Cognitive Behavioral Therapy vs Zopiclone for Treatment of Chronic Primary Insomnia in Older Adults
A Randomized Controlled Trial

Børge Sivertsen, PsyD
Siri Omvik, PsyD
Ståle Pallesen, PhD
Bjørn Bjorvatn, MD, PhD
Odd E. Havik, PhD
Gerd Kvale, PhD
Geir Høstmark Nielsen, PsyD
Inger Hilde Nordhus, PhD

Context Insomnia is a common condition in older adults and is associated with a number of adverse medical, social, and psychological consequences. Previous research has suggested beneficial outcomes of both psychological and pharmacological treatments, but blinded placebo-controlled trials comparing the effects of these treatments are lacking.

Objective To examine short- and long-term clinical efficacy of cognitive behavioral therapy (CBT) and pharmacological treatment in older adults experiencing chronic primary insomnia.

Design, Setting, and Participants A randomized, double-blinded, placebo-controlled trial of 46 adults (mean age, 60.8 y; 22 women) with chronic primary insomnia conducted between January 2004 and December 2005 in a single Norwegian university-based outpatient clinic for adults and elderly patients.

Intervention CBT (sleep hygiene, sleep restriction, stimulus control, cognitive therapy, and relaxation; n=18), sleep medication (7.5-mg zopiclone each night; n=16), or placebo medication (n=12). All treatment duration was 6 weeks, and the 2 active treatments were followed up at 6 months.

Main Outcome Measures Ambulant clinical polysomnographic data and sleep diaries were used to determine total wake time, total sleep time, sleep efficiency, and slow-wave sleep (only assessed using polysomnography) on all 3 assessment points.

Results CBT resulted in improved short- and long-term outcomes compared with zopiclone on 3 out of 4 outcome measures. For most outcomes, zopiclone did not differ from placebo. Participants receiving CBT improved their sleep efficiency from 81.4% at pretreatment to 90.1% at 6-month follow-up compared with a decrease from 82.3% to 81.9% in the zopiclone group. Participants in the CBT group spent much more time in slow-wave sleep (stages 3 and 4) compared with those in other groups, and spent less time awake during the night. Total sleep time was similar in all 3 groups; at 6 months, patients receiving CBT had better sleep efficiency using polysomnography than those taking zopiclone.

Conclusion These results suggest that interventions based on CBT are superior to zopiclone treatment both in short- and long-term management of insomnia in older adults.

Trial Registration clinicaltrials.gov Identifier: NCT00295386
JAMA. 2006;295:2851-2858 www.jama.com

Among primary care physicians, the treatment of choice for insomnia has commonly been pharmacological intervention. The short-term efficacy of sleep medications has been demonstrated in numerous studies. How-
ever, a recent meta-analysis of sleep medications in older patients with insomnia concluded that effect sizes and clinical benefits both were small.

Furthermore, a consensus statement by the National Institutes of Health concluded that, while pharmacological treatments may be useful for acute and situational insomnia, long-term use involves risks of dependency and tolerance. There is also some current controversy in the media about the increasing use of zolpidem in the United States, related to next-day sleepiness and traffic collisions.

Cognitive behavioral therapy (CBT) is the most widely used psychological intervention for insomnia. Three meta-analyses have concluded that 70% to 80% of middle-aged adults with insomnia benefit from interventions based on CBT, and a study by Jacobs and colleagues suggested that young- and middle-aged individuals with insomnia improve more from CBT compared with pharmacotherapy. However, a Cochrane review stated that there is limited evidence available suggesting a clear effect of CBT on insomnia in older adults. Compared with placebo and no treatment, CBT was associated with sleep improvements, but effects were not as great as for CBT used with younger adults. Also, a recent meta-analysis concluded that behavioral interventions were more effective in the younger cohort in improving both total sleep time and sleep efficiency. However, all studies in this arena have been based on self-report or actigraphy. Surprisingly, only 1 randomized controlled clinical trial, by Morin and colleagues, has directly compared the clinical efficacy of both sleep medication (temazepam) and psychological interventions in older patients with primary insomnia. This study demonstrated that CBT and pharmacological interventions produced similar short-term effects but that CBT was superior at follow-up as indicated by sleep diaries. However, no studies have compared the newer nonbenzodiazepine sleep medications with nonpharmacological treatments and, to our knowledge, no studies have examined whether CBT affects slow-wave sleep (stages 3 and 4) in treating insomnia. This is of particular interest because lack of slow-wave sleep has been believed responsible for much of the daytime impairment experienced by patients with insomnia.

The present study was a randomized controlled trial conducted to evaluate the short- and long-term clinical efficacy of both CBT and the non-benzodiazepine sleep medication zopiclone. In contrast to previous research, we used both polysomnography (PSG) and sleep diaries at all 3 assessment points including follow-up. Providing independent estimates of sleep and wake time in addition to classification of sleep stages, the inclusion of PSG at follow-up was of particular importance because patients' subjective perceptions of actual sleep time have deviated from PSG-based recordings. We also wanted to compare the treatment conditions in their ability to improve slow-wave sleep.

**METHODS**

**Participants**

Participants were recruited through newspaper advertisements that stated the aim of the study as comparing the effects of sleep medications with psychological treatment. No additional information about the study hypothesis or type of interventions was provided.

Inclusion criteria were that participants (1) be 55 years or older; (2) fulfill the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria for insomnia, including difficulties initiating sleep, maintaining sleep, and/or early morning awakenings with no ability of return to sleep; (3) have insomnia duration of at least 3 months insomnia; and (4) complain of impaired daytime functioning.

The following exclusion criteria were used: (1) use of hypnotic medication in the last 4 weeks before project inception; (2) use of antidepressive or antipsychotic medications; (3) signs of dementia or other serious cognitive impairment defined by a score of less than 23 on the Mini-Mental State Examination; (4) presence of a major depressive disorder or other severe mental disorder as identified by a clinical assessment based on the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; (5) presence of sleep apnea defined as apnea-hypopnea index greater than 15 or periodic limb movements during sleep (PLM index with arousal >15); (6) working night shifts and unable or unwilling to discontinue this work pattern; (7) unwillingness or inability to stop taking sleep medication before study participation; or (8) having a serious somatic condition preventing further participation.

**Procedure**

Participants who responded to the advertisement (N = 92) underwent several screening processes before they were included. A 15-minute telephone interview by 2 clinical psychologists ensured that participants fulfilled the basic criteria for inclusion. Accepted participants (n = 75) met at the Department of Clinical Psychology, University of Bergen, for a structured clinical interview (SCID-I) screening for severe psychopathology and cognitive impairment. The final screening phase included 2 consecutive nights of ambulant polysomnography. Randomization was performed by the project leader using blocks of 3 with no stratification. After 12 participants had been assigned to each of the 3 conditions, blocks of 2 were used to randomly assign the remaining participants into 1 of the active treatments (CBT or zopiclone). Allocation concealment was implemented using sealed, sequentially numbered boxes that were identical in appearance for the 3 treatment groups. All study personnel in contact with the participants were unaware of the randomization sequence. Double-blinding was achieved with pills with identical appearance, smell, and flavor containing either zopiclone or placebo. In all, 48 participants were randomized into either CBT (n = 18), hypnotics (7.5 mg of zopiclone each night; n = 18), or pharmacological placebo treatment (n = 12). However, 2 participants withdrew from the zopiclone condition immediately after randomization and were excluded from
the modified intent-to-treat analysis. The flowchart in Figure 1 outlines the design of the study.

**Instruments**

PSG. Sleep variables were assessed by ambulant clinical PSG performed in the participants’ homes. The PSG montage included electroencephalographic, electromyographic, and electro-oculographic monitoring.

Sleep stages, respiratory disturbances, and limb movements were scored according to standard criteria by 2 technicians blinded to the participants’ condition. Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis electromyographic readings were recorded to detect sleep apnea or periodic limb movements. Participants underwent 2 consecutive nights of PSG at pretreatment to allow for adaptation to the PSG montage. At both posttreatment and follow-up assessment, only 1 night of PSG was recorded because recent studies have demonstrated that the so-called “first night effect” is only present in the first night in the first assessment period. All electrophysiological signals were acquired using Embla A10 (Flaga-Medcare Somnologica 3.2 software package, Buffalo, NY).

The sleep outcome measures included total wake time (sum of sleep-onset latency, wake time after sleep onset, and early morning awakening), total sleep time, sleep efficiency (ratio of total time spent asleep to the actual time spent in bed, multiplied by 100), and slow-wave sleep (time spent in sleep stages 3 and 4 registered by PSG).

Sleep Diaries. Participants completed sleep diaries every morning for 2 weeks at all 3 assessment points. The sleep diary provided self-reported information about the same sleep parameters collected from PSG registration. To increase the reliability, data analysis was based on the mean scores compiled during the 2-week period.

**Treatment Conditions.** CBT. Participants receiving CBT attended 6 weekly individual treatment sessions, with each lasting approximately 50 minutes. The rationale of this treatment condition is based on a manualized multicomponent approach that includes several modules introduced at different stages in the treatment process. These components include sleep hygiene education, sleep restriction, stimulus control, cognitive therapy, and progressive relaxation techniques. Table 1 provides an overview of the treatment principles in the CBT condition. The therapy sessions were facilitated by 2 clinical psychologists (B.S. and S.O.) and administered at the outpatient university clinic be-
This hypnotic agent has a market share way during the last decade, and overall less likely to have con-foundings in the present study. Due to the nature of CBT, neither therapists nor participants were blinded to it.

Zopiclone. Zopiclone, first introduced in 1988, is a cyclopyrrolone derivative that is chemically unrelated to benzodiazepines or barbiturates. Zopiclone works by enhancing the actions of the neurotransmitter γ-aminobutyric acid and is a racemic mixture of 2 stereoisomers, only 1 of which is active. The active stereoisomer, eszopiclone, was introduced on the US market in April 2005, and although the dosages are different (7.5 mg of zopiclone is equivalent to about 3.75 mg of eszopiclone), the 2 drugs are identical in effect. Zopiclone has demonstrated efficacy equivalent to and in some cases greater than both long- and short-acting benzodiazepines.14,15 Zopiclone is documented to be well-tolerated in elderly patients and is generally less likely to produce adverse effects than benzodiazepines.15 Zopiclone was chosen because it has been the most commonly prescribed hypnotic in Norway for the last decade, and overall this hypnotic agent has a market share of 45% of the total sales of hypnotics and tranquilizers in Norway.60

Participants in the active sleep medication group were administered 7.5 mg of zopiclone by a physician. Participants met at the sleep laboratory every week for a 10-minute meeting to report any adverse effects and to obtain the following week’s dosage of 7 pills. No behavioral recommendations regarding sleep were given during these short meetings, and the main focus was on encouraging the participants to adhere to the treatment program. After treatment completion, the patients were given the opportunity to continue their medication for 6 additional months.

Placebo. Participants receiving placebo treatment were subject to the same treatment protocol as those in the active medication group. As with zopiclone, the placebo capsules were made of gelatin and there were no differences in appearance, smell, or flavor between the active and inactive pills. After 6 weeks, participants in the placebo group were immediately randomized into 1 of the 2 active treatment conditions. Thus, the present study provides no follow-up data after 6 months for the placebo condition. Data from participants who received an active treatment following the placebo condition were not considered in the 6-month statistical comparisons. The rationale for omitting these data was to compare solely the effects of CBT vs zopiclone without including participants who had received both placebo medication and subsequently either zopiclone or CBT. Because the zopiclone and placebo medications were administered in a standard double-blind fashion, neither the patients nor the therapists should have known whether the patient received the active or inactive medication, but patients and therapists were not asked to guess to which treatment patients were randomized.

**Ethics**

The study was approved by the National Data Inspectorate, the Norwegian Medicines Agency, and the Regional Committee for Medical Research Ethics in western Norway. Written informed consent was obtained from all participants included in this study. Participants received no payment to participate in the study, and they were informed that they could withdraw from the study at any time without stating the reason.

**Statistics**

We used SPSS statistical software (SPSS Inc, Chicago, Ill) for Windows 13 for all statistical analyses. Analysis of variance with Bonferroni post hoc comparison or Pearson χ² tests were used to examine demographic and clinical variables at pretreatment. Modified intention-to-treat analyses (excluding the 2 individuals randomized to zopiclone who withdrew before the study began) based on end point data were used throughout the study. Pretreatment data were brought forward and used as 6-week and 6-month data for participants who dropped out during treatment (n = 1), whereas 6-week data were used as follow-up for individuals lost during 6-month follow-up (n = 7). A 2 × 3 (time × intervention) analysis of covariance analysis with Bonferroni-corrected post hoc comparisons was used to investigate differences between the interventions in terms of treatment effects. Analysis of covariance was also used to examine the treatment effects at 6-month follow-up, and

---

**Table 2. Pretreatment Demographic and Clinical Variables of Participants Included in the Study**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Cognitive Behavioral Therapy (n = 18)</th>
<th>Zopiclone (n = 16)</th>
<th>Placebo (n = 12)</th>
<th>Total (n = 46)</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>59.8 (4.3)</td>
<td>61.3 (6.9)</td>
<td>61.8 (5.0)</td>
<td>60.8 (5.4)</td>
<td>.58</td>
</tr>
<tr>
<td>Sex, women/men</td>
<td>7/11</td>
<td>6/10</td>
<td>9/3</td>
<td>22/24</td>
<td>.09</td>
</tr>
<tr>
<td>Education, mean years (SD)</td>
<td>14.2 (2.1)</td>
<td>13.7 (2.1)</td>
<td>14.6 (3.3)</td>
<td>14.1 (2.4)</td>
<td>.67</td>
</tr>
<tr>
<td>Insomnia duration, mean y (range)</td>
<td>15.8 (2-43)</td>
<td>13.7 (3-35)</td>
<td>12.0 (1-30)</td>
<td>14.1 (1-43)</td>
<td>.66</td>
</tr>
<tr>
<td>Current smoker, No. (%)</td>
<td>7 (38.9)</td>
<td>10 (62.5)</td>
<td>3 (25.0)</td>
<td>20 (43.5)</td>
<td>.12</td>
</tr>
<tr>
<td>More than 2 cups coffee daily, No. (%)</td>
<td>6 (33.3)</td>
<td>5 (31.3)</td>
<td>4 (33.3)</td>
<td>15 (32.6)</td>
<td>.99</td>
</tr>
<tr>
<td>Body mass index, mean (SD)†</td>
<td>30.8 (6.6)</td>
<td>32.3 (8.7)</td>
<td>29.9 (6.1)</td>
<td>31.1 (7.0)</td>
<td>.70</td>
</tr>
<tr>
<td>Previously treated for insomnia, No. (%)</td>
<td>12 (66.7)</td>
<td>4 (25.0)</td>
<td>4 (33.3)</td>
<td>20 (43.5)</td>
<td>.04</td>
</tr>
<tr>
<td>Self-reported chronic condition, No. (%)‡</td>
<td>7 (38.9)</td>
<td>9 (56.3)</td>
<td>3 (25)</td>
<td>19 (41.3)</td>
<td>.24</td>
</tr>
<tr>
<td>Currently taking other medication, No. (%)§</td>
<td>6 (33.3)</td>
<td>4 (25.0)</td>
<td>9 (75.0)</td>
<td>19 (41.3)</td>
<td>.02</td>
</tr>
</tbody>
</table>

*P values are based on analysis of variance or Pearson χ² test.
†Calculated as weight in kilograms divided by height in meters squared.
‡Heart disease, hypertension, chronic pain, urinary tract problems, headache, migraine.
§Other than sleep medication or antidepressive or antipsychotic medications.
paired-sample t tests were used to compare the posttreatment and follow-up levels. The clinical significance of the treatment effects was estimated by the proportion of participants who reached PSG-recorded sleep efficiency level of at least 85%, and Pearson χ² tests were used to test for group differences. Within-group effect sizes (pooled SD) were calculated using the Cohen d formula. Multicomponent CBT-based treatments for older adults with insomnia have been shown to yield effect sizes varying up to 2.0 on some sleep variables. Hence, expecting a difference between multicomponent CBT and pharmacotherapy at posttreatment and follow-up equivalent to an effect size of approximately 1.0, with a power of 80% at P = .05, the number of participants needed in each group was estimated to be 17.

**RESULTS**

**Pretreatment**

Forty-five of the 46 participants originally enrolled in the study completed the 6-week treatment protocol (22 women, 24 men). Mean age was 60.8 (SD, 5.4) years (median, 59; interquartile range 6), and the duration of insomnia was on average 14.1 years (SD, 11.3) (median, 10; interquartile range 15). Age, sex, educational level, insomnia duration, smoking, caffeine intake, body mass index, comorbid chronic condition, or sleep measures did not differ significantly between the treatment groups at pretreatment assessment (Table 2). More participants in the CBT condition had previously received treatment for insomnia compared with the other conditions (P = .04), and more participants receiving placebo treatment were also taking other non–sleep-related medications compared with the CBT and zopiclone groups (P = .02) (Table 2). Mean PSG-registered sleep efficiency at pretreatment was 81.0 (SD, 10.5) across the treatment conditions, while the participants’ sleep diaries yielded a mean sleep efficiency of 66.2 (SD, 11.8).

**6-Week Follow-up**

PSG. Total wake time showed both a significant time effect (P < .001), indicating that the participants spent less time
awake during the night following treatment than prior to treatment (TABLE 3), and a significant time × group interaction (P<.001), indicating that treatment groups differed significantly. The total wake time for the CBT group improved significantly more than both the placebo group at 6 weeks and the zopiclone group at 6 weeks. The zopiclone group did not differ significantly from the placebo group (P = .62). Total wake time at 6 weeks was reduced by 52% in the CBT group compared with 4% and 16% in the zopiclone and placebo groups, respectively.

Total sleep time showed no significant time effects (P = .70), indicating that total sleep time did not change with treatment interventions. However, sleep efficiency demonstrated both a significant time effect (P = .005), and time × group interaction (P = .004), with the CBT group having significantly higher sleep efficiency at 6 weeks than the placebo group (P = .004). CBT was not significantly different from zopiclone (P = .09), and zopiclone was not significantly different from placebo (P = .62) (FIGURE 2).

The amount of PSG-recorded slow-wave sleep (stage 3 and 4) improved significantly over time in the CBT group compared with both the placebo (P = .03) and zopiclone groups (P = .002). The zopiclone group had significantly less slow-wave sleep after treatment compared with before treatment (P = .01).

Sleep Diary. Total wake time (P = .001), total sleep time (P = .003), and sleep efficiency (P < .001) all improved over time as recorded in participants’ sleep diaries, but no differences were seen by group (TABLE 3).

6-Month Follow-Up
PSG. Total sleep time increased significantly in the CBT group at 6 months compared with 6 weeks (P = .05). The zopiclone group showed no significant change at 6 months, maintaining improvements seen at 6 weeks (TABLE 3). Comparing the 2 active treatment conditions, total wake time, sleep efficiency, and slow-wave sleep were all significantly better in the CBT group than in the zopiclone group; total sleep time was not significantly different (TABLE 3).

Sleep Diary. Similar to PSG, the sleep diaries showed an increase in total sleep time in the CBT group at 6 months compared with 6 weeks of follow-up (P = .004). Total wake time declined in the CBT group compared with the zopiclone group (P = .03) (TABLE 3).

Clinical Significance
The clinical significance of the treatment effects in both active conditions was examined by calculating the proportion of participants who reached PSG-recorded sleep efficiency level of at least 85%. In the CBT group, 13 individuals (72%) had a sleep efficiency level of at least 85% at the 6-week posttreatment assessment, while 14 (78%) fulfilled this criterion 6 months after treatment completion, compared with 6 (33%) at pretreatment. In contrast, only 7 (47%) of the participants in the zopiclone group had a sleep efficiency of at least 85% at the 6-week posttreatment assessment (vs 6 [40%] at pretreatment), a proportion which declined to 6 (40%) at 6-month follow-up. The group differences were statistically significant both at posttreatment ($\chi^2 = 8.94; P = .01$), and follow-up ($\chi^2 = 4.89; P = .03$).

Treatment Attendance and Adherence
Participants rated their adherence to CBT, zopiclone, and placebo on a 5-point scale, ranging from 0 (“never”) to 5 (“every night”) showing to what extent they took the pills or followed the advice and instructions in the treatment condition. The attendance rate in the CBT condition was 100%, and participants in the zopiclone condition who cancelled an appointment were sent the week’s dosage by mail. Overall level of adherence across all treatment groups was high (mean 4.6 [SD, 0.6]). There were no significant differences between the CBT (mean 4.8 [SD, 0.1]) and zopiclone (mean 4.5 [SD, 0.9]) condition on self-reported adherence at posttreatment, while participants in the placebo condition scored lower than the CBT recipients (mean 4.3 [SD, 0.6]; P = .05). At 6-month follow-up, the adherence rate did not differ between the 2 active treatment groups, but had declined to 4.1 (SD, 0.3) (P < .001) in the CBT condition and 3.6 (SD, 1.3) in the zopiclone condition (P = .02 compared with 6-week posttreatment). All participants in both the CBT and the zopiclone condition reported that they had continued their treatment to some extent during the 6-month follow-up period. However, 3 participants (13%) in the zopiclone condition reported that they only took their sleep medication “some of the days” after posttreatment assessment, of which 1 participant stopped taking the drug 1 month after treatment completion. The remaining
We found that CBT was more effective immediately and long-term compared with both zopiclone and placebo in older adults with chronic primary insomnia. On average, participants receiving CBT improved their PSG-registered sleep efficiency by 9% at posttreatment, compared with a decline of 1% in the zopiclone condition, a difference that was both statistically and clinically significant. These improvements in the CBT group were maintained at 6-month follow-up. Furthermore, participants in the CBT group spent significantly more time in slow-wave sleep (stages 3 and 4) compared with the other conditions.

A Cochrane review concluded that CBT had only a mild effect on sleep problems in older adults.26 By contrast, our present findings of zopiclone to other sleep medications at follow-up assessment. However, the reliability and validity of patients' self-reported sleep diaries have previously been questioned, as patients' self-reported sleep time has been shown to deviate from findings based on PSG, ranging from underestimations to overestimations.32

There are some limitations to the present study. Only participants with chronic primary insomnia were included, and thus, our results may not generalize to patients whose sleep problems are secondary to psychiatric or medical conditions. It remains to be seen whether CBT for insomnia may yield similar positive results in primary care settings, in which sleep problems may be part of a more complex clinical picture. Also, one may argue that the average sleep quality of included participants at pretreatment assessment was relatively high (PSG-registered sleep efficiency of 81% across all treatment conditions). However, the participants' subjective reports based on sleep diaries yielded a sleep efficiency of 66%, indicating that they did experience their sleep as impaired. Also, no information was available to examine the prolonged treatment effects beyond the last follow-up assessment at 6 months after treatment completion. As zopiclone resulted in a decrease of 1% in the zopiclone group compared with 33% at pretreatment. Our findings that CBT recipients showed lasting improvements in slow-wave sleep (63.1 minutes at study inception to 84.4 minutes at follow-up) were striking, especially as zopiclone resulted in a decrease (76.8 minutes at study inception to 59.2 minutes at follow-up). This is an intriguing finding that needs to be replicated, as lack of slow-wave sleep may be responsible for impaired daytime functioning and sleepiness.31

In contrast to the results by Morin et al,30 we found no significant treatment effects in the pharmacological group, neither at 6-week posttreatment or at 6-months follow-up. Zopiclone showed no better effect than placebo and produced significantly less slow-wave sleep at posttreatment compared with pretreatment. This is somewhat surprising as numerous clinical trials have shown that short-term use of zopiclone is as effective as the older benzodiazepines in patients with insomnia.45 However, almost no studies have investigated the effects of zopiclone beyond 4 weeks, and we cannot rule out that participants in this condition may have developed tolerance when they were assessed after 6 weeks. On the other hand, 5 participants withdrew by 6 months and 2 were no longer taking the study drug, so these results should be replicated in additional 6-month or longer-term studies.

The observed discrepancies between changes in PSG and sleep diary–recorded sleep time in both active-treatment conditions should be noted. However, the reliability and validity of sleep diaries have already been questioned, as patients' self-reported sleep time has been shown to deviate from findings based on PSG, ranging from underestimations to overestimations.32

Regardless of these limitations, the present findings have important implications for the clinical management of chronic primary insomnia in older adults. Given the increasing amount of evidence of the lasting clinical effects of CBT and lack of evidence of long-term efficacy of hypnotics, clinicians should consider prescribing hypnotics only for acute
insomnia. At present, CBT-based interventions for insomnia are not widely available in clinical practice, and future research should focus on implementing low-threshold treatment options for insomnia in primary care settings. As recently demonstrated by Bastien et al., telephone consultations and CBT-based group therapy for younger patients with insomnia produced equally significant improvements as individual therapy sessions. In another study, CBT delivered via the Internet in a self-help format showed significant improvements in individuals with chronic insomnia.

In conclusion, this study demonstrated superior benefits of CBT over zopiclone for treatment of chronic insomnia in older adults at 6-week and 6-month follow-up. Future research should require effects in slow-wave sleep and define effects on daytime sleepiness.

Author Contributions: Dr Nordhus had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Svetsen, Omvik, Pallesen, Bjorvatn, Havik, Kvåle, Nielsen, Nordhus.
Acquisition of data: Svetsen, Omvik, Pallesen, Bjorvatn, Havik, Kvåle, Nielsen, Nordhus.
Data analysis and interpretation: Svetsen, Omvik, Pallesen, Bjorvatn, Havik, Kvåle, Nielsen, Nordhus.
Statistical analysis: Svetsen, Omvik, Pallesen, Havik, Nordhus.
Obtained funding: Pallesen, Nordhus.
Administrative, technical, or material support: Svetsen, Omvik, Pallesen, Bjorvatn, Havik, Kvåle, Nielsen, Nordhus.
Study supervision: Pallesen, Bjorvatn, Havik, Nordhus.
Financial Disclosures: None reported.
Funding/support: This research was funded by grants from the University of Bergen; the Melzer Fund, and the EXTRA funds from the Norwegian Foundation for Health and Rehabilitation.

Role of the Sponsors: The funding organizations had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.

REFERENCES

18. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. Health Psychol. 2006;25:3-14.