Characteristics of Patients with Subjective Sleep Problems after Cognitive Behavioral Therapy for Insomnia: Secondary Analyses of a Randomized Controlled Trial

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Abstract

Objective Cognitive behavioral therapy for insomnia (CBT-I) is the first-line treatment for insomnia, but half of the patients do not reach remission. This study aimed to explore subjective remission by investigating the characteristics of patients who reported lingering sleep problems after CBT-I.

Methods Secondary analyses of a randomized controlled trial of group CBT-I in 72 primary care patients with insomnia disorder. Sociodemographic characteristics and outcomes (insomnia severity, sleep variables, hypnotics use, fatigue, depressive symptoms, and dysfunctional beliefs/attitudes), including baseline data and symptom change, were investigated in relation to patients’ posttreatment response to the yes-or-no question “Would you say that you have sleep problems?”

Results A total of 56.9% of patients reported sleep problems after CBT-I. At baseline, they had worse depressive symptoms (14.9 (SD 7.5) vs. 10.2 (SD 5.9), $p = 0.006$) and more awakenings (2.6 (SD 1.5) vs. 1.8 (SD 1.3), $p = 0.034$) than those in subjective remission from sleep problems. Patients in the non-remission and remission groups showed similar improvements in sleep, fatigue, and depressive symptoms, but patients in the non-remission group had improved less in insomnia severity, dysfunctional beliefs/attitudes about sleep, and hypnotic use. In patients with more pronounced depressive symptoms before CBT-I, change in depressive symptoms during treatment partially explained subjective remission from sleep problems.
Introduction

Insomnia, found in approximately 10% of the population, is mainly assessed and treated in primary health care. This sleep disorder is experiential in nature and is diagnosed on the basis of subjective sleep problems and daytime impairments. Sleep-related worry, hyperarousal, and safety behaviors (e.g., taking a daytime nap) contribute to its persistence. Insomnia is often comorbid with health problems, such as diabetes, cardiovascular disease, chronic pain, depression, anxiety, and work-related burnout.

Cognitive behavioral therapy for insomnia (CBT-I) is the recommended first-line treatment for the disorder. This recommendation is based on a considerable body of scientific evidence showing that CBT-I is effective in adults regardless of age or the presence of a comorbid disorder, and that CBT-I is safer and more effective, especially over time, than hypnotics.

Despite the effectiveness of CBT-I, a significant proportion of people do not fully recover from insomnia after treatment. Following CBT-I, about 1/3 will not improve, and 2/3 will continue to have insufficient sleep (i.e., >30 min sleep onset latency and/or time awake after sleep onset). About half will fail to achieve clinically meaningful improvement.

A few studies have aimed to identify the characteristics of people who are most or least likely to benefit from CBT-I. These studies have found that sleep durations of <6 hours, lower subjective sleep quality, and longer sleep onset latency and/or time awake after sleep onset prior to treatment predict non-response or non-remission after CBT-I. Higher levels of anxiety and depression predict positive treatment response or insomnia remission.

There is no consensus about how to assess whether a person has responded to CBT-I or is fully recovered from insomnia after the treatment. Typically, the assessment of treatment response or insomnia remission is based on clinically significant symptom reduction assessed with symptom severity scales and/or with sleep diaries or objectively measured sleep. Examples of previously used definitions of treatment response or insomnia remission include a posttreatment score of <8 points on the Insomnia Severity Index (ISI), ≤5 points on the Pittsburgh Sleep Quality Index (PSQI), or ≤30 minutes of sleep onset latency or time awake after sleep onset (sleep diary) posttreatment; a change of ≥3 points on the PSQI; a 50% reduction in sleep onset latency and/or number of nocturnal awakenings; 50% reduction in time awake after sleep onset; or a 10% increase in sleep efficiency. Moreover, some studies have used posttreatment cut-off scores on the Insomnia Symptom Questionnaire (ISQ). A posttreatment sleep efficiency of >80 to 85%, and a total sleep time of >6.5 hours (sleep diary).

A few studies have measured treatment response based on patients’ experiences. One study assessed perceived sleep improvements with the Clinical Global Improvement Scale, in which participants rated themselves as very much improved to very much worse. Another study used a guided interview to identify people who no longer met the diagnostic criteria for insomnia after treatment.

Given the experiential nature of the disorder, this study aimed to explore subjective remission by investigating the characteristics of patients who reported lingering sleep problems after CBT-I. Sociodemographic characteristics and insomnia outcomes, including baseline data and symptom change, were investigated.

Materials and Methods

Participants

The participants in the present study were included in a randomized controlled trial of group CBT-I in primary health care. The control condition was treatment as usual (mainly hypnotics). The trial was conducted between 2011 and 2014 at 7 primary health care centers in the region of Stockholm, Sweden. Approval to conduct the trial was obtained from the Regional Ethical Review Board in Stockholm, Sweden. Approval to conduct the trial was obtained from the Regional Ethical Review Board in Stockholm, Sweden.

Patients were assessed for eligibility via structured interviews. They had to be ≥18 years and have symptoms consistent with the Diagnostic and Statistical Manual for Mental Disorders (DSM) criteria for insomnia. Patients with comorbid conditions were included unless they had severe untreated somatic and/or mental illness, bipolar disorder, and/or symptoms indicating an untreated sleep disorder other than insomnia (e.g., sleep apnea or restless legs syndrome). Other exclusion criteria included current stressful life events, such as death in the immediate family or life-threatening illness, shift work, an ISI score of <7, and language or cognitive difficulties. Ninety patients were randomized to CBT-I and 75 to treatment as usual. The present analyses focused on the CBT-I participants who completed treatment and responded to the yes-or-no question “Would you say that you have sleep problems?” posttreatment (n = 72). Patients in the control group who responded to the posttreatment question (n = 59) were included in a mediational analysis.
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**Intervention**

The intervention consisted of 7 sessions of nurse-delivered group CBT-I, including 6 weekly 2-hour sessions and 1 follow-up session 4 weeks later. The intervention was delivered by 8 nurses to, in total, 17 groups of patients. Each treatment group included four to seven patients. The treatment consisted of sleep education, relaxation, worry time, paradoxical intention, sleep restriction, stimulus control, stepwise reduction of hypnotic drugs, cognitive restructuring, stress reduction strategies, and strategies to cope with daytime symptoms. Patients who attended ≥ 5 sessions were considered to have received treatment.

**Data Collection and Variables**

**Background Variables**

Information on sociodemographic and clinical characteristics was collected during the individual structured interview before inclusion in the trial. These data included age, sex, educational status, employment status, marital status, current health problems, and duration of insomnia symptoms.

**Outcome Variable**

The outcome variable in this study was subjective remission status as defined by the participants’ posttreatment response to the question “Would you say that you have sleep problems?” Those who responded yes were categorized as in subjective non-remission from sleep problems, and those who responded no, as in subjective remission from sleep problems. The question is an item on the 27-item Uppsala Sleep Inventory (USI-27), which participants completed at baseline and posttreatment.

**Explanatory Variables**

Insomnia severity was assessed with the 7-item Insomnia Severity Index (ISI), which measures the severity of insomnia-related impairments and emotional distress during the prior 2 weeks on a Likert scale. Total scores range from 0 to 28. Higher scores indicate more severe problems.

Sleep was assessed with a 14-day sleep diary. Mean values were calculated for sleep onset latency, time awake after sleep onset, total sleep time, time spent in bed, sleep efficiency (total sleep time / time spent in bed * 100), number of nocturnal awakenings, and sleep quality (0 = very bad to 5 = very good).

Use of hypnotics was assessed with the question “How often do you use hypnotic drugs?” The possible answers were never (0), a few times a year (1), a few times a month (2), a few times a week (3), almost daily (4), and daily (5).

Fatigue was measured with the 9-item Fatigue Severity Scale (FSS). Responses range from strongly disagree (1) to strongly agree (7); higher scores indicate more severe problems. Total scores range from 0 to 63.

Depressive symptoms were assessed with the self-assessment version of the Montgomery-Asberg Depression Rating Scale (MADRS-S). MADRS-S asks about 9 domains: mood, feelings of unease, sleep, appetite, ability to concentrate, initiative, emotional involvement, pessimism, and zest for life. Each domain is followed by a list of statements that range from 0 (positive) to 6 (negative). Total scores range from 0 to 54; higher scores indicate more severe symptoms. Score from 0 to 12 indicate no depression; 13 to 19, mild depression; 20 to 34, moderate depression; and ≥ 35, severe depression.

As the MADRS-S sleep item might overlap with other sleep outcomes, we also computed a variable without the sleep domain (total scores range from 0–48).

Dysfunctional beliefs and attitudes were assessed with the 16-statement Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale. Each statement on this scale is accompanied by a 100-mm horizontal line with strongly disagree (0) on the left and strongly agree (100) on the right. The higher the score, the stronger the dysfunctional belief or attitude.

**Statistical Analyses**

Analyses were performed with IBM SPSS Statistics for Windows, version 26.0, (IBM Corp., Armonk, NY, USA). The significance level was 5% (two-tailed). Patients in the CBT-I group who completed treatment and responded to the outcome question at posttreatment were included in the analyses, which used observed data.

All variables were summarized with standard descriptive statistics, such as frequency, mean, and standard deviation (SD). To compare sociodemographic and clinical characteristics and outcomes in patients not in subjective remission and patients in subjective remission, the Pearson χ² (or Fisher exact test if expected cell frequency was 5 or less), or the Mann Whitney U-test was used to analyze categorical variables. The Student t-test was used to analyze continuous variables. Differences in change between the two groups from baseline to follow-up were analyzed with analysis of variance (ANOVA) for repeated measurements, in which differences in change between groups appear as a significant interaction effect (group * time).

The mediational analysis was conducted with the PROCESS computational tool, which was added to the IBM SPSS Statistics for Windows, version 26.0 (IBM Corp. Armonk, NY, USA). Treatment method (CBT-I and treatment as usual) was the predictor, and subjective remission status was the outcome. Bootstrap confidence intervals were chosen to run inference testing of indirect effects, as this method does not make assumptions about normal sampling distribution and has higher power than the Sobel test. Moreover, an advantage with the PROCESS addon to SPSS is that it is sensitive to detect reliable change in smaller samples.

**Results**

**Subjective Remission Status and Sociodemographic Characteristics**

At posttreatment, 72 patients who completed the CBT-I program responded to the question, “Would you say that you have sleep problems?” The 56.9% (n = 41) who responded yes were categorized as not in subjective remission (non-remission), and the 43.1% (n = 31) who responded
no, as in subjective remission. All had reported sleep problems at baseline.

At baseline, there were no significant differences in sociodemographic or clinical characteristics (sex, age, educational level, employment status, marital status, current health problems, and duration of insomnia) between patients in the non-remission group and the remission group after CBT-I (► Table 1).

**Outcomes and Subjective Remission Status**

Before treatment, patients in the non-remission group had significantly worse depressive symptoms (MADRS-S, \( p = 0.006 \)) after CBT-I than those in the remission group (► Table 2). A total of 58.8% (n = 24) in the non-remission group and 22.6% (n = 7) in the remission group had a MADRS-S score that indicates mild to moderate depression (\( p = 0.002 \)). The results of complementary analyses with the MADRS-S sleep item removed were almost identical to the full-scale results (data available on request). The results of the full-scale analyses are thus presented here. Patients in the non-remission group did not differ in sleep diary variables at baseline, except that they had significantly more frequent nocturnal awakenings than the remission group (\( p = 0.034 \)).

There were no significant differences between the non-remission group and the remission group in improvements after CBT-I in sleep onset latency, time awake after sleep onset, sleep duration, sleep efficiency, awakenings, sleep quality, fatigue, or depressive symptoms (► Table 2). However, patients in the non-remission group improved less in insomnia severity (\( p = 0.001 \)), decreased their use of hypnotics less (\( p = 0.012 \)), and decreased less in dysfunctional beliefs and attitudes about sleep (\( p < 0.001 \)) than patients in the remission group.

**The Mediating and Moderating Role of Depressive Symptoms**

Because patients in the non-remission group had higher scores for depressive symptoms at baseline than those in...
the remission group, we tested change in the severity of depressive symptoms from baseline to posttreatment as a mediator of subjective remission status, using baseline depressive symptoms and insomnia severity as covariates (►Figure 1). The predictor variable was the treatment method (CBT-I, n = 71 vs. treatment as usual, n = 59). All patients in the control group who responded to the outcome question were, thus, included in the mediation analysis, whereof 100% reported sleep problems at baseline, versus 94.9% at posttreatment.

In a model that took baseline severity of depressive symptoms and insomnia into account, change in the severity of depressive symptoms during treatment mediated the relationship between treatment and subjective remission status (direct effect, $b = -2.58, CI -3.99, -1.16$; indirect effect, $b = -1.64, CI -3.39, -0.80$). Baseline severity of depressive symptoms was a significant predictor of subjective remission status ($p < 0.001$), whereas baseline severity of insomnia was not ($p = 0.265$).

### Discussion

The most salient characteristic of patients who still reported sleep problems after CBT-I was the relative severity of their depressive symptoms before entering treatment. This was true even though their improvements in most outcomes were comparable to those of patients in subjective remission. Some previous studies have found that higher levels of depressive symptoms at baseline are associated with better CBT-I outcomes,\(^{13,16}\) and some have found the opposite.\(^{18}\) When changes in depressive symptoms were investigated, the current study found that decreased severity of depressive symptoms during treatment partially explained subjective remission from sleep problems.

Pretreatment MADRS-S data showed that 24 of 41 patients had scores that suggested they had undiagnosed depression before starting CBT-I. Even for these patients, CBT-I was likely an appropriate treatment. Cognitive behavioral therapy for insomnia is effective in people with

### Table 2

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-remission (n = 41)</th>
<th>Remission (n = 31)</th>
<th>Student t-test</th>
<th>ANOVA(^b), group (^c), time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>$p$</td>
<td>$p$</td>
</tr>
<tr>
<td>Insomnia Severity Index (total score)</td>
<td>Baseline</td>
<td>18.8 (3.9)</td>
<td>17.4 (4.2)</td>
<td>0.140</td>
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<td></td>
<td>Posttreatment</td>
<td>11.8 (5.0)</td>
<td>6.4 (3.7)</td>
<td></td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>Baseline</td>
<td>67.0 (44.3)</td>
<td>67.5 (50.8)</td>
<td>0.969</td>
</tr>
<tr>
<td></td>
<td>Posttreatment</td>
<td>34.8 (20.4)</td>
<td>30.8 (18.6)</td>
<td></td>
</tr>
<tr>
<td>Time awake after sleep onset (min)</td>
<td>Baseline</td>
<td>99.3 (69.3)</td>
<td>74.3 (47.0)</td>
<td>0.088</td>
</tr>
<tr>
<td></td>
<td>Posttreatment</td>
<td>51.9 (42.0)</td>
<td>36.3 (31.6)</td>
<td></td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>Baseline</td>
<td>348.7 (60.0)</td>
<td>378.6 (68.8)</td>
<td>0.055</td>
</tr>
<tr>
<td></td>
<td>Posttreatment</td>
<td>381.9 (43.7)</td>
<td>409.7 (41.5)</td>
<td></td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>Baseline</td>
<td>69.1 (13.9)</td>
<td>74.0 (12.3)</td>
<td>0.130</td>
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<tr>
<td></td>
<td>Posttreatment</td>
<td>82.5 (7.6)</td>
<td>86.7 (7.3)</td>
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<tr>
<td>Nocturnal awakenings (nr)</td>
<td>Baseline</td>
<td>2.6 (1.5)</td>
<td>1.8 (1.3)</td>
<td>0.034</td>
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<tr>
<td></td>
<td>Posttreatment</td>
<td>1.7 (1.11)</td>
<td>1.3 (0.8)</td>
<td></td>
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<tr>
<td>Sleep quality</td>
<td>Baseline</td>
<td>2.7 (0.6)</td>
<td>2.9 (0.5)</td>
<td>0.073</td>
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<tr>
<td></td>
<td>Posttreatment</td>
<td>3.2 (0.6)</td>
<td>3.6 (0.5)</td>
<td></td>
</tr>
<tr>
<td>Fatigue Severity Scale</td>
<td>Baseline</td>
<td>38.8 (10.8)</td>
<td>35.2 (12.0)</td>
<td>0.188</td>
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<tr>
<td></td>
<td>Posttreatment</td>
<td>32.8 (12.0)</td>
<td>26.9 (13.7)</td>
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<td>Montgomery-Asberg Depression Rating</td>
<td>Baseline</td>
<td>14.9 (7.5)</td>
<td>10.2 (5.9)</td>
<td>0.006</td>
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<td>Scale – Self-assessment</td>
<td>Posttreatment</td>
<td>10.8 (5.9)</td>
<td>4.9 (3.8)</td>
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</tr>
<tr>
<td>Dysfunctional Beliefs and Attitudes</td>
<td>Baseline</td>
<td>53.8 (15.2)</td>
<td>47.8 (13.1)</td>
<td>0.226</td>
</tr>
<tr>
<td>about Sleep scale (total score)</td>
<td>Posttreatment</td>
<td>42.6 (18.7)</td>
<td>25.8 (11.1)</td>
<td></td>
</tr>
<tr>
<td>Frequency of hypnotic use</td>
<td>Baseline</td>
<td>2.5 (2.2)</td>
<td>2.8 (1.9)</td>
<td>0.529</td>
</tr>
<tr>
<td></td>
<td>Posttreatment</td>
<td>1.6 (2.1)</td>
<td>0.8 (1.5)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ANOVA, analysis of variance (general linear model); SD, standard deviation.

\(^a\)Subjective remission status was based on participants' response to the post-treatment yes-or-no question "Would you say that you have sleep problems?"

\(^b\)Number of observations varied due to attrition.
comorbid insomnia and depression, and treating insomnia has the potential to decrease depressive symptoms, as it did in the present study. It is noteworthy that an analysis of dropouts from the main trial showed that they had worse depressive symptoms than completers. Given the findings of the previous dropout analysis and the current study, it is possible that patients with worse depressive symptoms would have benefited from more targeted depression treatment, before or after CBT-I, or a more tailored insomnia treatment.

It is possible that the current CBT-I program was too comprehensive and general for patients with poorer mental health. Previous research suggests that CBT-I tailored to patients with pronounced depressive symptoms should include behavioral activation (a key element in CBT for depression) and more attention to dysfunctional beliefs.

The proxy for subjective remission in the present study may reflect aspects of remission beyond those captured by calculations and normative values, such as treatment satisfaction. Treatment satisfaction is associated with adherence to and outcomes of psychological treatments for insomnia. If depressive symptoms made it hard to adhere to treatment, patients may have experienced lower treatment satisfaction and lack of full recovery. In future studies, it could be useful to explicitly measure treatment satisfaction.

The non-remission and the remission groups did not differ notably in pretreatment sleep, and sleep improved significantly in both groups. This finding contrasts with the findings of previous studies, in which subjectively short and poor sleep were the main predictors of non-response or non-remission from insomnia after CBT-I.

Patients’ response to the subjective outcome question in this study may be related to their experiences of overall sleep quality. One study found that perceptions of sleep quality may reflect phenomena other than sleep, such as mental and physical health, which may explain the predicting role of depressive symptoms for subjective remission from sleep problems found in the present study. Another study on the subjective meaning of sleep quality in people with insomnia and normal sleepers found that people typically judged sleep quality based on feelings of fatigue, anxiety, worry, and mood on waking and during the day, and that those with insomnia have higher standards for what counts as good quality sleep. Patients in the non-remission group continued to have depressive symptoms after CBT-I and may, therefore, have felt that the sleep improvements were not sufficient to help them feel better during the day. The remission groups did not differ in fatigue severity before or during treatment. Thus, fatigue does not seem to have played a vital role in patients’ perceptions of sleep problems.

Both insomnia and depression seem to be maintained by negative thoughts and beliefs. For example, a study showed that a decrease in dysfunctional beliefs about sleep mediated the relationship between CBT-I and insomnia and CBT-I and depression. Negative beliefs may, therefore, perpetuate both symptomatologies. Our findings are consistent with this interpretation, as the patients who were not in subjective remission improved less in dysfunctional beliefs and attitudes about sleep than those in subjective remission. However, such beliefs at baseline were not a significant predictor for remission status in the present study.

To the best of our knowledge, this is one of few studies to explore subjective remission from sleep problems after CBT-I via participants response to a single yes-or-no question.
Some methodological shortcomings should be kept in mind when interpreting the results. This study is a secondary analysis of data from an RCT not designed to answer the current study question. Variables outside the original study’s scope might shed further light on factors associated with perceived remission. One unanswered question is whether the depressive symptoms in the non-remission group affected their adherence to therapy content and, thus, the study results. Previous studies have found that patients with depressive symptoms can have trouble adhering to some components of CBT-I. The reliability of the dichotomous measure to investigate remission has not been assessed previously, although it has been used to assess sleep problems in cross-sectional studies, and to determine the ISI cut-off score of 10 to detect clinical insomnia in a community sample (86.1% sensitivity and 87.7% specificity). However, the intention with the present study was to explore subjective remission based on the general and straightforward yes-or-no question, not to propose an additional measurement to determine remission from insomnia treatment.

In conclusion, regardless of patients’ subjective remission status, CBT-I improved sleep, fatigue, and depressive symptoms. However, still perceiving oneself as having sleep problems may lead to distress and to more health care seeking. The subjective reports are, therefore, important to consider. The findings that the severity of depressive symptoms prior to treatment and change in severity of depressive symptoms during treatment were related to whether patients continued to report that they had sleep problems, have some clinical implications. Screening for depressive symptoms prior to treatment might shed some light on treatment response. Further, patients with pronounced depressive symptoms may need more tailored treatment to perceive that they have fully recovered.

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Conflict of Interests
The authors have no conflict of interests to declare.

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