Title

Comparing internet-delivered cognitive therapy and behavior therapy with telephone support for insomnia disorder: a randomized controlled trial.

Journal

SLEEP, 43(2)

Authors

Sunnhed, Rikard

Hesser, Hugo

Andersson, Gerhard

et al.

Publication Date

2020-02-13

DOI

10.1093/sleep/zsz245

SLEEPJ, 2020, 1–15

doi: 10.1093/sleep/zsz245
Advance Access Publication Date: 14 October 2019
Original article

ORIGINAL ARTICLE

Comparing internet-delivered cognitive therapy and behavior therapy with telephone support for insomnia disorder: a randomized controlled trial

Rikard Sunnhed[1,*], Hugo Hesser[2], Gerhard Andersson[2,3], Per Carlbring[1], Charles M. Morin[4], Allison G. Harvey[5] and Markus Jansson-Fröjmark[3]

[1]Department of Psychology, Stockholm University, Stockholm, Sweden, [2]Department of Behavioural Sciences and Learning, Linköping University, Linköping, Sweden, [3]Centre for Psychiatry Research, Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden, [4]School of Psychology, Université Laval, Québec, Canada and [5]Department of Psychology, University of California, Berkeley, USA

*Corresponding author. Rikard Sunnhed, Department of Psychology, Stockholm University, 106 91 Stockholm, Sweden. Email: rikard.sunnhed@psychology.su.se.

Abstract

Study Objectives: Our aim was to compare the effects of Internet-delivered cognitive therapy (CT) and behavior therapy (BT) against a waitlist (WL) condition to better understand their unique contribution in the treatment of insomnia.

Methods: Two hundred and nineteen participants with insomnia disorder were randomized to CT (n = 72), BT (n = 73), or WL (n = 74). The treatment arms consisted of 10 weekly internet-delivered modules with 15 min of telephone support per week. At pre, post, and follow-up, participants completed measures of insomnia severity, sleep diaries, functional impairment, anxiety, depression, quality of life, adverse events, satisfaction and perception of content, workload, and activity in treatment. Measures of completed exercises, modules, therapist support, and platform logins were also measured at posttreatment.

Results: Moderate to large effect sizes for both CT and BT outperformed the WL on the majority of outcomes, with significant differences in favor of both therapy groups. Both treatment groups had significantly larger proportion of treatment remitters (CT: 35.8%, BT: 40%, WL: 2.7%) and responders (CT: 74.6%, BT 58.6%, WL: 10.8%) compared to the WL at posttreatment. There were no significant differences between the two therapy groups in terms of outcomes, except for sleep onset latency in favor of BT (6 min difference at posttreatment) and adverse events in favor of CT (CT 14.1% vs BT 43.2%).

Conclusions: This study indicates that both Internet-delivered CT and BT are effective as stand-alone therapies for insomnia disorder. Results highlight the need for examining which therapy and subcomponents that are necessary for change.

ClinicalTrials.gov Identifier: NCT02984670

Statement of Significance

The unique contribution of this study is that both cognitive therapy and behavior therapy (the main therapies in CBT-I) are compared against a waitlist (WL) condition, that the therapies are internet-delivered, and a wide range of measures are included, e.g. adverse events and treatment activity. Both therapies were significantly more effective than the WL condition and only differed significantly on one outcome (sleep onset latency), thus indicating that both cognitive therapy and behavior therapy are effective as stand-alone therapies. These findings pave the way for health care to more flexibly choose between one of the therapies for treating insomnia. Future research is still needed on the unique efficacy of specific CBT-I components and what moderates and mediates the effects of both therapies.

Key words: behavior therapy; cognitive therapy; cognitive behavior therapy; insomnia; internet-delivered

Submitted: 13 December, 2018; **Revised:** 19 August, 2019

Introduction

Difficulties initiating, returning to, or waking up too early from sleep, in combination with daytime consequences (worry or functional impairment), are the main symptoms experienced by people suffering from insomnia disorder. Insomnia disorder affects 6%–10% of the population [1, 2] and is for the individual associated with a number of negative effects (e.g. psychological distress, decreased daytime functioning, higher sick leave) [3–6], which, together with the fact that insomnia tends to remain if untreated [7], highlights the importance of effective treatments for the condition. Cognitive Behavioral Therapy for Insomnia (CBT-I) has a solid empirical base and is considered the treatment of choice for insomnia since it has both short- and long-term effects [8, 9]. However, although CBT-I is thoroughly studied, little is still known about the unique effects of its main therapies, behavior therapy (BT) and cognitive therapy (CT) [1, 10]. This is a critical knowledge gap because CT and BT represent two distinct therapeutic approaches, with fairly distinct theoretical underpinnings and techniques, which hypothetically also suggest unique effects on treatment outcomes. Comparative examinations of the main therapies in CBT-I could be one way to gain knowledge about potential unique effects of CT and BT, as well as under- or over-targeted maintaining factors in CBT-I, which subsequently could aid in optimizing the effectiveness of CBT-I.

Behavioral therapy and cognitive therapy thus represent two distinct approaches to managing insomnia that builds on separate theoretical grounds. BT, on the one hand, is based on two biological theories or models of sleep: the circadian system and the homeostatic system, which are proposed to regulate our sleep–wake pattern and our drive for sleep, based on interaction with the dark–light cycle, and based on our time spent awake or asleep [11–14]. The aim of BT is to regulate these biological systems for optimal sleep to occur by applying two behavioral techniques: sleep restriction (SR) and stimulus control (SC) [12, 13]. Cognitive therapy (CT), on the other hand, rests on a cognitive model of insomnia [15] that, building on important prior contributions [16–18], proposes that cognitive arousal (worry about sleep and daytime impairment fueled by dysfunctional beliefs) triggers autonomic arousal and distress, which are both viewed as unhelpful for sleep initiation and maintenance, as well as daytime functioning. In the process of coping with this anxious state of insomnia, the individual engages in selective attention to sleep-related threat and safety behaviors to avoid potential sleep difficulties or daytime impairment. Together these processes result in a vicious cycle, leading to misperception of symptoms, more worry, and associated arousal that maintain the insomnia symptoms. CT aims to break this vicious cycle and its effects on sleep and daytime symptoms by targeting the abovementioned cognitive processes. This is achieved by the use of behavioral experiments and the identification and challenging of negative automatic thoughts.

In terms of evidence, research on the relative effects of BT and CT is limited, especially regarding CT and in terms of comparative studies in which both therapies are included as separate arms in a randomized controlled study. The therapy most thoroughly examined is BT, involving SR and SC. BT has in a number of trials demonstrated efficacy and is considered a well-established psychological therapy for insomnia disorder [10]. When it comes to CT, the evidence is more limited [10], with one open trial [19] and more recently one randomized controlled trial (RCT) comparing CT and BT to CBT-I [20], all indicating that CT can be effective. However, the paucity of comparative studies of CT and BT hinders a solid conclusion. Although the dismantling study by Harvey et al. [20] showed effects of both CT and BT, a couple of questions still remain. First, since this was the first study directly comparing CT with BT in the same randomized design, further replication is warranted. Second, although internet-delivered CBT-I for insomnia is well established with comparable effects to face-to-face CBT-I [9], little or no information is available on the unique effects of CT and BT delivered over the internet. Thus, examining the effects of CT and BT delivered over the internet could add further evidence of their unique efficacy across delivery modes. Third, since there was no waitlist (WL) in the former trial, we cannot rule out alternative explanations for the positive effects of treatments including spontaneous recovery, regression to the mean, and effects of measurement. Thus, adding a WL can further add to the current evidence base. Additionally, when contrasting CT against BT, there are important additional outcomes that could elucidate potential differences, such as adverse effects, treatment satisfaction, participants' perception of texts, exercises, and treatment workloads is of interest to more thoroughly contrast these therapies. Hence, further comparative examination of CT and BT is a strategy that subsequently could aid in elucidating how each therapy has an impact on insomnia symptomatology.

Therefore, the aim of this study was to directly compare CT and BT against a WL condition and to examine the differential effects of treatment on a broad range of outcomes related to therapy (expectancy/credibility, completion, satisfaction, treatment dropout, and activity), adverse effects and efficacy on insomnia severity, sleep-diary measures, functional impairment, symptoms of anxiety and depression, and quality of life.

Based on their theoretical underpinnings, we hypothesized that BT's aim is to regulate nighttime processes would result in greater improvements on sleep-diary outcomes relative to CT, and CT's focus on both night and daytime symptoms to be more effective on outcomes related to the day (i.e. functional impairment, anxiety, depression, and quality of life), relative to BT. It was further hypothesized that both therapies would outperform the WL condition.

Methods

Study design and conditions

The study was registered at clinicaltrials.gov, approval number: NCT02984670, as a randomized controlled trial (RCT). The trial was conducted at a university setting in Sweden and included a total of 219 participants randomized into three conditions: two active treatment groups, CT (n = 72) and BT (n = 73), and a WL control group (WL; n = 74). The total number of included participants in this trial was estimated, using a priori analyses with G*Power 3.1.9 [21]. Using a fixed effect mixed design ANOVA for superiority testing comparing all three conditions against each other with three repeated assessments of Insomnia Severity Index (ISI) under standard power conditions (80%, two-tailed alpha 0.05), with an expected study dropout of 24.7% [9], the study was powered to detect an overall time by group interaction effect size of small magnitude (f = 0.1). Thus, the study was sufficiently powered to detect an overall small effect between all

three groups. This trial will be presented in several papers (e.g. outcomes, moderators, and mediators), but the focus of this article will be on comparing outcomes between the groups.

The study was reviewed and approved by the Regional Ethical Board in Stockholm, Sweden (reference number 2016/856–31). Participants gave written informed consent as a digital signature, and all data related to participation were handled through a secure online platform, which ensured anonymity and safety during the whole treatment period [22].

Participants

Participants were recruited through advertisements in the daily press and in social media as well as on an internet platform for ongoing internet CBT studies, from August 2016 to February 2017. To be eligible for this trial, participants had to undergo three screening phases that consisted of a questionnaire on the web, a telephone interview, and the completion of a sleep-diary (7 days). A summary of the flow of participants through the study is presented in Figure 1. The criteria used for inclusion and exclusion in the three screening phases were based on expert recommendation for a standard research assessment of insomnia and The Diagnostic and Statistical Manual of Mental Disorders 5 [23–26].

To be eligible for the study, participants had to register themselves on the Internet platform. The registration included a web-based screening questionnaire that took approximately 30 min to complete, and which served as part 1 out of the 3 screening phases. At this first phase, participants had to meet the following criteria for inclusion: occurrence of sleeping difficulties 3 nights or more per week during at least 3 months despite adequate opportunities for sleep and the following scores on ISI: a total score of 11 or more, a score of at least 2 on one of the three questions regarding nighttime symptoms (items 1–3), and a score of 2 or more on at least one of the two questions regarding daytime deficits (items 5 and 7) [27, 28]. It was further required that participants had time and opportunity to participate in therapy for 10 weeks, read approximately 15 pages per week, and execute homework on a daily to weekly basis. Finally, participants needed to have access to a computer, a cellphone, email, and the Internet. The following criteria were used for exclusion: severe depression (more than 30 points on the self-rated Montgomery-Asberg Depression Rating Scale [MADRS-S]) [29] and high suicidal ideation (4 points or more on item 9 in MADRS-S).

Potential participants eligible for phase 2 were contacted for a 30 min semi-structured telephone interview based on the Duke Structured Interview for Sleep Disorders (DSISD) and the Mini International Neuropsychiatric Interview (MINI) [30, 31] to document sleep and mental disorders. In addition, these interviews aimed to assess and exclude participants if sleeping problems were due to obvious environmental conditions (e.g. pregnancy, small children, animals, or disturbing sounds in the sleep environment), night or rotating shift work (>3 shifts a week), high intake of alcohol (>2 standard drinks a day) or caffeine (>4 beverages a day or >2 after 6 pm), or if participation in CBT-I had occurred within the past 5 years. For inclusion, it was further required that reported somatic conditions were stable and/or that the candidate was receiving treatment for the condition. Insomnia should also be the most disabling and distressing condition, or that insomnia was still present despite treatment for any somatic or psychiatric comorbidity if comorbidities were present. For medication, the following criteria were used: (1) if sleep medication was used, it had to be relatively stable for the last three months, (2) if selective serotonin reuptake inhibitor use was reported, the onset of the medication, or the last change of dosage should be at least three months prior to the telephone interview, and (3) if participants were regularly consuming sleep-disturbing medications, they were excluded. Further criteria for exclusion were participants with a history of psychotic or bipolar disorders and candidates with the following primary sleep disorders: sleep apnea, restless legs syndrome, periodic limb movement disorder, circadian rhythm disorder, and parasomnias.

Those eligible for phase 3 of the screening process were invited, as a final step, to fill in a 7-day sleep diary (for description, see assessment measures below). For inclusion we required at least 3 days of registered sleep difficulties, i.e. initiating, maintaining, or waking up to early, defined as 30 min or more for each occasion.

Participants who met the study criteria were randomly allocated by one of the authors, using randomization data provided by a second author (from an Internet-based random generator www.randomizer.org [Accessed August 15, 2016]) to one of the three groups (CT, BT, or WL). In total, 219 participants diagnosed with insomnia disorder were randomized to CT (n = 72), BT (n = 73), or WL (n = 74). After randomization, each participant received a message with information about his or her group allocation. Those allocated to the active treatment arms were informed that their therapist would contact them within 2 weeks for initiation of therapy. Those allocated to WL were informed about their inclusion in the study, that they were in the WL group, that they would initiate their treatment in 10 weeks, and were during the treatment period for CT and BT administered outcome measures at pretreatment, biweekly (primary outcome), and at posttreatment.

Treatments

The treatment was delivered over the Internet in a self-help format over 10 weeks, containing one module per week, with telephone support. The number of weeks was determined based on previous research [10, 19, 20]. The self-help format meant that all information needed for the participant to apply the cognitive and behavioral techniques in exercises on their own was presented in the PDF files supplied to each participant. Besides presenting each module as PDF files, the online platform also contained registration sheets for the exercises. In addition, each participant was offered 15 min a week of telephone support. This consisted of feedback on registered homework, help to problem solve issues with completing or understanding homework assignments, and ended with delivery of the next internet module (for participants to proceed to the next module in treatment we required, as a minimum, either a registration of one exercise or the participation in the weekly telephone call). The telephone support call was delivered by either a licensed clinical psychologist or a master student who were at the end of their clinical training. Prior to their participation in the study, therapists were required to read all 10 self-help internet modules, a therapist manual used to secure integrity and minimize risk of contamination, and finally participate in a therapist workshop. The therapist manual consisted of weekly protocols for the therapist and a

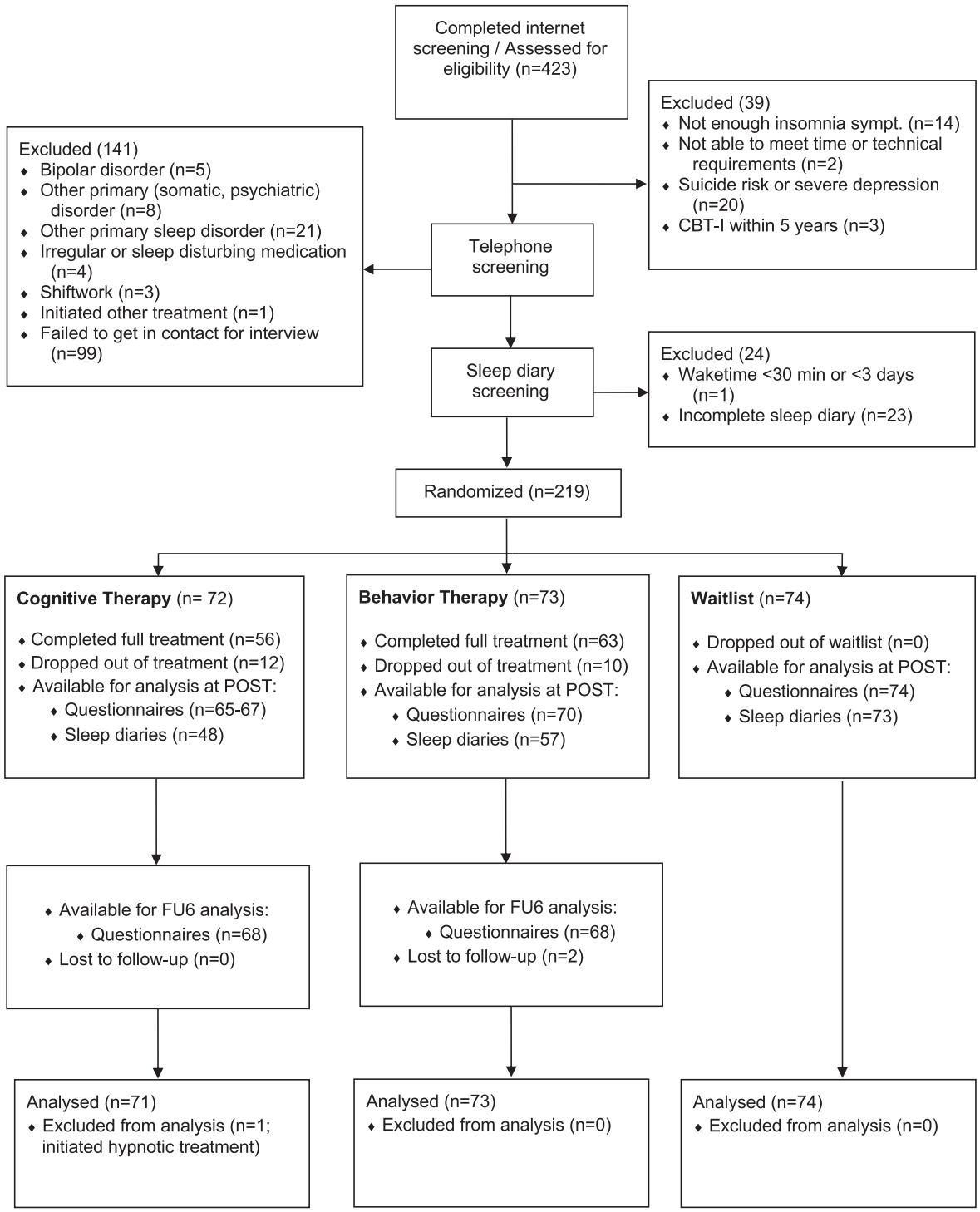

Figure 1. Flowchart.

general description of how to deliver the 15-min support call, as well as how to handle potential questions relating to treatment integrity. The workshop was used as a way of discussing and solving questions regarding delivery, integrity, and contamination. Integrity and contamination issues were also automatically handled by the fact that each therapy was delivered in text where participants in each group thus received exactly the same material. After treatment had been initiated all therapist were allowed supervision on a need to basis.

BT consisted of SR, SC, and sleep hygiene (SH) (for a treatment outline over the 10 weeks, see Table S1 in supplemental materials). SR evolved as a technique to target the proposal that insomnia is perpetuated by excessive time in bed (TIB). The aim of SR is therefore to limit TIB in alignment to actual sleep time and thereafter gradually increasing TIB until an optimal sleep time is achieved [13]. SC rests on the hypothesis that conditioning between temporal (around bedtime in the bedroom) and environmental stimuli (e.g. worry/frustration and arousal) have

occurred, which is incompatible with sleep and decreases likelihood for sleep. The aim of SC is to recondition the bed with sleep by increasing sleep compatible behaviors and limit sleep-disturbing behaviors around bedtime. This is achieved by a number of techniques or prescribed rules to follow, such as go to bed only when sleepy, getting out of bed whenever unable to fall asleep within 15 min, and having a fixed risetime for getting out of bed in the morning [12, 32, 33]. SH is intended to enhance, or at least remove obstacles to progress, by providing general guidelines about health and environmental practices that might interfere or promote sleep. The aim of SH is to optimize these health and environmental factors (e.g. diet, exercise, substance use, light, noise, temperature) for sleep [34, 35].

Cognitive therapy [15, 17, 19, 20, 36] as used in this trial (see Table 1 for a treatment outline over the 10 weeks) rests on increasing evidence that the following cognitive maintaining processes are involved in insomnia: (1) sleep-interfering or sleep-related worry, (2) unhelpful beliefs about sleep, (3) attentional bias and monitoring for sleep-related threat, (4) misperception of sleep, and (5) safety behaviors. The aim of CT is to reverse these maintaining mechanisms' influence on both day and nighttime symptoms in insomnia, through cognitive restructuring, achieved mainly by behavioral experiments [19, 34].

Assessment measures

We administered included measures at the following assessment points: diagnostic measures at pretreatment, treatment credibility during the first week of therapy, primary outcome biweekly from pre- to posttreatment, and also at 6-month follow-up (follow-up), nighttime symptoms pre- and posttreatment as well as biweekly for the treatment groups, secondary outcomes pre- and posttreatment and at follow-up, and treatment satisfaction, sick leave, healthcare consumption, concomitant insomnia treatment at posttreatment. Since the WL received treatment after 10 weeks, only CT and BT received the follow-up measures. In addition, the two therapy groups completed measures on their experience of treatment, therapists, workload, and activity at posttreatment.

All self-report measures were delivered via the internet platform. Specifically, each questionnaire was delivered by email that contained a unique link to the questionnaire (the link was also available on the study platform). In total, three automatic email reminders were sent out if an assessment had not been completed.

Diagnostic measures

In order to identify the psychiatric comorbidities, we used the following modules of The Mini International Neuropsychiatric Interview (M.I.N.I. 6.0.0 (2009-02-20). Swedish version): Major depressive disorder, Suicidality, Mania, Panic disorder, Social phobia, Posttraumatic stress disorder, Psychotic disorder, and Generalized anxiety disorder. The MINI is a semi-structured clinical interview with good reliability and validity [31]. To assess diagnostic criteria for sleep disorders, DSISD was used [30]. The DSISD is a semi-structured interview with good reliability and validity [30]. The structured interviews were conducted by the first and last authors as well as by two trained master students at the end of their clinical training who received supervision on a need to basis.

Table 1. Participant characteristics at baseline

	Cognitive therapy (n = 72)				Behavior therapy (n = 73)				Waitlist (n = 74)			
	%	n	M	SD	%	n	M	SD	%	n	M	SD
Gender (female)	76.4	55			69.9	51			73	54		
Age			51.5	12.5			51.8	14.5			54.2	14.6
Marital status												
Single	30.6	22			31.5	23			24.3	18		
Married/partner /separated	69.4	50			68.5	50			75.7	56		
Education												
High School	19.4	14			21.9	16			23	17		
University	80.6	58			78.1	57			77	57		
Employment												
Employed/stud.	83.3	60			75.3	55			66.2	49		
Unemployed	5.6	4			4.1	3			6.8	5		
Retired	11.1	8			20.5	15			27	20		
Insomnia duration			12.0	10.7			11.1	10.3			11.9	13.7
Insomnia severity			19.9	3.4			19.0	3.2			19.3	3.2
Medication												
Hypnotic	40.3	29			46.6	34			40.5	30		
Other*	45.8	33			46.6	34			44.6	33		
Comorbidity												
Somatic†	33.3	24			16.4	12			23.0	17		
Psychiatric ‡	16.7	12			13.7	10			18.9	14		

*Types of medication for the sample in total: antidepressants 7.3%, anti-inflammatory medications 2.3%, central stimulants 0.9%, for allergy 4.1%, for asthma 3.2%, for cancer 0.5%, for diabetes 1.4%, for epilepsy 0.9%, for gastric issues 1.4%, for headache 2.7%, for heart diseases 15.5%, for parkinson 0.5%, for thyroid gland 8.2%, tranquilizers 8.2%.

†Types of somatic diseases for the sample in total: autoimmune diseases 5.0%, cancer 1.8%, chronic pain 6.8%, endocrinological diseases 1.4%, gastric diseases 3.2%, headache 2.7%, heart diseases 4.1%, respiratory diseases 1.8%, neurological diseases 0.9%.

‡Depression 5.9%, GAD 8.2%, panic disorder 0.5%, PTSD 0.9%, social phobia 5.9%.

Primary outcome

The ISI [27, 28] was used to assess participants' global perception of insomnia severity. ISI was also used to categorize the number of responders and remitters at posttreatment. The seven-item questionnaire is rated on a 5-point scale (0–4) with a total score of 0–28 and assesses both night and daytime symptoms (difficulties with initiating and maintaining sleep, satisfaction, and concern with sleep and daytime impairment due to sleep). Response to treatment was defined as achieving a change of 8 points or more and remission as a final score below 8 [28]. The scale has demonstrated adequate internal consistency (Cronbach's α = .91) and temporal stability (r = .80) as well as being sensitive to therapeutic changes [37–39]. ISI has also been validated for use over the Internet [40].

Nighttime symptoms

To assess nighttime symptoms, a 7-day sleep diary [41] was used [17]. The diary assessed bedtime, lights out time, sleep onset latency (SOL), wake time after sleep onset (WASO), early morning awakening (EMA), and risetime. From these measures, the online diary automatically calculated total sleep time (TST). For those receiving BT, the diary also calculated TIB and sleep efficiency (SE). Outcome measures at pre- and posttreatment were calculated as weekly means of SOL, WASO, EMA, and TST. The sleep diary is considered the gold standard subjective measure of sleep with reliable estimates of sleep [25].

Secondary symptoms

To assess functional impairment, the Work and Social Adjustment Scale was used (WSAS) [42]. The WSAS assesses functioning across work, home management, social and private leisure activities, and relationships with others. The five-item questionnaire is rated on a 9-point scale (0–8) with a total score of 0–40. A previous study showed robust psychometric properties for the WSAS in insomnia patients [43].

To measure anxiety and depression, The Hospital Anxiety and Depression Scale was used (HADS) [44]. The HADS is a brief questionnaire containing 14 items designed to detect emotional disturbances in nonpsychiatric populations. It contains two subscales, of seven items each, assessing anxiety and depression. The scale has demonstrated acceptable psychometric properties [45, 46].

To evaluate quality of life, the Brunnsviken Brief Quality of Life was used (BBQ) [47]. The BBQ consists of 12 items assessing quality of life in six areas of life. The scale has good psychometric properties and is sensitive for distinguishing clinical and nonclinical samples as well as response to treatment [47, 48].

Treatment credibility, expectancy, and satisfaction

To assess treatment expectancy and credibility of both interventions, the Credibility/Expectancy Questionnaire (CEQ) [49] was administered. CEQ is a six-item questionnaire with demonstrated acceptable psychometric characteristics and has indicated ability to predict outcome [49, 50].

To assess treatment satisfaction, the Client Satisfaction Questionnaire was used (CSQ-8) [51]. CSQ-8 has demonstrated high internal consistency [51].

Treatment dropout and adherence

To assess treatment attrition and activity, data from the digital platform were summarized concerning treatment dropout, number of modules, number of logins on the study platform during the active treatment period, degree of total exercises completed, number of calls, and total minutes of telephone support.

Self-rated experience of treatment, therapists, workload, and activity

At posttreatment, participants were asked to rate how they perceived the therapy, the help from their therapist, and their activity during treatment. The therapies were rated in terms of how relevant and interesting the texts were, the amount of text, how demanding treatment was to undergo, and how strenuous it was to complete exercises. Degree of help received and how often issues were brought up with the therapists were also reported by participants. Their activity in terms of hours devoted per week for reading and performing exercises, degree of text read in total during the course of treatment (%), and amount of work invested in exercises was also collected.

Sick leave, healthcare consumption, concomitant insomnia treatment

To control for potential group differences on sick leave, healthcare consumption, and concomitant insomnia treatment, we assessed the occurrence of these at posttreatment and at follow-up with one item per domain. For sick leave, we asked how many days the participants had been on sick leave using three response categories (0 days, 1–14 days, and 15–180 days). To assess healthcare consumption, we asked whether participants had sought health care for the sleeping problems during the past 10 weeks or 6 months using three response categories (no, yes: in regular care, or yes: outside regular care). For concomitant insomnia treatment, we asked whether participants had undergone other insomnia treatments for their sleeping problems during the past 10 weeks or 6 months using three response categories [yes: with sleeping pills, yes: with other pharmacological agents, or yes: with nonpharmacological treatments (i.e. psychological and alternative treatments)].

Adverse events

Adverse events were assessed based on a method used in prior research where participants were asked, after treatment, to rate if any of 14 adverse events had occurred as a result of treatment [52].

Data analytic plan and statistical analyses

To contrast outcomes on variables related specifically to the treatments (CEQ, CSQ, treatment dropout, module completion, adverse effects, therapist support, and activity) and analyze differences between treatment completers and treatment dropouts, we used one-way ANOVA and t-tests for continuous distributed variables and chi-squared test of independence for categorical variables.

The primary data analytic models for continuous and categorical outcomes were fitted using full information maximum

likelihood estimation with nonnormality robust standard errors using Mplus vs. 7.4 [53]. Following the intention-to-treat principle, all models made use of all available data from all individuals who were randomized. Full information maximum likelihood estimation, which models all available observations jointly to estimate model parameters without imputation [54], is one of two recommended methods for handling missing data given that it provides unbiased estimates and standard errors under a more lenient missing data assumption (i.e. missing at random) [54, 55]. Throughout, comparisons were two tailed and treated as statistically significant at the level of $p < 0.05$ using normal theory tests (i.e. estimate[est]/standard error[S.E.]). Confidence intervals (CIs) are given with 95% margin. For continuous outcomes, effect sizes in the form of standardized mean differences (d) were derived from the model-implied means at endpoint assessment and the standard deviation at baseline [56]. For categorical outcomes, odds ratio effect sizes were calculated based on the unstandardized beta coefficient from the regression models.

Two sets of primary outcome analyses were conducted. The first set aimed to determine the immediate effects of therapies compared with the WL on outcomes measured during the active treatment phase (from baseline to week 10), whereas the second set aimed to compare the two active therapies using all available measurement points, from pre- to posttreatment for sleep-diary outcomes (six assessments points) and over the extended follow-up period (from baseline to 6-month follow-up) for the other outcomes. Given that individuals in the WL received treatment following postassessment, only the two active therapies were compared over the follow-up in the second set of analyses.

Latent growth modeling with random effects (person-specific trajectories) was used as the primary analytic approach to model individual change as a function of conditions and handle dependence in the data due to repeated-measures over time [57]. We followed recommendations for model building for growth models [57, 58], and population change was determined by visual inspection of observed means and individual trajectories and analytically by assessing model fit using fit indices for growth models [59]. Correlated subject-specific random coefficients (i.e. random intercept and slopes) were retained whenever they significantly contributed to the model. For the primary outcome measure ISI, which was measured biweekly during the active treatment period, a growth model with a random intercept, linear, and quadratic slope was fitted. Two binary variables representing the three conditions (with the WL as the reference category) were included as fixed predictors of linear and quadratic trajectories in the model to examine average differential change as a function of conditions (i.e. CT versus WL and BT versus WL). For the secondary measures, a random intercept and fixed linear slope were fitted and the same binary treatment variables were included as fixed predictors of the linear trajectories. Error terms in the models were constrained to be equal over time.

For the second set of models that used all measurement points and compared the two active therapies, piecewise growth models [57, 60] were fitted to adequately model nonlinear change and capture change and differential change as a function of treatment during the distinct phases of the trial (i.e. pre-post and follow-up phase). For the primary outcome ISI, change during the first piece (pre- and postassessments) was modeled with a random linear and quadratic slope, whereas change during the second piece (post and follow-up assessments) was modeled as a fixed linear slope. For the secondary outcome measures, two fixed linear slopes were used to model change during the pre–postassessment (piece 1) and follow-up period (piece 2). Averaged population change across conditions (i.e. main effect of time), and the averaged differential rates of change per therapy were assessed in each phase by including the treatment variable (CT = -0.5, BT = 0.5) as a fixed predictor of trajectories in the first and second piece.

The rates of response and remission extracted from the primary outcome ISI (defined under measures) were analyzed from pretreatment to posttreatment and from pretreatment to 6-month follow-up with logistic regression holding constant the pretreatment scores on ISI.

Results

Sample and patient characteristics

The sample mean age was 52.5 (N = 219) years, and 73.1% (n = 160) were females. Across conditions, 42.5% (n = 93) reported use of sleeping pills, 16.4% (n = 36) stated a psychiatric and 24.2% (n = 53) a somatic comorbid disorder, and 45.7% (n = 100) used medications for somatic conditions.

Treatment dropout and adherence

Table 2 presents descriptive statistics on treatment dropout (overall treatment dropout was 15.3%) and adherence to treatment (number of modules, number of logins, exercises completed, therapist support, and module at dropout) as a function of conditions along with results from inferential tests of differences between conditions. One significant difference between conditions was observed on one of the variables measuring adherence to treatment, indicating longer support calls for the CT group as compared with BT.

No significant differences were found between those who completed and those who dropped out of treatment concerning all variables in Table 1 (range p = 0.102–0.857). Zero participants dropped out of the WL.

Treatment credibility/expectancy and satisfaction

Both therapies were rated with high credibility, expectancy, and client satisfaction, with no statistically significant differences between the therapies. (For details see Table 2.)

Self-rated experience of treatments, therapists, workload, and activity

Concerning the participants' experiences of treatment, therapists, workload, and activity, the following significant differences were found: text perceived as interesting in favor of CT, hours/week spent on treatment with CT spending more, and on the degree of work invested with participants in BT investing more work. (For details see Table 2.)

Analysis of immediate treatment effects

Primary outcome

Figure 2 depicts the observed and estimated means for the biweekly measurements on ISI during the active treatment phase for both treatment arms and the WL. As observed in Table 3, the term in the growth model that tested differential change

Table 2. Treatment credibility, expectancy, satisfaction, and self-rated: activity and user-experience

	Cognitive therapy (*n* = 71)			Behavior therapy (*n* = 73)			
	M (SD)	*n*	%	M (SD)	*n*	%	χ2/F/t
CEQ							
Credibility	19.2 (3.9)			19.6 (3.3)			−0.743, $p = 0.459$
Expectancy	17.3 (5.4)			18.8 (4)			1.868, $p = 0.064$
CSQ	25.7 (4.6)			25.2 (5.7)			0.546, $p = 0.586$
Activity measures							
Treatment dropout		12	16.9		10	13.7	0.285, $p = 0.593$
Module at dropout*	3.67 (1.9)			3 (1.8)			0.840, $p = 0.411$
Number of modules†	8.89 (2.5)			9.05 (2.5)			−0.403, $p = 0.688$
Number of logins (*n*)	32.8 (16.9)			30.5 (18.5)			0.776, $p = 0.439$
Exercises completed			77.1			81.6	−0.917, $p = 0.361$
Number of support calls	8.41 (2.6)			8.86 (2.6)			1.042, $p = 0.299$
Total minutes of support calls	111.1 (42.1)			97.0 (38.8)			4.357, $p = 0.039$
Perception of treatment							
Amount of text‡	3.46 (0.7)			3.67 (0.7)			−1.762, $p = 0.080$
Text perceived as interesting/relevant§	3.95 (0.7)			3.69 (0.8)			2.116, $p = 0.036$
How strenuous exercises were to execute‖	2.89 (0.9)			2.59 (1.2)			1.597, $p = 0.113$
Difficulties in following through with treatment¶	2.97 (1.0)			3.13 (1.2)			−0.849, $p = 0.397$
How often issues with treatment were brought up with the therapist#	3.95 (1)			3.96 (1.1)			−0.022, $p = 0.982$
Degree of help received from therapist with issues brought up**	4.14 (0.9)			4.12 (0.9)			0.155, $p = 0.877$
Self-rated activity							
Hours/week spent on the treatment††							
0–2 h		28	35		52	65	13.597, $p < 0.001$
>2 h		37	67.3		18	32.7	13.597, $p < 0.001$
Amount of text read during 10 weeks‡‡	4.49 (0.8)			4.46 (0.7)			0.274, $p = 0.784$
Degree of work invested in exercises§§	3.68 (0.7)			4.16 (0.8)			−3.701, $p < 0.001$

CEQ, Credibility/Expectancy Questionnaire; CSQ, Client Satisfaction Questionnaire.
*Represents the mean module at which treatment dropout occured.
†Represents the mean number of modules reached, as a measure of adherence.
The following response alternatives were used for:
‡1–5 (way too little, too little, ok, too much, way too much).
§1–5 (never, rarely, sometimes, often, always).
‖1–5 (some, a little, partly, much, very much).
¶1–5 (hard, pretty hard, neither hard or easy, pretty easy, easy).
#0–5 (never, rarely, sometimes, often, always).
**0–5 (no help, a little help, some help, to a huge degree, much help).
††1–5 (0–1 h, 1–2 h, 3–4 h, 4–5 h, >5 h).
‡‡1–5 (0%, 25%, 50%, 75%, 100%).
§§1–5 (not at all, rarely, now and then, often, very often).

between CT and WL was statistically significant. Similarly, the term in the growth model that tested differential change between BT and WL was also statistically significant. As shown in Figure 3, the two therapies improved rapidly over the active treatment phase, whereas the WL only had a small decrease over this time period. Associated effect sizes at an endpoint of the active treatment phase (week 10), derived from the model-implied means, were $d = 2.15$ for CT compared with WL, and BT compared with WL $d = 2.01$.

Figure 3 presents the observed proportion of responders and remitters according to the ISI thresholds. Logistic regression revealed a statistically significant difference in terms of response between CT and WL (est = 3.34, S.E. = 0.49, $p < 0.001$) and BT and WL (est = 2.73, S.E. = 0.47, $p < 0.001$) with associated odds ratio effect sizes of 28.34 and 15.27, respectively. For remission, logistic regression revealed a statistically significant difference between CT and WL (est = 3.12, S.E. = 0.74, $p < 0.001$) and BT and WL (est = 3.19, S.E. = 0.74, $p < 0.001$) with associated odds ratio effect sizes of 22.57 and 24.26, respectively.

Sleep-diary and secondary outcomes

Table 4 provides the results from the estimated growth models, along with associated model-implied unstandardized and standardized mean differences (*d*) at week 10 (endpoint of active treatment phase). For the sleep-diary outcomes, the terms in the model that tested the difference between active treatments and the WL in linear change between pre- and postassessment were statistically significant in all models ($p < .001$), with an exception for WASO in which only BT differed significantly from WL (see Table 4). All effects favored the therapies over the WL and estimates of between-group effect sizes were in the range of moderate to large for the difference between treatments and WL at week 10 (range $d = 0.37$–0.83). For secondary outcomes, the terms in the model that tested the difference between active treatments and WL in linear change between pre- and postassessment were statistically significant in all models ($p < 0.001$), with the exception of BBQ in which only CT differed significantly from WL (see Table 4). All effects favored the therapies over the WL and estimates of between-group effect sizes

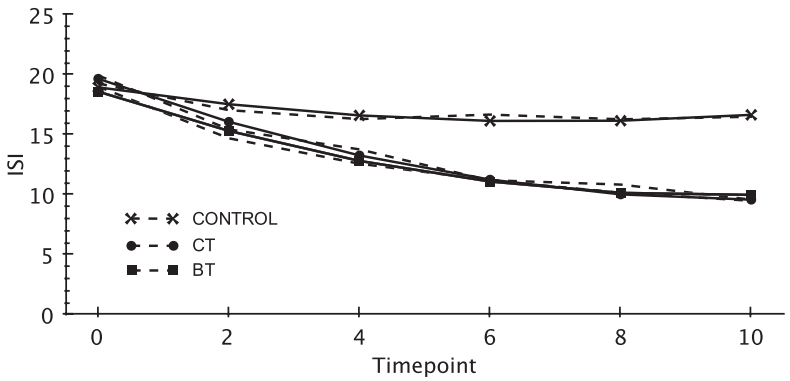

Figure 2. Observed and estimated means for the biweekly measurements on ISI.

Table 3. Observed means and results from the quadratic growth model examining the controlled effects of therapies on ISI during the active treatment phase

	Observed means				Results from growth models				
	PRE		POST		Effect on linear slope		Effect on quadratic slope		
ISI	N	M (SD)	N	M (SD)	Estimate (S.E.)	P	Estimate (S.E.)	P	Effect size
CT	71	19.9 (3.4)	67	9.3 (4.1)	−2.299 (0.45)	<0.001	0.144 (0.079)	0.066	2.146
BT	73	19.0 (3.2)	70	9.6 (5.6)	−2.050 (0.44)	<0.001	0.157 (0.076)	0.039	2.014
WL	74	19.3 (3.2)	74	16.5 (4.4)	—	—		—	—

The growth model is based on available data for the intention-to-treat sample (N = 218). Treatment conditions were included as fixed binary coded predictors using the control condition as the reference category. The estimate is the unstandardized regression coefficient and can be interpreted as an effect size in the original metric of the scale (one-time unit is 2 weeks). The effect size was derived from the model estimates and represented the standardized mean difference between treatment and control at posttreatment assessment.
BT, behavior therapy; CT, cognitive therapy; ISI, Insomnia Severity Index; S.E., standard error; WL, waitlist.

were in the range of moderate to large for the difference between treatments and WL at week 10 (range d = 0.38–0.92).

Comparative analysis of active treatments on sleep-diary outcomes

Table 5 presents the results from the estimated growth models, along with associated model-implied unstandardized and standardized mean differences (d) at week 10 (endpoint of active treatment phase). The terms that tested differential linear change over the treatment phase as a function of therapy were nonsignificant in all models (p > 0.05), except for SOL which indicated a significant difference in favor of BT. The estimated 10 week between-group difference in effect sizes were all small (range d = 0.01–0.28).

Analysis of active treatments over the full assessment period

Primary outcome.

The terms in the first piece (i.e. pre- to postperiod) that tested averaged population change across therapies were statistically significant, whereas the term that tested averaged population change in the second piece (i.e. follow-up period) was nonsignificant (see Table 6, for details). This suggests that the improvements made in both therapies during the active treatment phase were sustained through the follow-up. None of the terms in the model that tested differential change as a function of therapy were statistically significant (all p's > .45). Associated between-group effect size at 6-month follow-up, derived from the model-implied means, was d = 0.04.

Figure 3 presents the response and remission rates at follow-up. According to the logistic regression, there were no significant difference between the therapies in terms of response (est = -0.19, S.E. = 0.37, p = .614) with an associated odds ratio effect size of 0.83; nor in terms of remission (est = 0.13, S.E. = 0.37, p = .719) with an associated odds ratio effect size of 1.17.

Secondary outcomes.

The results of the piecewise growth models, along with model-implied mean differences at follow-up, are presented in Table 6. The term in the piecewise growth model that tested average change across therapies over the active treatment phase was statistically significant in all models (p < 0.001), whereas the term in the model that tested linear change during the follow-up was nonsignificant (p > 0.05). The terms that tested differential linear change over the treatment phase and follow-up as a function of therapies were nonsignificant in all models (p > 0.05). The estimated endpoint follow-up between-group mean difference effect sizes were all small (range d = 0.02–0.23).

Sick leave, healthcare consumption, concomitant insomnia treatment

One significant difference was found between the groups at posttreatment [$\chi^2(2) = 7.55$, p = 0.023], indicating that participants in the WL group underwent other treatments to a higher degree

at posttreatment (for a more detailed description of these results, see Supplementary Table S2).

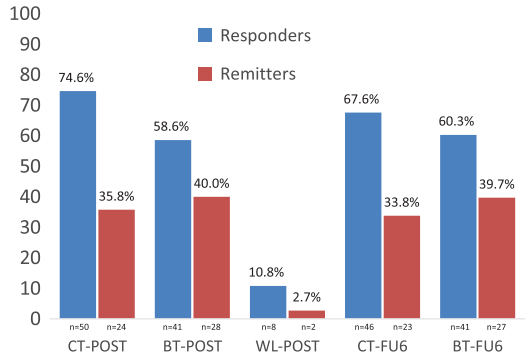

Figure 3. Percent response and remission based on ISI (observed means).

Adverse events

Of the total sample, 29% reported an adverse event due to therapy at posttreatment. A significant difference was found between the treatments (CT = 14.1%, BT 43.2%), [$\chi^2(1) = 14.97$, $p < 0.001$]. (For more details, see Supplementary Table S3.)

Discussion

The aim of this study was to compare internet-delivered CT and BT against a WL control on a broad range of outcomes. To the best of our knowledge this is the first study to test CT delivered over the internet. Also, this is most likely the first RCT comparing CT and BT against a WL. The overall finding was that both CT and BT outperformed the WL. Moreover, CT and BT produced comparable effects on the majority of outcomes. The results thus indicate that both CT and BT are effective as stand-alone therapies for insomnia disorder [10, 20].

Table 4. Observed means and results from the linear growth model examining the controlled effects of therapies on secondary outcomes and sleep diaries

	Observed means				Results from linear growth models			
	Pre		Post		Effect on linear slope		Group diff at week 10	
	N	*M* (*SD*)	*N*	*M* (*SD*)	Estimate (S.E.)	*P*	Mean diff [95% CI]	Effect size
WSAS								
CT	71	23.3 (8.0)	66	9.7 (8.4)	−9.867 (1.321)	<0.001	−7.529 [−10.469, −4.589]	0.879
BT	73	20.9 (9.7)	70	9.4 (7.8)	−7.721 (1.448)	<0.001	−7.844 [−10.644, −5.044]	0.915
WL	74	21 (8.1)	74	17.2 (9.5)	—	—	—	—
HADS-A								
CT	71	9.3 (3.9)	65	6.8 (3.4)	−1.643 (0.563)	0.004	−1.807 [−3.042, −0.571]	0.480
BT	73	8.8 (3.9)	70	5.8 (3.3)	−2.113 (0.528)	<0.001	−2.819 [−4.011, −1.627]	0.749
WL	74	9.5 (4.0)	74	8.6 (4.0)	—	—	—	—
HADS-D								
CT	71	6.5 (3.7)	65	3.7 (2.9)	−2.152 (0.479)	<0.001	−2.025 [−3.101, −0.950]	0.616
BT	73	6.3 (3.4)	70	3.7 (2.7)	−2.038 (0.461)	<0.001	−2.129 [−3.143, −1.115]	0.648
WL	74	6.3 (3.3)	74	5.8 (3.6)	—	—	—	—
BBQ								
CT	71	50.3 (19.1)	65	59.8 (18.2)	10.696 (2.578)	<0.001	7.735 [1.234, 14.237]	0.388
BT	73	56.2 (19.0)	70	59.6 (21.7)	4.885 (3.156)	0.122	7.793 [0.759, 14.827]	0.391
WL	74	53.3 (20.6)	74	52.1 (21.8)	—	—	—	—
SOL								
CT	71	47 (40)	48	25 (17)	−14.887 (5.180)	0.004	−14.305 [−23.282, −5.328]	0.370
BT	73	55 (38)	57	20 (13)	−31.831 (5.114)	<0.001	−23.712 [−32.112, −15.312]	0.613
WL	74	47 (37)	73	42 (32)	—	—	—	—
WASO								
CT	71	49 (44)	48	32 (26)	−11.075 (6.014)	0.066	−23.380 [−34.867, −11.894]	0.552
BT	73	56 (36)	57	18 (17)	−29.711 (5.764)	<0.001	−35.174 [−45.266, −25.081]	0.831
WL	74	62 (46)	73	52 (39)	—	—	—	—
EMA								
CT	71	41 (36)	48	22 (25)	−18.551 (5.994)	0.002	−23.380 [−27.542, −7.250]	0.523
BT	73	38 (30)	57	14 (15)	−23.658 (5.046)	<0.001	−35.174 [−33.414, −16.502]	0.750
WL	74	40 (32)	73	39 (32)	—	—	—	—
TST								
CT	71	339 (65)	48	386 (48)	34.566 (8.986)	<0.001	41.468 [18.985, 63.951]	0.588
BT	73	325 (67)	57	377 (57)	41.723 (10.105)	<0.001	36.205 [12.551, 59.860]	0.514
WL	74	332 (80)	73	344 (81)	—	—	—	—

Note. The growth model is based on available data for the intention-to-treat sample ($N = 218$). Treatment conditions were included as fixed binary coded predictors using the control condition as the reference category. The estimate is the unstandardized regression coefficient and can be interpreted as an effect size in the original metric of the scale (one-time unit is 10 weeks). The unstandardized mean difference (unstandardized effect size) and effect size (standardized mean difference) were derived from the model estimates.

BT, behavior therapy; BBQ, Brunnsviken Brief Quality of Life Scale; CT, cognitive therapy; EMA, Early morning awakening; HADS-A & HADS-D, Hospital Anxiety and Depression scale; S.E., standard error; SOL, sleep onset latency; TST, total sleep time; WASO, Wake after sleep onset; WL, waitlist; WSAS, Work and Social adjustment Scale.

Table 5. Results from growth models contrasting CT and BT on six assessment points from pre- to posttreatment on sleep-diary outcomes

Results from linear growth models				
Effect of the predictor			Group diff at week 10	
Outcome/predictor	Estimate (S.E.)	*P*	Mean diff [95%CI]	Effect size
SOL				
Time (linear)	−10.269 (1.218)	<0.001		
Time (quadr)	1.107 (0.198)	<0.001		
Time (linear) on Tx	−5.975 (2.702)	0.027		
Time (quadr) on Tx	0.969 (0.406)	0.018	−6.154 [−11.758, −0.550]	0.150
WASO				
Time (linear)	−12.605 (1.823)	<0.001		
Time (quadr)	1.618 (0.307)	<0.001		
Time (linear) on Tx	−5.650 (3.318)	0.089		
Time (quadr) on Tx	0.665 (0.562)	0.237	−10.708 [−18.779, −2.637]	0.277
EMA				
Time (linear)	−9.899 (1.355)	<0.001		
Time (quadr)	1.179 (0.229)	<0.001		
Time (linear) on Tx	−3.914 (2.795)	0.161		
Time (quadr) on Tx	0.754 (0.474)	0.112	−6.351 [−13.110, 0.409]	0.188
TST				
Time (linear)	16.113 (2.813)	<0.001		
Time (quadr)	−1.412 (0.498)	0.005		
Time (linear) on Tx	−5.810 (5.614)	0.301		
Time (quadr) on Tx	1.855 (0.994)	0.062	0.394 [−17.374, 18.163]	0.006

The growth model is based on available data for the intention-to-treat sample (n = 144). Time (linear) and time (quadr) in the model are the time coefficients representing averaged population change across conditions for the active treatment phase. Tx is a group variable representing treatment assignment (BT = 0.5, CT = −0.5). The estimate is the unstandardized regression coefficient and can be interpreted as an effect size in the original metric of the scale (one-time unit is 2 weeks). The unstandardized mean difference (unstandardized effect size) and effect size (standardized mean difference) were derived from the model estimates. The negative mean difference at week 10 indicates a beneficial effect for BT relative to CT on SOL, WASO, and EMA, and the positive mean difference on TST also indicates a beneficial effect for BT relative to CT.

EMA, Early morning awakening; S.E., standard error; SOL, sleep onset latency; Time (linear) and Time (quadr), estimation of linear and quadratic time; TST, total sleep time; WASO, Wake after sleep onset.

On the primary outcome (ISI), both therapies outperformed the WL with no significant differences between the two active treatments. CT achieved larger effect sizes—larger refers to an in-group effect size, similar to, or ≥0.5 [61]—compared to former trials of CT, BT and a meta-analysis of CBT-I [8, 9, 20]. BT achieved similar or larger effect sizes in comparison to earlier BT and meta-analysis of CBT-I [8, 9, 20, 62]. CT in the present study compared to the one in Harvey et al. [20], yielded larger rates of response at posttreatment and comparable rates at follow-up, while remission rates yielded slightly larger rates at post while smaller at follow-up, reflecting a maintained response and remission in contrast to a gained response and remission in Harvey et al. [20]. For BT, the response rate was smaller at posttreatment and larger at follow-up as compared with those presented in Harvey et al. [20]., while the remission rates were comparable at both post and follow-up, reflecting a maintained response in contrast to a decrease compared to BT. Compared to full CBT-I, the response rates were comparable to larger, while for remission they were comparable or lower [20, 63]. Different trajectories of change from posttreatment to follow-up were also evident on the ISI score, with a similar symptom development for both CT and BT, compared to Harvey et al. [20]. in which BT deteriorated and CT improved during the follow-up period.

On the eight secondary outcomes, both CT and BT outperformed the WL with comparable effects on the majority of outcomes. Although there was a trend for slightly higher effect sizes for BT on all eight outcomes, there was only one significant difference on SOL. Whether the difference on SOL, which consisted of an estimated 6 min group difference at posttreatment, is of clinical significance is an open question. However, one interpretation would be to consider this a small difference given the long latencies among patients with insomnia disorder (36–48.9 min) [64–66]. In comparison with previous trials of CT, BT, and CBT-I [20, 67, 68], this study yielded an overall, similar or larger effect sizes on the secondary outcomes. It is worth mentioning that CT in this trial yielded larger effects on SOL compared to former outcomes for CT and full CBT-I [8, 9, 20], and similar to larger effects on total sleep time compared to CT-I and CBT-I, respectively [8, 9, 20].

In terms of theory, where CT has a focus on both night- and daytime symptoms, and BT is primarily focused on nighttime symptoms [12, 13, 15], our findings confirm prior research showing that BT is equally effective for symptoms related to the day as CT [20]. Also, the cognitive approach to nighttime difficulties by reversing cognitive processes (in contrast to changing sleep-disturbing behaviors and contextual factors in BT) does not seem to make a clinical difference on overall insomnia severity and sleep-diary outcomes, since CT produced comparable effects overall as BT. Neither does the broader approach of CT (that targets several perpetuating factors), compared to BTs focus on improving sleep, generate a stronger effect on daytime measures, compared to BT. Our hypotheses that BTs primary focus on nighttime processes would result in greater improvements on sleep-diary outcomes and that CTs focus on both night and daytime symptoms would be more effective on outcomes related to daytime symptoms was, thus, not supported (with one notable exception).

Table 6. Observed means and results from piecewise growth models examining change over the pre-post assessment and 6-month follow-up for the primary and secondary outcomes

	Observed means		Results from linear growth models				
	FU6		Effect of the predictor			Group diff at FU6	
Outcome	*N*	*M* (*SD*)	Predictor	Estimate (S.E.)	*P*	Mean diff [95% CI]	Effect size
WSAS							
CT	68	9.4 (7.9)	Time 1	−12.510 (0.788)	<0.001		
BT	68	9.3 (8.9)	Time 2	−0.061 (0.310)	0.844		
			Time 1 on Tx	2.052 (1.574)	0.192		
			Time 2 on Tx	0.095 (0.621)	0.879	−0.182 [−2.976, 2.612]	0.022
HADS-A							
CT	68	7.2 (3.5)	Time 1	−2.706 (0.275)	<0.001		
BT	68	6.4 (4.1)	Time 2	0.198 (0.110)	0.072		
			Time 1 on Tx	−0.448 (0.551)	0.415		
			Time 2 on Tx	0.186 (0.220)	0.398	−0.545 [−1.805, 0.715]	0.148
HADS-D							
CT	68	4.3 (2.9)	Time 1	−2.611 (0.255)	<0.001		
BT	68	3.7 (2.9)	Time 2	0.156 (0.085)	0.068		
			Time 1 on Tx	0.146 (0.509)	0.774		
			Time 2 on Tx	−0.220 (0.171)	0.198	−0.600 [−1.576, 0.375]	0.192
BBQ							
CT	68	58.3 (18.6)	Time 1	6.527 (1.522)	<0.001		
BT	68	62.7 (20.7)	Time 2	0.256 (0.683)	0.708		
			Time 1 on Tx	−5.873 (3.047)	0.054		
			Time 2 on Tx	1.881 (1.364)	0.168	4.508 [−2.007, 11.023]	0.232
ISI							
CT	68	10.1 (4.9)	Time 1(linear)	−3.841 (0.239)	<0.001		
BT	68	10.1 (5.7)	Time 1 (quadr)	0.395 (0.041)	<0.001		
			Time 2	0.032 (0.033)	0.327		
			Time 1(linear) on Tx	0.201 (0.485)	0.678		
			Time 1(quadr) on Tx	0.020 (0.084)	0.816		
			Time 2 on Tx	−0.049 (0.066)	0.454	−0.133 [−1.886, 1.620]	−0.039

Note. The growth model is based on available data for the intention-to-treat sample (n = 144). Time 1 and Time 2 in the model are the time coefficients representing averaged population change across conditions for the active treatment phase and the follow-up phase, respectively. Tx is a group variable representing treatment assignment (BT = 0.5, CT = −0.5). The estimate is the unstandardized regression coefficient and can be interpreted as an effect size in the original metric of the scale (one-time unit is 10 weeks for all outcomes except for ISI where one-time unit is 2 weeks at Time 1 and 6 months for Time 2). The unstandardized mean difference (unstandardized effect size) and effect size (standardized mean difference) were derived from the model estimates.

BT, behavior therapy; BBQ, Brunnsviken Brief Quality of Life Scale; CT, cognitive therapy; HADS-A & HADS-D, Hospital Anxiety and Depression scale; S.E., standard error; Time 1 and Time 2, first and second piece of the linear growth model; Time 1 (quadr), estimation of quadratic time; WL, waitlist; WSAS, Work and Social adjustment Scale.

Some other theoretical questions related to the construct validity of the treatments are also of value to address, in particular the length of treatment, dose–response relationship, and amount of therapist support. A treatment length of 10 weeks for a monotherapy (CT or BT) with telephone support may seem unnecessarily long and resource consuming in comparison to multicomponent CBT-I, which in clinical trials average 6 weeks with a trend toward briefer therapies, and to fully automated Internet programs, thus questioning both the scalability of the program and the necessity of 10-week therapy for response. Although questions of scalability and necessary dose for response are valid concerns, there are also some additional issues that need to be taken into account. First, the variant of CT used in this trial is of a more comprehensive nature (6–22 with an average of 17[19] and 8[20] sessions) than cognitive interventions usually incorporated in CBT-I. Second, although shorter therapies are relevant for effective clinical management, the focus in our study was on comparing two theoretically distinct therapies on equal grounds in order to evaluate their individual efficacy. Third, the optimal dose of CBT-I is still a relatively unaddressed issue that most previous trials with briefer therapies alone cannot answer. Fourth, the evidence that participants in this trial improved continuously for all 10 weeks (see Figure 2) indicates that no floor effect was reached and contradicts that treatment was unnecessarily lengthy in this trial. In terms of therapist support, it is worth to highlight that our mode of delivery stands in contrast to internet-delivered treatments that are fully automated [69, 70] in that we included 15 min of telephone support. Thus, these 10 weeks of treatment with 15 min of telephone support per week seem to generate promising results for CT and BT as stand-alone therapies, but whether these results are generalizable to shorter versions (6 or 8 weeks) without telephone support is still unknown. Finally, it is of value to note that this trial seems to indicate that this version of CT[19] can also be delivered with minimal contact and supervision over the Internet, despite its comprehensive nature.

There were some notable differences on the participants' perception of each therapy, the degree of support, and the number of adverse events. CT received significantly more time of telephone support, rated the text as more interesting, and reported that they spent more time on treatment, whereas those in BT reported that they invested more work in exercises. These

findings might possibly be explained by the difference in treatment and exercise structure, where exercises in BT are more repetitive but also demanding in terms of effort and planning, whereas CT consists of more variation in texts and exercises, thus generating more time spent on treatment. Finally, those who underwent BT experienced triple the amount of adverse events as those in the CT condition. This might probably be explained by evidence showing that total sleep time (subjective and objective) decreases initially during SR in BT [71]. These findings, while interesting in terms of the contrasting study aim, could unfortunately also limit the interpretations to be drawn from the main outcomes, in that patients in CT could be affected by extra attention and patients in BT being less compliant with homework assignments due to adverse events. Since our posttreatment assessment lacked time-sensitive information on where events occurred during therapy, future research could aim to assess adverse events during the course of therapy and their association to compliance and long-term outcomes.

There are also some other limitations that need to be considered. First, the sample was self-referred, which limits generalization, since self-referred samples are likely different compared to patients in regular care [72]. The sample was demographically characterized by a high mean age and being well-educated, which may restrict generalization. Second, although the screening process were guided by manuals with supervision on demand, there were no assessment of this process, thereby imposing an uncertainty in the validity of the screening procedure. Third, this was an internet-delivered treatment with telephone support (similar to support offered for BT-I) [73], where the active ingredients (information, instructions, and exercises) were presented in the format of text, thus requiring an effort of participants. This could, in combination with our well-educated sample, limit generalization of our findings to other populations. However, noteworthy is that previous research has, so far, not provided any clear evidence of differential outcomes as a function of delivery mode (i.e. face-to-face vs. Internet) [9, 74], thus attenuating this limitation. Fourth, although this study contains several digitally administered measures that minimize risk for observer bias, other important outcomes, such as therapist compliance, actigraphy, polysomnography, and diagnostic interview, were not included. The use of the MINI can further be criticized for being a screening, rather than a diagnostic measure. Fifth, although therapies were delivered in text, and telephone support was guided by a manual with instructions for maintaining the treatment integrity, there was no registration of compliance with this manual, thus raising the possibility of therapist drift and treatment contamination. Finally, although the total rate of treatment dropout (15%) was in line with mean dropout rates in individual CBT for depression (17.5%) [75], and no significant difference emerged between the therapies, it may still limit the interpretation of the findings. Worth to mention, however, is that the primary method (full information maximum likelihood estimation) used to handle missing data is considered state of the art and produces accurate estimates under a lenient missing data assumption [55].

To advance knowledge in the field, we recommend future research to address abovementioned limitations, by examining other patient groups (e.g. patients in clinical settings), including additional measures (e.g. actigraphy and post-diagnostic assessment), and measuring therapeutic behavior. Since CT and BT may demand a different amount of time to achieve its optimal effect, it is of interest to investigate dose–response relationships. Exploring long-term effects and cost–benefit analysis are also of importance to provide more information of the therapies' comparative effectiveness. Of further interest are examinations of components in CBT-I in more detail, i.e. assessing effects of the separate components inherent in each therapy (CT and BT), as are examinations of moderators and mediators for CT and BT. Finally, the efficacy of each therapy and their components in regular care is also of interest.

As a whole, this trial showed that both CT and BT had similar effects and are both effective as stand-alone therapies for insomnia disorder. These findings raise further questions about which therapy and subcomponents that are necessary and sufficient for change, how they are optimally combined, and how change for each therapy is mediated and moderated. This underaddressed research area could aid in the development of new therapies that combine components to optimize outcomes for the individual patient. For the research community, the results highlight the need for further dismantling of the therapies and components in CBT-I. Clinically, these results suggest a higher flexibility for therapists in choosing and implementing one of the two therapy models for treatment.

Supplementary Material

Supplementary material is available at *SLEEP* online.

Acknowledgements

We would like to acknowledge the following individuals for their work as therapist: Sven Alfonsson, Susanna Bitter, Sara Brandell, Amanda Eneroth, Hanna Fischer, Hampus Forslin, Josefin Garberg, Sofi Granert, Jenny Killgren, Andreas Kivinen, Philip Lindner, Hedvig Nettelblad, Anders Rayner, Martin Samuelsson, Kerstin Sindemark, Johan Sjöberg, Sigge Skårman, Ella Thiblin, Elin Zetterlund, and Emma Way and Patrik Johansson for participating in both the telephone-screening and as therapists. We also wish to thank George Vlaescu for his invaluable help in designing the internet platform. Finally, we would like to acknowledge and give a special thanks to Philip Lindner for his invaluable support and aid in the management and preparation of data for statistical analyses.

Funding

This study has received funding from Vetenskapsrådet (421-2013-996).

Conflict of interest statement. C.M.M. reports personal fees from Cereve, Phillips, personal fees from Merck, and Abbott, outside the submitted work. M.J.-F. reports a grant from the Swedish Research Council. All other authors have no conflicts to disclose.

References

1. Morin CM, *et al*. Chronic insomnia. *Lancet*. 2012;**379**(9821):1129–1141.
2. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev*. 2002;**6**(2):97–111.

3. Daley M, *et al*. Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Med*. 2009;**10**(4):427–438.
4. Sivertsen B, *et al*. Does insomnia predict sick leave? The Hordaland Health Study. *J Psychosom Res*. 2009;**66**(1):67–74.
5. Baglioni C, *et al*. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. *J Affect Disord*. 2011;**135**(1-3):10–19.
6. Breslau N, *et al*. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol Psychiatry*. 1996;**39**(6):411–418.
7. Morin CM, *et al*. The natural history of insomnia: a population-based 3-year longitudinal study. *Arch Intern Med*. 2009;**169**(5):447–453.
8. van Straten A, *et al*. Cognitive and behavioral therapies in the treatment of insomnia: a meta-analysis. *Sleep Med Rev*. 2018;**38**:3–16.
9. Zachariae R, *et al*. Efficacy of internet-delivered cognitive-behavioral therapy for insomnia – a systematic review and meta-analysis of randomized controlled trials. *Sleep Med Rev*. 2016;**30**:1–10.
10. Morin CM, *et al*. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004). *Sleep*. 2006;**29**(11):1398–1414.
11. Borbély AA. A two process model of sleep regulation. *Hum Neurobiol*. 1982;**1**(3):195–204.
12. Bootzin RR, *et al*. Stimulus Control Instructions. In: Hauri PJ, ed. *Case Studies in Insomnia*. Boston, MA: Springer; 1991:19–28.
13. Spielman AJ, *et al*. Treatment of chronic insomnia by restriction of time in bed. *Sleep*. 1987;**10**(1):45–56.
14. Webb WB. An objective behavioral model of sleep. *Sleep*. 1988;**11**(5):488–496.
15. Harvey A. A cognitive model of insomnia. *Behav Res Ther*. 2002;**40**(8):869–893.
16. Lundh LG, *et al*. Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *J Psychosom Res*. 2000;**49**(5):299–310.
17. Morin CM. *Insomnia: Psychological Assessment and Management*. New York, NY: Guilford Publication; 1993. http://psycnet.apa.org/record/1993-98362-000. Accessed October 29, 2019.
18. Wicklow A, *et al*. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav Res Ther*. 2000;**38**(7):679–693.
19. Harvey AG, *et al*. An open trial of cognitive therapy for chronic insomnia. *Behav Res Ther*. 2007;**45**(10):2491–2501.
20. Harvey AG, *et al*. Comparative efficacy of behavior therapy, cognitive therapy, and cognitive behavior therapy for chronic insomnia: a randomized controlled trial. *J Consult Clin Psychol*. 2014;**82**(4):670–683.
21. Faul F, *et al*. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*. 2007;**39**(2):175–191.
22. Vlaescu G, *et al*. Features and functionality of the Iterapi platform for internet-based psychological treatment. *Internet Interv*. 2016;**6**:107–114.
23. Edinger JD, *et al*.; American Academy of Sleep Medicine Work Group. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep*. 2004;**27**(8):1567–1596.
24. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders : DSM-5*. Washington, DC: American Psychiatric Association. 2013.
25. Buysse DJ, *et al*. Recommendations for a standard research assessment of insomnia. *Sleep*. 2006;**29**(9):1155–1173.
26. Lichstein KL, *et al*. Quantitative criteria for insomnia. *Behav Res Ther*. 2003;**41**(4):427–445.
27. Bastien CH, *et al*. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med*. 2001;**2**(4):297–307.
28. Morin CM, *et al*. The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*. 2011;**34**(5):601–608.
29. Svanborg P, *et al*. A new self-rating scale for depression and anxiety states based on the Comprehensive Psychopathological Rating Scale. *Acta Psychiatr Scand*. 1994;**89**(1):21–28.
30. Edinger J, *et al*. Testing the reliability and validity of DSM-IV-TR and ICSD-2 insomnia diagnoses: results of a multitrait-multimethod analysis. *JAMA Psychiatry*. 2011;**68**(10):992–1002. doi:10.1001/archgenpsychiatry.2011.64
31. Sheehan D, *et al*. The validity of the Mini International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. *Eur Psychiatry*. 1997;**12**(5):232–241.
32. Bootzin RR. Stimulus Control Treatment for Insomnia. Proceedings of the American Psychological Association. 1972;**7**:395–396.
33. Bootzin RR. Effects of self-control procedures for insomnia. *Am J Clin Biofeedback*. 1979;**2**(2):70–77.
34. Perlis ML, *et al*. *Behavioral Treatments for Sleep Disorders: A Comprehensive Primer of Behavioral Sleep Medicine Interventions*. San Diego, CA: Academic Press; 2011.
35. Hauri PJ. *Case Studies in Insomnia*. Boston, MA: Springer; 1991.
36. Harvey AG. A cognitive theory and therapy for chronic insomnia. *J Cogn Psychother*. 2005;**19**(1):41–59.
37. Morin CM, *et al*. Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. *Am J Psychiatry*. 2004;**161**(2):332–342.
38. Morin CM, *et al*. Self-help treatment for insomnia: a randomized controlled trial. *Sleep*. 2005;**28**(10):1319–1327.
39. Morin CM, *et al*. Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. *JAMA*. 2009;**301**(19):2005–2015.
40. Thorndike FP, *et al*. Validation of the insomnia severity index as a web-based measure. *Behav Sleep Med*. 2011;**9**(4):216–223.
41. Carney CE, *et al*. The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep*. 2012;**35**(2):287–302.
42. Mundt JC, *et al*. The Work and Social Adjustment Scale: a simple measure of impairment in functioning. *Br J Psychiatry*. 2002;**180**:461–464.
43. Jansson-Fröjmark M. The work and social adjustment scale as a measure of dysfunction in chronic insomnia: reliability and validity. *Behav Cogn Psychother*. 2014;**42**(2):186–198.
44. Zigmond AS, *et al*. The hospital anxiety and depression scale. *Acta Psychiatr Scand*. 1983;**67**(6):361–370.
45. Olssøn I, *et al*. The Hospital Anxiety and Depression Rating Scale: a cross-sectional study of psychometrics and case finding abilities in general practice. *BMC Psychiatry*. 2005;**5**:46.
46. Norton S, *et al*. The Hospital Anxiety and Depression Scale: a meta confirmatory factor analysis. *J Psychosom Res*. 2013;**74**(1):74–81.
47. Lindner P, *et al*. The Brunnsviken Brief Quality of Life Scale (BBQ): development and psychometric evaluation. *Cogn Behav Ther*. 2016;**45**(3):182–195.

48. Andersson E, *et al*. Internet-based extinction therapy for worry: a randomized controlled trial. *Behav Ther*. 2017;**48**(3):391–402.
49. Devilly GJ, *et al*. Psychometric properties of the credibility/expectancy questionnaire. *J Behav Ther Exp Psychiatry*. 2000;**31**(2):73–86.
50. Borkovec TD, *et al*. Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *J Consult Clin Psychol*. 1993;**61**(4):611–619.
51. Attkisson CC, *et al*. The client satisfaction questionnaire. Psychometric properties and correlations with service utilization and psychotherapy outcome. *Eval Program Plann*. 1982;**5**(3):233–237.
52. Kyle SD, *et al*. No pain, no gain: an exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Med*. 2011;**12**(8):735–747.
53. Muthén LK, *et al*. *Mplus User's Guide*. 7th ed. Los Angeles, CA: Muthen & Muthen; 2015.
54. Enders CK. *Applied Missing Data Analysis*. New York: Guilford Press; 2010.
55. Schafer JL, *et al*. Missing data: our view of the state of the art. *Psychol Methods*. 2002;**7**(2):147–177.
56. Feingold A. Effect sizes for growth-modeling analysis for controlled clinical trials in the same metric as for classical analysis. *Psychol Methods*. 2009;**14**(1):43–53.
57. Bollen KA, *et al*. *Latent Curve Models: A Structural Equation Perspective*. Hoboken, NJ: Wiley; 2006.
58. Hesser H. Modeling individual differences in randomized experiments using growth models: Recommendations for design, statistical analysis and reporting of results of internet interventions. *Internet Interv*. 2015;**2**(2):110–120.
59. Wu W, *et al*. Evaluating model fit for growth curve models: Integration of fit indices from SEM and MLM frameworks. *Psychol Methods*. 2009;**14**(3):183–201.
60. Raudenbush SW, *et al*. *Hierarchical Linear Models: Applications and Data Analysis Methods*. Newbury Park, CA: Sage; 2002.
61. Kazdin AE, *et al*. Power to detect differences between alternative treatments in comparative psychotherapy outcome research. *J Consult Clin Psychol*. 1989;**57**(1):138–147.
62. Waters WF, *et al*. Behavioral and hypnotic treatments for insomnia subtypes. *Behav Sleep Med*. 2003;**1**(2):81–101.
63. Ritterband LM, *et al*. Effect of a web-based cognitive behavior therapy for insomnia intervention with 1-year follow-up: A aandomized clinical trial. *JAMA Psychiatry*. 2017;**74**(1):68–75.
64. Maich KHG, *et al*. Psychometric properties of the consensus sleep diary in those with insomnia disorder. *Behav Sleep Med*. 2018;**16**(2):117–134.
65. Levenson JC, *et al*. A quantitative approach to distinguishing older adults with insomnia from good sleeper controls. *J Clin Sleep Med*. 2013;**9**(2):125–131.
66. Lineberger MD, *et al*. Defining insomnia: quantitative criteria for insomnia severity and frequency. *Sleep*. 2006;**29**(4):479–485.
67. Blom K, *et al*. Internet-vs. group-delivered cognitive behavior therapy for insomnia: a randomized controlled non-inferiority trial. *Behav Res Ther*. 2015;**70**:47–55.
68. Lancee J, *et al*. Guided online or face-to-face cognitive behavioral treatment for insomnia: a randomized wait-list controlled trial. *Sleep*. 2016;**39**(1):183–191.
69. Thorndike FP, *et al*. Development and perceived utility and impact of an Internet intervention for insomnia. *E J Appl Psychol*. 2008;**4**(2):32–42.
70. Espie CA, *et al*. A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. *Sleep*. 2012;**35**(6):769–781.
71. Kyle SD, *et al*. Sleep restriction therapy for insomnia is associated with reduced objective total sleep time, increased daytime somnolence, and objectively impaired vigilance: implications for the clinical management of insomnia disorder. *Sleep*. 2014;**37**(2):229–237.
72. Davidson JR, *et al*. Characteristics of individuals with insomnia who seek treatment in a clinical setting versus those who volunteer for a randomized controlled trial. *Behav Sleep Med*. 2009;**7**(1):37–52.
73. Buysse DJ, *et al*. Efficacy of brief behavioral treatment for chronic insomnia in older adults. *Arch Intern Med*. 2011;**171**(10):887–895.
74. Andersson G, *et al*. Internet-delivered psychological treatments: from innovation to implementation. *World Psychiatry*. 2019;**18**(1):20–28.
75. Cooper AA, *et al*. Dropout from individual psychotherapy for major depression: a meta-analysis of randomized clinical trials. *Clin Psychol Rev*. 2015;**40**:57–65.