting stimulus dyscontrol. One alternative explanation is that SCT prevents microsleeping (sleep episodes that are not perceived as sleep but when they occur diminish sleep pressure) and enforces full wakefulness at a time of day where this may result in "twice the prime" for subsequent sleep (Klerman et al., 2019). These perspectives have led some to formulate versions of SCT that do not counsel the patient to engage in sedentary/nonactivating activities while practicing SCT.

Finally, while it is clear that the mechanism of action for, and the delivery of, SCT is more nuanced than one might expect, it remains the case that sharing the rules with the patient is only half of what constitutes good treatment. The other half pertains to how the prescription is delivered (i.e., what problem is addressed by SCT, how this applies to the specific patient, how it is to be implemented, what problems the patient is likely to encounter, etc.). As above, the metaphors, examples, exercises, and dialogs are what make the delivery vehicle as important as the prescription itself (i.e., the pill capsule is as important as the medication it contains). For examples of therapist "deliveries," the reader is referred to the various treatment manuals (Edinger & Carney, 2014; Manber & Carney, 2015; Morin & Espie, 2007; Perlis et al., 2006).

Cognitive therapy (CT)

Cognitive therapy for insomnia includes at least three specific exercise-based procedures, including the disputation of dysfunctional beliefs, decatastrophization, and "behavioral experiments" (Perlis, Aloia, & Kuhn, 2010). These are briefly described below.

Disputing dysfunctional beliefs is a psychoeducational procedure where faulty beliefs are identified and addressed. For example, "everyone needs 8 h of sleep and if one fails to achieve this, their next day function will be significantly compromised." Using this form of CT,

one would share what is known empirically to make the points that "how much sleep one needs and how one functions given that they don't meet their sleep need" varies dramatically from person-to-person (e.g., the normative range for sleep duration ranges from 7 to 9 h and there are many people who suffer no next-day effects with 5–6 h of sleep).

Decatastrophization is a dyadic exercise for dealing with the projected extreme consequences of dysfunctional beliefs. For example, "if I don't sleep 8 h tonight, I will [*suffer some terrible thing*] tomorrow." The exercise requires that the therapist and the patient (1) review how long the patient has had insomnia (and tabulate how many affected days); (2) identify the catastrophic thoughts that come to mind for the patient when they are sleepless (e.g., I will wreck my car, lose my job, strike someone in anger, have a heart attack, etc.); (3) assign a value to how certain they are that such an outcome will occur when they are sleepless and "think the thought"; (4) determine the actual frequency of occurrence of the anticipated "catastrophes"; and (5) calculate the frequency rate that corresponds to the certainty estimate. These data are used to show the patient that there is a huge mismatch between the patient's level of certainty regarding negative outcomes and their actual occurrence. This ultimately allows for a discussion of how it is that one is prone to such overestimates and what to do when such intrusive thoughts come to mind.

Behavioral Experiments are conducted to gather the data needed to challenge dysfunctional beliefs and/or catastrophic thoughts. Thus, the method may be behavioral, but the intent is cognitively focused (address anxiogenic sleep preventing thoughts). For example, if the patient believes and/or has the recurrent thought "when I don't sleep, I look dreadful, and the change in my appearance will be obvious to everyone." One possible behavioral experiment is to gather daily photographs (standardizing the time of day and conditions for the photograph). The photographs (which should be day and time-stamped) then can be reviewed at the next session where the therapist asks the patient to identify which picture was taken following their worst nights' sleep. Typically, most people cannot choose a specific photo (because they all look similar) or they choose a photo that does not correspond to the morning following their worst sleep (Harvey & Talbot, 2010; Harvey, Tang, & Browning, 2005).

Sleep hygiene (SH)

The earliest mention of sleep hygiene was by Nathaniel Kleitman in 1939, where he presented and reviewed the evidence regarding sleep satiety, body position, bedtime rituals, sleep surface, and ambient temperature (Kleitman, 1939). His work, however, does not resemble the "Do's and Don'ts" of contemporary SH instructions. In fact, it was not until

1977 that Peter Hauri formally codified this intervention (Hauri, 1977). Hauri expanded on the ideas of Kleitman by suggesting that SH should include information on lifestyle behaviors, relaxation, and environmental factors. The Hauri formulation was intended to offer rules to follow that would ameliorate sleep initiation and maintenance problems.

SH usually includes a one-page handout that outlines various methods to decrease the risk of having "a bad night's sleep" (e.g., limit caffeine and alcohol use, napping, and exercise before bed, etc.). SH is thought to be most useful when it is explained in full, where the patient's understanding is reflected to the therapist and a specific and agreed-upon plan is put into place. Ideally, within the context of CBT-I, SH should focus solely on sleep hygiene issues as opposed to adopting proxy rules for SRT and SCT. The following 10 rules (imperatives and rationales) are offered as optimal (see Table 2).

Please note, it is generally held that sleep hygiene is not effective as monotherapy. This common belief may be a bit of overgeneralization from the existing facts that are more consistent with the 2006 AASM Standards of Practice Committee's conclusion that there is "insufficient evidence to recommend sleep hygiene as a single therapy" (p. 221) (Morin, Culbert, & Schwartz, 1994). In sum, the most reasonable conclusion about sleep hygiene is this: it is a useful addition to CBT-I in that engaging in improved sleep hygiene may enhance outcomes or at least remove obstacles to progress.

Efficacy and effectiveness of CBT-I

There is an overwhelming preponderance of the evidence that in-person CBT-I is effective (Irwin, Cole, & Nicassio, 2006; Koffel, Koffel, & Gehrman, 2015; Miller et al., 2014; Montgomery & Dennis, 2003; Murtagh & Greenwood, 1995; Okajima, Komada, & Inoue, 2011; Pallesen, Nordhus, & Kvale, 1998; Perlis et al., 2006; van Straten et al., 2018), as effective as sedative-hypnotics during acute treatment (4–8 weeks), and is more effective than sedative-hypnotics in the long term (e.g., 3+ months following treatment) (Rios et al., 2019; Smith et al., 2002). This overall profile, along with CBT-I's low propensity for side effects or harm, is likely what prompted the American College of Physicians to recommend that CBT-I be considered the first-line treatment for chronic insomnia (Qaseem, Kansagara, Forciea, Cooke, & Denberg, 2016).

Pre-to-Post Treatment Change (Overall). In terms of symptom reduction (pre-post treatment change), subjective sleep latency (SL) and wake after sleep onset (WASO) times are reduced from baseline averages of about 60 min to 35 minutes at treatment end. These absolute changes corresponded to average treatment effects of about 50% reductions in symptom

TABLE 2 Sleep hygiene.

1. Exercise in the early evening.
Rationale: There is evidence to suggest that exercise in the early evening allows one to fall asleep more quickly, stay asleep for longer and to sleep more deeply.
2. Do not take naps during the day.
Rationale: Napping during the day decreases one's overall "pressure" to sleep at bedtime, and therefore, the overall chance that one will fall and/or stay asleep [at bedtime]. There are caveats for "safety naps" and those with medical illness (e.g., cancer), and in those situations, the patient should be instructed to nap in bed, with an alarm, and for a maximum of 30 min
3. Eat a light snack before bedtime.
Rationale: Eating a light carbohydrate snack one hour before bedtime (e.g., crackers or fruit) may help improve sleep quality.
4. Avoid liquids before bedtime.
Rationale: Increased liquids before bedtime may increase the chance that one has to wake-up to use the restroom.
5. Avoid caffeinated products ~6 h before bedtime.
Rationale: Although stimulants, like caffeine, don't have to be entirely discouraged and may even improve the chances that one functions better during the day, caffeine's biological half-life is on average about 6 h and therefore consuming caffeine within 6 h of bedtime may increase sleep latency and/or wake after sleep onset problems.
6. Avoid nicotine.
Rationale: Like caffeine, nicotine's stimulant properties may impede sleep continuity.
7. Use ear plugs and/or white noise to mitigate outside noise.
Rationale: Outside noise can be disruptive to sleep, using earplugs may improve the chances that one falls and/or stays asleep.
8. Avoid co-sleeping with pets.
Rationale: Co-sleeping can be disruptive to sleep, thus removing pets from the bed at night may be helpful in the long term.
9. Make sure the sleep environment is conducive to sleep (e.g., bedding, temperature, light).
Rationale: A comfortable bed, temperature and minimal light has been shown to be important for adequate sleep. Thus, assessing these items with the patient will be helpful to better understand whether these factors may be contributing to their sleep problems.
10. Keep a regular sleep schedule (e.g., set an alarm).
Rationale: Keeping a regular sleep schedule decreases the chance for sleep extension and increases the chance for a match between sleep ability and opportunity, which is a core component of CBT-I.

severity and pre-to-post effect sizes of about 1.0 (Murtagh & Greenwood, 1995; Smith et al., 2002). Please note, Early Morning Awakenings (EMA) are generally collapsed into WASO measures, and therefore, little is known about the effects of CBT-I on EMA. In the context of acute treatment, total sleep time (TST) is minimally affected during 6–8 session CBT-I with only about 45% of patients exceeding baseline TST at treatment end (Perlis et al., 2019). Such outcomes generally correspond to mean changes of <30 min and <10% pre-post change with within-subject effect

sizes of < 0.5. When assessed in terms of pre-post insomnia severity (e.g., the Insomnia Severity Index (Bastien, Vallières, & Morin, 2001)), the within-subject and between-subject effect sizes are reliably larger than single measures of sleep continuity effects, with within-subject effects sizes of around 2.0 (Smith et al., 2002). This is not surprising as the ISI not only assesses illness severity (magnitude of sleep continuity disturbance) but also the degree of insomnia-related daytime impairment/complaint. When evaluated in terms of the percent of patients who exhibit treatment responses (variably defined), between 70 and 80% of patients achieve a therapeutic response during acute treatment (Beaulieu-Bonneau, Ivers, Guay, & Morin, 2017; Morin & Benca, 2012; Morin, Colecchi, Stone, Sood, & Brink, 1999).

Durability of CBT-I Effects Over Time. In what are perhaps the first long-term RCTs of CBT-I, it was found that SL and WASO effects are remarkably stable over time periods of up to 24 months (Beaulieu-Bonneau et al., 2017; Morin et al., 1999; Morin et al., 2009; Morin & Benca, 2012). This is to say that clinical gains are maintained for months and years *after treatment is discontinued*. Interestingly, TST effects (which are initially marginal) appear to accrue with time. That is, when followed longitudinally, patients exhibit an average increase in TST of about 50 min. These gains do not appear to be related to additional improvements in SL and WASO, but instead are likely to be related to increased time in bed while maintaining good sleep efficiency. When evaluated in terms of percent of patients who exhibit remission, 50%–60% of treatment responders achieve remission in the 6–12 months that follow therapy. Similar findings were recently presented for a large-scale clinical case series study (Castronovo et al., 2018). In this study, mean ISI values from end-of-treatment (T1) to follow-up (T2 [4–10 years]) were found to be remarkably stable (baseline ISI 17.1 ± 4.5, $T_1 = 9.7 \pm 4.6$, and $T_2 = 9.9 \pm 6.3$). In contrast to these studies, a recent meta-analysis on the durability of CBT-I showed that CBT-I continues to be effective at 3, 6, and 12 months as compared to nonactive controls, but that the clinical gains in the active treatment group appeared to decline over time (van der Zweerde, Bisdounis, Kyle, Lancee, & van Straten, 2019). While these differences in claims remain to be reconciled, it may be the case that the meta-analytic findings differ owing to between-study differences in the application of CBT-I. If protocol differences yield different long-term outcomes, and the individual studies summarized here used the most robust methodologies, then the durability outcomes in the meta-analysis may represent the inclusion of studies with small magnitude outcomes and/or greater variability in the effect size estimates. Given the absence of end of treatment effect sizes in the meta-analysis, one cannot be sure if such data were comparable to the single studies summarized here or other meta-analytic studies regarding the acute effects of CBT-I.

The Efficacy of "Real World" CBT-I (RCTs vs. Clinical Case Series Studies). The conclusions that can be drawn from RCTs are often subject to some skepticism as the most that can be said is that RCT outcomes represent the best-case scenario. This perspective is rooted in the reasonable belief that RCTs are populated by exceptionally healthy patients (individuals that have the illness of interest but little else in the way of comorbidity) while in-clinic patients often have complex medical, psychiatric, and psychosocial profiles and therefore may be (understandably) less responsive to targeted treatments. In perhaps the first evaluation of this idea, clinical case series data were obtained on 47 subjects (Perlis et al., 2000). In this analysis, it was found that, on average, subjects who completed at least a minimum dose of CBT-I ($\geq$4 sessions) experienced about a 23-min decrease in SL ($d=1.00$), a 39-min decrease in WASO ($d=1.09$), and a 20-min increase in TST ($d=0.36$) (Perlis, Sharpe, Smith, Greenblatt, & Giles, 2001). Note, while it stands to reason that the sum of SL+WASO (i.e., total wake time or TWT) decreases should equal TST gains, this is often not true, and for two reasons. First, with sleep restriction, the change in TIB limits potential gains in TST. Second, patients rarely calculate TST but instead provide their impression of how much sleep was obtained. That is, given TIB and SL, WASO, and EMA, it is possible to estimate TST in a manner that is not a "guesstimate", but instead may be arithmetically calculated, and in a manner that is internally valid (i.e., TST = TIB − [SL+WASO+EMA]). Given these two considerations, it is rarely the case that the decrease in TWT equals the increase in TST. With this caveat in mind, these data are clearly comparable to the "meta-analytic norms", if not significantly better. In sum, it may indeed be the case that "real world" patients benefit from CBT-I to the same extent as subjects in clinical trials, perhaps more. This surprising result may be ascribable to a variety of factors including better outcomes due to professional therapists, the tailoring of the clinical treatment regimen to the individual case, and/or a variety of other nonspecific factors such as cognitive dissonance (e.g., paying for treatment vs. no payment or being paid to be treated).

Efficacy of CBT-I in "Secondary Insomnia" (Comorbid Insomnia). By and large, the effects summarized above are from foundational RCTs that were undertaken from 1990 until the early 2000s. The overwhelming majority of these trials were conducted in subjects with "Primary Insomnia" (i.e., individuals without comorbid illness that includes insomnia as a symptom). This was the case because insomnia was construed as both a symptom and a disorder, and it was believed that so-called secondary insomnia could only be effectively treated with therapies for the primary disorder (Zorick & Walsh, 2000). For example, insomnia occurring in the context of chronic pain would only be ameliorated to the extent that the analgesic therapy was successful. Implicit in this perspective was that direct treatment of "secondary insomnia" with CBT-I (i.e., by targeting behavioral

factors) would yield little to no positive effects, as pain (not sleep extension) was considered to be the perpetuating factor for that form of chronic insomnia. In the last two decades, literally dozens of CBT-I RCTs have been conducted in patients with "secondary insomnia" (including but not limited to patients with depression (Carney et al., 2017; Cunningham & Shapiro, 2018), bipolar disorder (Harvey et al., 2015), PTSD (Simon et al., 2019), generalized anxiety disorder (Ye et al., 2015), schizophrenia (Hwang, Nam, & Lee, 2019), cancer (Ma et al., 2020), heart failure (Redeker et al., 2018), chronic pain (McCrae et al., 2019), Alzheimer's disease (Siengsukon et al., 2020), multiple sclerosis (Siengsukon, Alshehri, Williams, Drerup, & Lynch, 2020), alcoholism (Chakravorty et al., 2019), chronic obstructive pulmonary disease (Kapella et al., 2011), obstructive sleep apnea (Ong et al., 2020), period limb movement disorder (Edinger et al., 1996), etc. To our knowledge, all of these studies found that CBT-I was effective. Most studies found that the treatment outcomes were similar to those observed in patients with primary insomnia, and several studies found superior treatment outcomes (Carney et al., 2017; Chakravorty et al., 2019; Cunningham & Shapiro, 2018; Edinger et al., 1996; Harvey et al., 2015; Hwang et al., 2019; Kapella et al., 2011; Ma et al., 2020; McCrae et al., 2019; Ong et al., 2020; Redeker et al., 2018; Siengsukon, Alshehri, et al., 2020; Siengsukon, Nelson, et al., 2020; Simon et al., 2019; Ye et al., 2015). At least one clinical case series study (Perlis et al., 2000) was conducted on this topic and there have been several meta-analyses. Geiger-Brown and colleagues (Geiger-Brown et al., 2015) summarized 37 studies (1379 subjects), where SL decreased by 26 min ($d = 0.85$), WASO decreased by 36 min ($d = 0.92$), and TST increased by 29 min ($d = 0.43$). Moreover, there was a 13-point improvement in SE ($d = 1.20$) and about a 10-point decrease in ISI ($d = 2.1$). These treatment effects were found to be durable for up to 18 months post-treatment. Not only do these data demonstrate the efficacy of CBT-I with insomnia in the context of other comorbidities, but there is now an accumulating evidence base to show that treating insomnia with CBT-I has a beneficial "halo" effect on other medical and behavioral health disorders. For example, CBT-I as an augmenting strategy to the medical management of depression has been found to double antidepressant treatment response and to reduce suicidality by half (Manber et al., 2008; Trockel, Karlin, Taylor, Brown, & Manber, 2015). A similar set of outcomes were found for iCBT-I as compared to iCBT-D (Blom, Jernelöv, Rück, Lindefors, & Kaldo, 2017), where only insomnia treatment produced positive insomnia outcomes, but both forms of iCBT produced positive depression outcomes. Despite secondary insomnia data showing changes in sleep continuity that are comparable to those found in primary insomnia, the effect sizes tend to be smaller. The reason for this may be, as mentioned above, that the meta-analyses contain studies that are not "best in class" (studies conducted by established experts with best methods and/or professional

therapists). This issue aside, it may be that there are fewer treatment responders but the response itself is of equal magnitude to those found in primary insomnia. This may speak to issues of adherence in patients with other comorbidities (e.g., those diagnosed with cancer may focus on cancer and not insomnia as the primary issue). In other words, it may be harder to garner adherence day-to-day and week-to-week in patients who have other significant areas of concern. This may, in turn, account for lower treatment responder rates. Note: These data support the notion that standard CBT-I should be adapted in order to enhance treatment responder rates in specific clinical populations. Which populations require this and what adaptations should be made remains to be empirically determined.

Common alternative rules & therapies

Common alternative rules

Sleep Restriction. In a recent and seminal work by Kyle and colleagues (Kyle et al., 2015), it was shown (in the context of RCTs) that SRT is rarely delineated component-by-component in the methods sections of such investigations; and when details are provided they often vary from study to study.

Stimulus Control. There are a variety of ways that SCT has been modified, relative to its core components. In the absence of a formal study of its variants, a cursory review of the rules as they are presented in three treatment manuals suggests that the application of SCT is also highly variable.

In Tables 1 and 3, the rules for SRT and SCT are listed from the four published treatment manuals. It is interesting to note for SRT, there is no particular rule for which there is unanimous agreement.

Alternative variants or adjuvants to SRT and SCT

Counter-Control. This particular variant of SCT was originally developed as a comparator condition for the empirical evaluation of the mechanism of action for stimulus control. More specifically, it was developed to be a "counter control"; the exact opposite of SCT instructions. If the central element of SCT is to be awake somewhere other than the bed and bedroom, the counter condition is to intentionally be awake in the bed and bedroom (both during the sleep period and for 30 min a day when not intending to sleep). In what is now a classic study by Zwart and Lisman (1979b), it was shown that SCT did not significantly differ from the counter control condition (i.e., both conditions worked about equally well to ameliorate sleep onset problems) (Zwart & Lisman, 1979b). Given

TABLE 3 Stimulus control therapy (SCT) rules based on the first four published treatment manuals.

	Manual			
Issue	**Morin and Espie (2007)**	**Perlis et al. (2006)**	**Edinger and Carney (2014)**	**Manber and Carney (2015)**
When to get out of bed?	After 15 min of wakefulness (1)	After 15 min of wakefulness (1)	After 20 min of wakefulness	When feeling alert (1)
Where to go?	Out of bedroom	Out of the bedroom	Out of the bedroom	Out of bed
What to do when out of bed?	Not specified (2)	Anything enjoyable (2)	Not specified (1)	Things that are not activating (2)
When to return to bed?	When sleepy	When sleepy (3)	When sleepy	When sleepy
	(1) The 15 min rule is to be applied to both SL and WASO. In regards to WASO, the manual suggests that patient's go back to bed "when sleepy again" but to wait no longer than 15 min to try to go back to sleep.	(1) The 15 min rule is acknoweldged as the standard but it is reccomended that, in an effort to prevent "clock watching" behavior, patients should leave the bedroom as soon as they feel "clearly awake" or are annoyed at being awake.		(1) In order to discourage clock watching, it is recommended that patients use their own sensation of alertness, to determine when they should get out of bed or go back to the bedroom.
	(2) While no rule of thumb or potential lists are given, it is clear from the dialogs that a plan should be enacted with each patient.	(2) A later development to this protocol was the stipulation that the behavior must be something that can be done during the day (i.e., not a prohibited behavior) or a behavior that reinforces wakefulness.		(2) In addition to the list of activites provided in the manual, it is recommended that the therapist and the patient work to collaboratively make a list of things to do when out of the bedroom.
		(3) A later development to this protocol was a modifcation to this instruction to have the patient simply pick a time frame to be awake (30, 60, or 120 min) and then to return to bed, sleepy or not.		

these findings, Davies and colleagues evaluated counter control as a therapeutic intervention for individuals with sleep maintenance problems, with an end goal of producing a more practical therapy for older adults (Davies, Lacks, Storandt, & Bertelson, 1986). While they did not find a "by age" effect for efficacy, counter control was again found to be effective, producing about a 30% improvement at the end of treatment, with gradual improvement continuing through a 4-week follow-up period.

Many understood these results to be a substantial challenge to the belief that SCT works via instrumental conditioning or by increasing the associational strength between sleep-related stimuli and sleep. While still a controversial issue, the manipulation may be successful owing to the instruction to "sit up and do something (other than trying to sleep)". This instruction still encourages a separation between sleep stimuli and sleep based on body position and the prescription has within it a component that derails sleep effort. In the final analysis, and irrespective of whether SCT works based on good stimulus control, the success of counter control instructions has prompted clinicians to adopt this strategy in patients that (1) cannot, or cannot safely, get up and out of bed at night or (2) live in dwellings, or under circumstances, that do not allow them to leave the bedroom and to be awake elsewhere.

Sleep Compression. Perhaps one of the first and most innovative adaptations to CBT-I came with the proposal to substitute sleep compression for sleep restriction. While the alternative therapy was explicitly proffered for patients with sleep continuity disturbance *without daytime sequelae* (i.e., short sleep patients with normal to high sleep opportunity and low sleep need and ability), it has become common (at least in clinical practice) to use this procedure for patients that are resistant to (and/or nonadherent with) sleep restriction. The method is comparable to sleep restriction in that sleep opportunity is matched to sleep ability (i.e., step 1 of therapy is to match average TIB to average TST). Unlike sleep restriction, where the "reset" is accomplished on night one, during sleep compression the reset is achieved over a series of weeks. In the original formulation of this treatment, therapy was discontinued when one reached the target SE%. There was no need for systematic sleep extension because it was assumed that the participant had achieved their true sleep need. In more recent iterations, as applied to patients with insomnia disorder, sleep extension remains a component of this treatment regimen. Lichstein and colleagues first demonstration of this method was in a sample of 74 older adults (59 years or older) (Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001). Subjects were randomized into three groups, relaxation ($n=27$), sleep compression ($n=24$), or placebo desensitization ($n=23$). The sleep compression condition fared better than placebo, where SL decreased by about 12 min ($d=0.48$), WASO by about 24 min ($d=0.75$), SE increased by about 8 points ($d=0.55$), and TST decreased by about 14 min ($d=0.20$). This study

provides preliminary evidence that sleep compression is effective for short sleepers who have low levels of daytime impairment.

Mindfulness. It can be argued that the single most influential change to CBT-I in the last two decades has been the adoption of mindfulness training. Mindfulness was first introduced, in the context of insomnia, by Ong, Shapiro, and Manber (2008). The stated goal for the adjuvant therapy was to better address sleep-related cognitive arousal. The approach, however, substantially differs from traditional cognitive therapy in that it is not focused on disputing, derailing, or disengaging worry or intrusive negative thoughts. Instead, mindfulness is focused on the nonjudgmental observation of one's cognitions, with the desired goal of changing one's relationship to their thoughts as opposed to fighting with them. In this way, the process encourages "acceptance." Ong et al. (2008) conducted a treatment-development study (using a simple pre-post design) to evaluate the effectiveness of mindfulness meditation as an adjuvant to six-session in-person CBT-I ($n=30$). Significant pre-post-treatment reductions were found in the study's primary outcome variable, total wake time (TWT [SL+WASO+EMA]). The corresponding pre-post effect size for this variable was -1.17. Significant reductions were also found in secondary analyses of the single component measures, including an 18-min decrease in SL ($d=-0.84$), 25-min decrease in WASO ($d=-0.62$), and a 5-min decrease in TST ($d=-0.11$). Improvements were also found in SE and the ISI, where SE improved by approximately 9 points ($d=-1.1$) and the ISI decreased by 5 points ($d=-1.32$). A follow-up study showed that these sleep-related benefits were maintained over a 12-months post-treatment (Ong, Shapiro, & Manber, 2009).

Dual Therapy with Medication. There have been a variety of RCTs that evaluate how treatment outcomes vary when sedatives (e.g., temazepam or zolpidem) are prescribed in conjunction with CBT-I (Beaulieu-Bonneau et al., 2017; Morin, 2006; Morin et al., 2009; Morin et al., 2014). Combined treatment and monotherapy with CBT-I have been found to produce equivalent gains in the short term (during acute treatment), that is, similar gains with respect to sleep initiation and maintenance (diary measures of SL, WASO, and TST), similar gains on multifactorial measures of insomnia severity (ISI), and similar percentages with respect to the subjects that achieve treatment responses. In contrast, there is some evidence that long-term durability of CBT-I may be attenuated in patients cotreated with hypnotics and/or they tend not to exhibit the increases in total sleep time that occur with CBT-I following treatment discontinuation (Perlis et al., 2001). Given this profile, one might wonder under what circumstances combined treatment might ever be indicated? The possibilities include: (1) when the time to a treatment response needs to be accelerated (Morin et al., 2014) and/or (2) when the short-term iatrogenic effects of CBT-I need to be blocked or attenuated (Perlis & Smith, 2008). Some have

argued that one or both of these considerations can be better managed with the short-term use of stimulants (e.g., modafinil qam or bid along with CBT-I [1st four sessions of SRT]) (Cheung, Bartlett, Armour, Laba, & Saini, 2018; Perlis et al., 2004). While there is some data to suggest that this strategy blocks iatrogenic sleepiness[77], there is no evidence that cotreatment with stimulants speeds the latency to treatment response or maintains the long-term benefits of monotherapy with CBT-I. Clinically, both strategies seem to be reasonable alternative approaches for the management of insomnia, when indicated.

Conclusion

In the absence of data regarding which clinical populations or demographic groups *require modified CBT-I*, it seems premature to recommend that standard CBT-I be altered. In an ideal world, this issue would be addressed systematically.

> First, demonstrate a need (i.e., standard CBT-I is not as effective for whom), conduct research into likely causes (factors associated with reduced efficacy or treatment nonresponse), and then develop and test adapted versions of CBT-I that address the factors for whether or not they produce superior outcomes as compared to standard CBT-I.

Given that such work is not fundable in the present climate, perhaps the next best approach is to develop alternative rules and therapies that can be adopted by clinicians on an as-needed basis. In this case, clinicians could decide to adopt nonstandard therapy a priori by asking and answering the question "Is there anything about the comorbid conditions or life circumstances of a given patient that would interfere with, or be worsened by, the conduct of CBT-I?"(Smith & Perlis, 2006). While one should be conservative about the need to adapt standard CBT-I, "interference" may be managed by using alternative forms of CBT-I, forms that are more compatible with the patients' status. For example, if the patient is immobile or has limited mobility, one may opt to go forward with treatment but to use "counter control" instructions (Davies et al., 1986; Zwart & Lisman, 1979b) rather than usual stimulus control instructions. "Worsening" (CBT-I may aggravate one or more of the comorbid conditions) is a more serious consideration, but perhaps less so than many think. On the one hand, there is the legitimate concern that (for example) sleep deprivation may trigger certain conditions such as parasomnias, manic episodes, and/or seizures in patients predisposed to, or diagnosed with, such problems. The counterpoint to this is that (1) sleep restriction is not sleep deprivation, and it is the latter that is the known risk factor for such adverse events (note: sleep restriction usually entails between 1 and 2 hours' sleep loss

while sleep deprivation entails between 4 and 8 h sleep loss); (2) if the comorbid disorder is stable owing to ongoing treatment, it is not a given that even sleep deprivation can trigger adverse events in such cases; and (3) CBT-I is a short duration intervention (usually 4–8 weeks), and thus, the window of vulnerability is narrow. If the risks of potential sleep loss in CBT-I are of concern, one may opt to not conduct CBT-I, to conduct CBT-I without the sleep restriction component, or to use sleep compression. Ultimately, the following chapters in this book can serve as a menu for the options that might be engaged, if the clinician is certain that standard CBT-I needs to be adopted for a given patient. The trick, or the art of assessment, will be to know when such adaptations are necessary versus when such adaptations are unwarranted or premature and may only serve to delay or attenuate therapeutic gains.

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ADAPTING COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA

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PART 1

Traditional CBT-I components and delivery