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Psychological Treatment of Insomnia in Hypnotic-Dependant Older Adults

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Abstract

Background—The existing literature does not address the question of whether cognitive-behavioral therapy would have an impact on insomnia in older adults who are chronic users of sleep medication and have current insomnia, but are also stable in their quantity of medication usage during treatment. The present report seeks to answer this question.

Methods—Hypnotic-dependant older adults, who were stable in their amount of medication usage and still met the criteria for chronic insomnia put forth by American Academy of Sleep Medicine, were treated using a cognitive-behavioral intervention for insomnia. The three-component treatment included relaxation training, stimulus control, and sleep hygiene instructions. Participants were randomly assigned to either the active treatment group or a comparably credible placebo control group, and were instructed not to alter their pattern of hypnotic consumption during treatment.

Results—The active treatment group had significantly better self-report measures of sleep at post-treatment. Statistically significant improvement was paralleled by clinically meaningful improvement for key sleep variables. As planned, there was no significant change in sleep medication usage from pre- to post-treatment.

Conclusions—The findings support the use of cognitive-behavioral therapy for insomnia in hypnotic-dependant older adults.

Keywords

insomnia; older adult; hypnotic dependant; psychological; treatment; RCT

Introduction

Data from our recent epidemiological study support the hypothesis that older adults (OA) report higher rates of insomnia and more severe insomnia than younger adults [1]. Across age groups, insomnia has been associated with difficulties such as impaired functioning, daytime fatigue,

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increased health care costs, and reduced quality of life [2,3]. These difficulties may be particularly salient for OA as they often experience other age-related decrements in general health. These findings highlight the importance of establishing optimum treatment strategies for insomnia in the OA population.

Hypnotic medication is typically the first line of defense for OA seeking treatment for insomnia, most likely because hypnotic medications are easily accessible through primary care physicians and promise rapid improvement for insomnia symptoms [4,5]. The rate of hypnotic usage for OA is approximately 10-15%, a rate nearly five times that of younger adults [6,7]. Although hypnotic medications have been shown to be effective in many cases, tolerance may lead to an attenuation of efficacy over time.

Psychological treatments such as cognitive-behavioral therapy (CBT) provide a second avenue of treatment for OA with insomnia. CBT for insomnia (CBTi) often consists of a combination of several techniques such as stimulus control, sleep restriction, sleep hygiene instruction, and relaxation practice. Individual studies have supported the use of CBTi with the OA population [6,8,9], and a recent Cochrane review of non-pharmacological therapies for sleep problems in later life concluded that CBTi was mildly effective in the short term but unproven in regards to long-term effects [10].

There is less published research regarding CBTi outcomes in OA who are chronic users of hypnotics. Recent results from a study using a mixed age population suggest that chronic users of sleep medication respond to CBTi [11]. The authors reported that in a hypnotic-dependant, mixed-age sample, participants significantly improved on global sleep quality as measured by the Pittsburgh Sleep Quality Index (PSQI) and reduced medication usage relative to a 'no additional treatment' control group. Other CBTi treatment research with hypnotic-dependant OA focused on withdrawal from those medications. For instance, Baillargeon and colleagues [12] reported that gradual benzodiazepine tapering in combination with CBTi was more effective in reducing benzodiazepine usage relative to benzodiazepine tapering regimen alone. Morin and colleagues [13] reported that CBTi given to hypnotic-dependant OA led to reduced medication usage and improved sleep in the form of self-report measures, including decreased total wake time, increased sleep efficiency, increased total sleep time, and decreased sleep onset latency.

The existing literature does not address the question: What impact does CBTi have on OA who are chronic users of sleep medication and have current insomnia but are also stable in their quantity of medication usage during treatment? As some OA may be hesitant to withdraw from their sleep medications but also want more improvement in their sleep, an exploration of CBTi in conjunction with stable medication use is called for. The lack of a psychological placebo control group has been a weakness in previous CBTi research with hypnotic-dependant OA. Thus, the current study is a randomized, placebo-controlled trial of CBTi in hypnotic-dependant OA that are stable in their amount of hypnotic use. It is hypothesized that this population will benefit from CBTi.

Method

Participants

The sample was a subset of participants recruited for a larger study of withdrawal from chronic hypnotic use. The relationship between the present study and the larger one will be described in more detail in the "Procedure" section below. Older adults, defined as age 50 years or older, with chronic insomnia, and regularly using prescription sleep medication, were recruited by newspaper announcements.

Participant Screening

A four-stage screening process established whether potential participants met the criteria for inclusion into the study. Stages included a 20 minute phone screen, an in-person interview, two weeks of sleep diaries, and a polysomnographic (PSG) assessment.

Chronic insomnia was defined in accordance with criteria put forth by the American Academy of Sleep Medicine [14] as difficulty initiating or maintaining sleep, concomitant with impaired daytime function for at least six months. We also included a quantitative component that required sleep onset latency (SOL) or wake time after sleep onset (WASO) to be in excess of 30 minutes at least three days per week [15].

To validate reports of insomnia, potential participants filled out a sleep diary (given in [1]) upon waking in the morning for a two-week period. The diary provided a subjective assessment of the time each participant entered bed the night before, how long it took him or her to fall asleep, number of nocturnal awakenings, the time spent awake during those awakenings, the time he or she woke up and rose from bed in the morning and an opinion of sleep quality.

To establish the presence of daytime impairments, potential participants filled out the following five questionnaires and met or exceeded the listed cutoff scores for at least one of them: Epworth sleepiness scale (ESS, [16]), score of 7.4; Insomnia Impact Scale (IIS, [17]), score of 125; Fatigue Severity Scale (FSS, [18]), score of 5.5; Geriatric Depression Scale (GDS, [19]) score of 10; and State-Trait Anxiety Inventory, Trait Scale (STAI, [20]), score of 37. The justification for these cutoffs, except for the GDS, is given in [15]. The cutoff for the GDS was established by using the threshold for mild depression (score of 10) described by Yesavage and colleagues [19].

Any prescription medication used specifically to improve sleep was allowed for inclusion into the study as long as use was sustained and frequent. Sustained use was defined as at least the past six months, and frequent use defined as at least three times per week. Volunteers using more than one prescription medication were also accepted. Potential participants must have also reported an interest in reducing their sleep medication.

The following also served as exclusionary criteria: the presence of other sleep disorders, a history of seizures, the presence of psychiatric or medical conditions that likely affected sleep, the use of sleep-active medications (for the past month) other than the designated hypnotic, or the use of high levels of substances (such as caffeine, nicotine, or alcohol) that impact sleep. Potential participants were excluded from participation if they admitted 1) consumption of caffeinated beverages past 2 pm on a regular basis, 2) smoking 10 or more cigarettes on an average day, or 3) consumption of more than four alcoholic beverages per week or consuming alcoholic beverages at bedtime once per week.

We used the Cornell Medical Index (CMI, [21]) to assess general physical health. The CMI consists of roughly 220 questions about a wide range of medical symptoms and illnesses. We followed up on any medical symptoms or conditions that may have affected the sleep of potential participants. If his or her insomnia severity mirrored a changing course of a medical problem, then the individual was disqualified.

We used the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Fourth edition (DSM-IV) (SCID), clinical version, for Axis I [22] and Axis II [23] to screen for psychiatric illness. If potential participants met the criteria for an Axis I or II disorder, we carefully explored its contribution to insomnia. Participants were screened out if the psychiatric disorder was deemed contributory.

We used the Mini Mental State Exam (MMSE, [24]) to make sure potential participants were capable of meeting the cognitive demands of taking part in the study. We chose a conservative score criterion of 26 for inclusion in the study. However, we were more flexible with individuals who had less than a ninth-grade education because research has suggested that performance on this exam is hindered by lower educational experience [25]. Thus, we chose a conservative cut-off score of 20 for those individuals who had a less than ninth-grade education.

Potential participants who showed clinically significant sleep apnea or periodic limb movements during PSG assessment were excluded from the study and referred for proper treatment. On the second night of each set of PSG assessments a urine sample was collected. Those potential participants who were positive for sleep medications other than the benzodiazepine of interest, or other drugs with sleep-active properties, were disqualified. These steps were overseen by a consulting physician board-certified in sleep medicine.

Research Setting and Apparatus

The study took place at two settings: The Sleep Disorders Center, Methodist Hospital and the Psychological Services Center, University of Memphis.

PSG sleep assessment was accomplished at the Sleep Disorders Center with Nihon Kohden #4212 polygraphs and Respironics' Alice 3 Infant and Adult computerized PSG system. Channels of PSG included two electroencephalographic (EEG), two electro-oculographic (EOG), and chin electromyographic (EMG). Auxiliary channels included oxygen saturation, bilateral anterior tibialis EMG, heart rate, thoracic strain gauge and an oral/nasal thermistor.

Electrode placement and sleep stage scoring was based on standards set by Rechtschaffen and Kales [26]. Each record was scored in 30-second epochs by a registered PSG technician. A subset (25%) of randomly selected records was scored independently by a second technician. There were meetings between the technicians and the primary investigator to compare the double-scored records and assess reliability and quality of the scoring.

Dependent Measures

The sleep variables of interest were sleep diary measures of sleep onset latency (SOL), number of awakenings (NWAK), wake time after sleep onset (WASO), total sleep time (TST), and sleep efficiency (SE). SE is the amount of time spent asleep divided by the amount of time spent in bed. The sleep diary also contained a sleep quality rating (SQR) that is a simple five-point rating of sleep quality ranging from poor (1) to excellent (5). All of these variables were obtained directly or calculated from data provided by the participants on sleep diaries. The five daytime function questionnaires described above were also used as outcome measures. Finally, the Medical Outcomes Study Short-Form Health Survey (SF-36, [27]) was used as a dependent measure of general health.

Treatment Conditions

The treatment took place during eight individual sessions that lasted about one hour and were spaced approximately one week apart. The treatment consisted of three well-established components: relaxation, stimulus control and sleep hygiene. Relaxation techniques were used to calm the mind and ease body tension, in order to minimize sleep-prohibiting worry and physiological arousal. This component was a 10-minute progressive but passive technique that focused on 16 different body parts [28]. Deep breathing and autogenic phrases were also used to help create and maintain a serene and accepting attitude.

The second component was stimulus control, which consisted of several instructions that help re-establish sleep-promoting associations with the bed and bedroom [29]. The participants were

first instructed to use the bed and bedroom for sleep and sex only. They were also told to leave the bed when they were not sleepy, were unable to fall asleep, or were engaging in a behavior, such as worrying, that was not compatible with sleep. They were also instructed to go to bed only when tired and maintain a regular wake time in the morning. The goal was to re-establish only sleep-promoting associations with the bedroom environment so that the bedroom becomes a cue only to sleep.

The third component was sleep hygiene, a set of five instructions designed to eliminate behaviors that are incompatible with sleep [30]. The instructions were as follows: avoid caffeine after noon, avoid exercise within two hours of bedtime, avoid nicotine within two hours of bedtime, avoid alcohol within two hours of bedtime, and avoid heavy meals within two hours of bedtime.

The placebo condition was a sham biofeedback treatment that closely mimicked a legitimate biofeedback regimen. The participants believed that they were learning to alter their brainwaves into a more sleep compatible rhythm. However, to make the placebo treatment inert, the brain wave feedback played back for the participant was actually a pre-recorded set of someone else's feedback. This placebo treatment reproduced a majority of the non-specific treatment factors of our active treatment but removed any active components. The only non-specific treatment factor not present in the placebo condition was a day-to-day homework practice.

Therapists

Advanced doctoral students in clinical psychology served as therapists. All therapists treated roughly the same number of participants from each treatment group. Each was trained per the parameters outlined in the following section.

Treatment Implementation Variables

Steps were taken based on our treatment implementation model [31] to ensure the treatment was delivered as intended (delivery), was comprehended by the participant as intended (receipt), and was practiced by the participant as intended (enactment). Consistent and accurate delivery was accomplished by the use of a detailed treatment manual and mock training sessions for all therapists. Quality and consistency of delivery was also assessed by listening to tapes of treatment sessions and giving constructive feedback to therapists. A stimulus control quiz and relaxation ratings were used to assess receipt, and compliance logs for each treatment component were used to assess enactment.

Treatment Credibility

Four statements that reflected dimensions of reasonableness of treatment, expectation for improvement, opinion of therapist, and willingness to recommend the treatment to others were rated on a 10-point scale, with higher ratings reflecting higher credibility.

Procedures

Volunteers received an initial telephone screening interview and those who passed moved on to a more thorough in-person interview at the University of Memphis Psychological Services Center, where informed consent was signed. Those volunteers who passed these screenings were given 14 nights of sleep diaries that were to be returned upon completion in a pre-stamped envelope. This procedure allowed for the verification of insomnia and served as a baseline measure of subjective sleep. Qualifying participants were randomly assigned to the two treatment conditions.

Baseline—After completion of baseline sleep diaries, participants submitted to two consecutive PSG assessments, including urine screens on night two. Participants determined their own lights-out time and arise time, with instructions to remain on their normal sleep/wake schedule.

Treatment—Treatment began within two weeks after completion of the baseline PSG. Participants were instructed to maintain baseline levels of hypnotic consumption throughout the treatment period. The treatment credibility questionnaire was given at the beginning of the third treatment session. This time point was selected because it gave the participant enough experience to make an informed judgment, but not enough experience to let treatment response bias the responses.

Post-treatment assessment—The measures described in the dependent measures section served as post-treatment measures and were given immediately following the CBTi treatment period.

It should be noted that the present report focused only on the CBTi treatment phase of the larger study that included a medication withdrawal regimen after CBTi. During the CBTi treatment phase, participants were instructed to maintain their baseline levels of medication usage. As such, a consistent level of medication use was expected between baseline and post-treatment assessments described in the present report.

Results

Demographic Variables

Groups were compared on the following demographic variables. Age in the treatment group ranged from 51 to 85 years ($M = 63.5$, $SD = 8.7$), and in the placebo group from 50 to 70 years (mean (M) = 64.82, standard deviation (SD) = 6.5); a between-subjects t-test revealed no significant difference between groups [$t(45) = 0.96$, ns]. Education level in the treatment group ranged from 12 to 22 years ($M = 14.7$, $SD = 2.7$), and in the placebo group from 12 to 20 years ($M = 14.9$, $SD = 2.3$) [$t(42) = -0.10$, ns]. In regards to gender, 12 of 20 participants in the treatment group and 18 of 27 in the placebo group were female. A chi-square test of independence revealed no significant difference between groups for gender [$X^2(1, n = 47) = 0.23$, ns]. In regards to ethnicity, 1 of 20 participants in the treatment group and 2 of 27 in the placebo group were African American [$X^2(1, n = 27) = 0.12$, ns].

Medication Usage

A within-subjects t-test revealed no significant change in quantity of medication use between baseline and post-treatment for the CBTi or placebo groups [$t(16) = 1.94$, ns, $t(21) = 0.32$, ns, respectively]. To compare medication types used between groups, type was categorized as follows: long-acting benzodiazepine, short-acting benzodiazepine, non-benzo-benzodiazepine agonist, sedating antidepressant, or other. A chi-square test of independence indicated that the frequency distribution of drug type was not significantly different between groups [$X^2(3, n = 42) = 1.04$, ns]. It should be noted that several participants in each group were using more than one hypnotic medication. There were four participants in the active treatment group using multiple medications and five in the placebo group [$X^2(1, n = 42) = 0.06$, ns].

Insomnia Type

The following four types of insomnia were reported by participants in the current study: initial, middle, mixed and combined. Initial insomnia is defined by having SOL as the primary sleep complaint at least three times per week, middle insomnia by the primary sleep complaint being WASO at least three times per week, mixed insomnia by having a mix of SOL and WASO

complaints at least three times per week, and combined by having three nights each of SOL and WASO per week. A chi-square test of independence revealed no significant difference between the frequency distribution of insomnia type between groups [$X^2(4, n = 40) = 4.85, p > 0.05$].

Sleep Variables

To control for differences in baseline values and correlations between baseline and post-treatment measures, post-treatment values were compared with baseline values as covariables. Analysis of covariance (ANCOVA) revealed significant differences between treatment and placebo groups at post-treatment for three sleep measures: SOL, $F(1,39) = 4.46, p < 0.05$, adjusted means and standard error for the treatment and placebo groups were $M = 19.39$, standard error = 3.94, and $M = 30.91$, standard error = 3.76, respectively; WASO, $F(1,39) = 4.55, p < 0.05$, adjusted means and standard error for the treatment and placebo groups were $M = 26.02$, standard error = 4.18, and $M = 38.38$, standard error = 3.99, respectively; SE, $F(1,39) = 9.49, p < 0.05$, adjusted means and standard error for the treatment and placebo groups were $M = 86.72$, standard error = 1.72, and $M = 79.40$, standard error = 1.64, respectively. For all three measures, the treated group exhibited better post-treatment sleep than the control group.

Clinical Significance

Effect sizes were calculated to assess clinical significance for statistically significant sleep variables. Medium effect sizes were found for SOL ($d = 0.55$) and WASO ($d = 0.53$). For SE, a composite variable that well summarizes the night's sleep, the effect size was large ($d = 0.89$).

Daytime Function Variables

Daytime function measures were analyzed the same way as sleep measures. ANCOVA revealed no significant differences between groups for scores on the STAI, ESS, GDS, IIS, or SF-36. Quizzically, the treatment group reported significantly higher levels of fatigue than the placebo group [$F(1,34) = 5.73, p < 0.05$].

Treatment Credibility

Differences in credibility ratings between groups were analyzed using t-tests. There were no significant differences between groups on any of the four statements used to assess credibility: reasonableness of treatment [$t(40) = 1.55, ns$], likeability of and confidence in therapist [$t(40) = -0.36, ns$], expectation of improvement [$t(40) = 1.094, ns$], and would recommend the treatment to a friend [$t(40) = 0.905, ns$].

Discussion

We found that CBTi led to significant improvements in subjective SOL, WASO, and SE in hypnotic-dependant OA. Significant improvements in these variables were paralleled by medium effect sizes for SOL and WASO, and a large effect size for SE. The magnitude of these effect sizes suggests that the results were not only statistically significant but also clinically meaningful. These results suggest that CBTi can be a valuable treatment for hypnotic-dependant OA who do not wish to withdraw from their sleep medications.

Improvement in sleep variables was not accompanied by comparable gains in daytime function. This finding is not unusual in insomnia treatment research. Previous research results have been equivocal regarding this issue. For example, Means and colleagues [32] and Lichstein and colleagues [33] reported a disengagement between sleep changes and daytime function, while Morin and colleagues [13] found improvements in symptoms of anxiety and depression after

successful insomnia treatment. Data from our recent epidemiological study also suggest that there is a weak and inconsistent relationship between sleep and daytime function in persons with insomnia and persons without [1].

The only daytime function measure that showed a significant difference between treatment and control groups at post-treatment was the FSS. With this questionnaire, the control group actually reported lower levels of fatigue than the treatment group. The reason for this result is unclear, and the importance of this finding is limited because both groups scored well below a threshold associated with clinically relevant impairment.

Several aspects of design and analysis strengthen our confidence in the current results. Participants were randomly assigned to treatment conditions after a thorough physical and mental health screening and PSG assessment. Analyses of demographic and other participant variables helped rule out the possibility that they contributed to differences between the treatment and placebo groups. Our placebo control group is perhaps the most important and unique strength of the current design. Our sham biofeedback treatment was highly credible, therapeutically inert, and mimicked most of the structural characteristics of the active treatment. Thus, we believe that it was an ideal placebo for CBTi.

Despite these strengths, several weaknesses in the current study should be considered. First, the design of the larger study from which the data was taken included a medication withdrawal program before any follow-up data were collected. As the goal of the present report was to focus on treatment outcome independent from any effect of reduction in medication usage, the withdrawal portion of the larger study would have served as a confound. Thus, we have no data representing long-term outcomes for hypnotic-dependant OA treated with CBTi. Second, the current report of post-treatment improvements is based solely on subjective measures. Objective measures of sleep change in the form of PSG data would have strengthened any conclusions drawn. The n in the current study was relatively small. A larger n would have allowed for follow-up analyses that explored the influence of such variables as insomnia type, medication type, gender, and ethnicity. Finally, it should be mentioned that the rigorous screening methodology used limits the generalizability of present results, especially in regards to individuals consuming moderate levels of nicotine and alcohol.

In conclusion, clinicians may want to consider treating the insomnia of hypnotic-dependant OA with CBTi without weaning patients from their medications. This option may be particularly useful to clinicians who do not have the medical supervision necessary to implement a medication withdrawal program or are working with OA who resist discontinuation of medication use. Further research in this area should include objective measurement of sleep variables, long-term assessment of treatment outcomes, and a more representative sample.

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Table 1
Pre- and Post-Treatment Means and Standard Deviations for Sleep Measures in Treatment and Placebo Groups

Sleep Measure	CBTi				Placebo			
	Pre-Treatment		Post-Treatment		Pre-Treatment		Post-Treatment	
	M	SD	M	SD	M	SD	M	SD
SOL *	44.61	35.77	19.85	15.37	41.42	22.63	30.50	22.09
NWAK	2.24	1.26	1.80	1.12	2.27	1.07	1.77	.77
WASO *	71.55	84.94	26.92	18.68	58.07	27.76	37.56	21.22
TST	353.16	81.45	408.08	50.22	355.29	54.34	404.84	52.16
SE *	72.87	15.69	86.80	7.37	72.36	7.98	79.32	9.25
SQR	2.73	.66	3.60	.52	2.79	.64	3.30	.62

* Analysis of covariance (ANCOVA) revealed significant differences between groups at post-treatment when differences in pre-treatment values were controlled for.