Psychological Treatment of Secondary Insomnia

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Psychological treatment of insomnia has focused on primary insomnia (i.e., having a psychological origin). Secondary insomnia, sleep disturbance caused by a psychiatric or medical disorder, although it is more common than primary insomnia, has received very little attention as a result of the belief that it would be refractory to treatment. The present study randomly assigned older adults with secondary insomnia to a treatment group, 4 sessions composed of relaxation and stimulus control, or a no-treatment control group. Self-report assessments conducted at pretreatment, posttreatment, and a 3-month follow-up revealed that treated participants showed significantly greater improvement on wake time during the night, sleep efficiency percentage, and sleep quality rating. The authors hypothesize that treatment success was probably due in part to difficulty in diagnostic discrimination between primary and secondary insomnia.

Chronic insomnia, characterized by delayed sleep onset, sustained awakenings during the night, or early morning awakening without returning to sleep, occurs in about 10% of the population (Ford & Kamerow, 1989). Insomnia is more common in women than men and occurs at disproportionately high rates in older adults (Mellinger, Balter, & Uhlenhuth, 1985). Individuals with insomnia not only suffer poor sleep but usually complain of impaired daytime functioning as well. Reports of sleepiness, fatigue, diminished physical well-being, mood disorder, and anxiety may all contribute to a compromised quality of life for these individuals (Riedel & Lichstein, in press).

The preponderance of the psychological research on insomnia has focused on psychophysiological insomnia, also known as primary insomnia, which is the formal name given to insomnia with a psychological basis (American Psychiatric Association, 1994; American Sleep Disorders Association, 1990). Typical causes of this form of insomnia are subclinical worry, blue mood, and/or conditioned arousal to the bedroom setting. Excluded by the definition of primary insomnia are individuals whose sleep disturbance is caused by a psychiatric or medical disorder. In the case of psychiatric disorders, the “psychological” disturbance is more severe and chronic than in primary insomnia, meriting recognition as a clinically significant condition, typified by anxiety or depression. The insomnia variant based on psychiatric or medical disturbance is given the formal diagnostic label of secondary insomnia (SI; American Psychiatric Association, 1994; American Sleep Disorders Association, 1990).

SI can arise from a wide range of sources. Many medical disorders can instigate insomnia, as exemplified by pulmonary disease, renal failure, heart disease, arthritis, and a variety of neurological diseases (Mitler, Poceta, Menn, & Erman, 1991; Wooten, 1989). Psychiatric disorders (Walsh & Sugarman, 1989), most notably anxiety and depression, can disturb sleep as well. The mechanisms of sleep disruption can vary. Insomnia may be a symptom of the illness itself, such as the vegetative symptom of early morning awakening in depression. Alternatively, symptoms associated with the illness, such as pain arising from cancer, may produce insomnia. Finally, insomnia may be a side effect of medications used to treat the primary condition. There are many prescribed medications that may cause insomnia, dependent on the dosage level, time of administration, age of the patient, and idiosyncratic response (Mitler et al., 1991; Monane, 1992). Examples are energizing antidepressants, anihypertensives, and bronchodilators.

Although it is difficult to estimate the prevalence of SI, it is not an uncommon disorder. A large epidemiological survey of older adults showed that both mental health and physical health complaints were strongly associated with the presence of insomnia, though no attempt was made to distinguish between comorbidity and a causal path supporting the diagnosis of SI (Foley et al., 1995). This study reminds us of the critical distinction between comorbidity and SI that we return to later in this article. Two disorders (e.g., insomnia and depression) are said to be comorbid when one does not necessarily influence the other, or there is a reciprocal interaction between them and one is not viewed as causing the other. To qualify as SI, the psychiatric or medical condition must be responsible for causing the insomnia.

Another epidemiological survey involved structured interviews that attempted to differentiate SI from comorbidity and estimated that 60% of people with insomnia had SI (Ohayon, 1997). The majority of individuals diagnosed with insomnia at sleep disorders centers are labeled as having SI (Lichstein, 2000b). However, SI has been largely ignored because conventional wisdom has assumed that the sleep disturbance is untreatable so long as the primary illness persists (Mendelson & Jain, 1995; National Insti-
tutes of Health, 1991; Walsh & Sugerman, 1989). This neglect has strongly characterized the research domain, but it is less clear that clinicians have been averse to offering insomnia treatment to these individuals.

There are health costs associated with the decision to leave SI untreated, and we question the prudence of withholding treatment of SI on three grounds. First, when a psychiatric or medical disease contributes to insomnia, some portion of the sleep disturbance may also be independent of the primary disease. Many cases of "secondary" insomnia may have a substantial primary insomnia component that would be responsive to treatment, and we have used the term partial SI to refer to a mix of primary insomnia and SI (Lichstein, 2000b). Second, a reciprocity may arise whereby the "secondary" insomnia partially sustains or exacerbates the primary illness. Thus, insomnia effects, such as fatigue and irritability, may contribute to the primary illness, as exemplified by depression. Successful treatment of the primary illness may be aided by the treatment of the SI. Third, the existing literature does contain a small amount of data suggesting that SI can be treated. These data encourage further efforts to address this problem.

Although behavioral treatments have been shown to be effective in treating insomnia (Lichstein & Riedel, 1994; Morin, Culbert, & Schwartz, 1994), nearly all studies of treatment efficacy have involved individuals with primary insomnia, excluding individuals whose insomnia may be due to medical-psychiatric disorders. To our knowledge, only five studies of psychological interventions have involved an attempt to treat SI, and four of these were case reports. The only randomized study (Cannici, Malcolm, & Peck, 1983) showed that relaxation improved latency to sleep in cancer patients. Of the remaining four, three were case studies of single participants (Morin, Kowatch, & O'Shanick, 1990; Stam & Bultz, 1986; Varni, 1980), and one involved a multiple baseline design (Morin, Kowatch, & Wade, 1989). Most of these studies targeted insomnia secondary to chronic pain, and no study to date has exclusively focused on the psychological treatment of insomnia secondary to psychiatric illness.

To our knowledge, there have been no studies of SI specifically among older adults, although this population is at particularly high risk for SI because of heightened rates of illness and increased polypharmacy exposure. Furthermore, the characteristic light sleep of older adults (Morgan, 1987) increases their sensitivity to sleep irritants. In the present clinical trial, we jointly presented two of the strongest treatments for primary insomnia, relaxation and stimulus control, to combat SI in older adults. Relaxation refers to a collection of methods whose common ground consists of focusing procedures that are arousal reducing (Lichstein, 1988). Stimulus control improves sleep by eliminating distractions and competing behaviors from the bedroom so that the bedroom becomes a sleep-conducive environment (Bootzin, Epstein, & Wood, 1991). These two insomnia treatments have been found to be effective in older populations (Lichstein, Riedel, & Means, 1999). According to a meta-analysis of insomnia treatments, stimulus control is particularly effective in regard to awakenings during the night, and cognitive relaxation (which is incorporated into the present method) is particularly effective in terms of difficulty falling asleep initially (Morin et al., 1994). As a pair, stimulus control and cognitive relaxation would appear to be an effective package addressing the variety of insomnia complaints. These two treatments were supplemented by sleep hygiene instructions. We combined treatments in the hope of maximizing efficacy, because we anticipated that sleep improvement would not be easily attained when directly treating insomnia symptoms secondary to another disorder.

Method

Participants

Forty-four participants 58 years of age or older were recruited through public service announcements and newspaper advertisements. To be eligible for the study, volunteers had to satisfy the following criteria: (a) be under the care of a health care provider for either a psychiatric SI (PSI) or medical SI (MSI) condition and (b) satisfy the diagnosis of SI. To qualify as having SI, participants had to first demonstrate the presence of insomnia. Participants were considered to have insomnia if they were concerned about their sleep, reported daytime impaired functioning, and reported disturbed sleep: (a) latency to sleep greater than 30 min, (b) awakenings during the night totaling 30 min or more, or (c) early morning awakenings. Disturbed sleep had to be present, on average, at least three times per week for at least 6 months. This definition of insomnia is consistent with standard diagnostic criteria (American Sleep Disorders Association, 1990). Participants were classified as having onset insomnia if difficulty falling asleep was their sole problem, maintenance insomnia if difficulty staying asleep was their sole problem, or terminal insomnia if early morning awakening was their sole problem, as determined by their baseline sleep diary data. If participants exhibited more than one type of sleep difficulty, they were classified as having mixed insomnia.

We applied strict criteria to determine whether the insomnia was secondary to another condition. For the insomnia to qualify as secondary, the history of the insomnia with respect to onset and variations in severity had to mirror the history of the primary disease. As a means of establishing this correspondence, the typical questions used in the diagnostic interview were "Did you have insomnia prior to the onset of your medical-psychiatric condition?" "Did your insomnia begin shortly after the onset of your medical-psychiatric condition?" "When your medical-psychiatric condition flares up, have you found that your insomnia gets worse?" and "When your medical-psychiatric condition goes through periods of improvement, have you found that your insomnia gets better?" The reliability of such information was, of course, dependent entirely on the accuracy of the participant's memory, and this was often difficult to determine. If the onset and course of the insomnia appeared to be independent of the medical-psychiatric condition, we concluded that this represented comorbidity rather than SI, and the participant was disqualified. Furthermore, there must be a plausible sleep-active mechanism linking the primary disease to the sleep disturbance, as typified by chronic pain or arousing cognitions associated with anxiety or depression. This definition of SI is also consistent with standard diagnostic criteria (American Sleep Disorders Association, 1990).

Participants taking medications for their primary condition were not excluded even if the medication had sleep-active properties. However, medication taken specifically for sleep was an exclusionary criterion, and participants were asked to refrain from taking any sleep medication during the course of the study. Decisions regarding the admissibility of medications were sometimes difficult. The most common challenging situation we encountered was with sedating antidepressants. If these medications were taken during the day, they were acceptable. If they were taken at night, we attempted to clarify with the participant and sometimes his or her physician the intent. If we could determine that the medication was primarily prescribed to aid sleep, it was not acceptable. Obviously, in a few cases, we had to make a fairly arbitrary decision. The presence of other sleep disorders (e.g., narcolepsy, periodic limb movements, sleep apnea, or irregular sleep schedules due to shift work), as ascertained by interview, was also an exclusionary factor.
This study required a substantial investment of time and effort by the participants and was particularly trying for our sample, because all who qualified had a chronic primary disease. As a means of compensating individuals for their time and effort, they were paid $200 on completion of their participation. Participants who were unable to complete the study as a result of illness or other circumstances beyond their control were given partial compensation.

**Measures**

**Sleep diaries.** Sleep diaries (this form is given in Lichstein, Riedel, & Means, 1999) were used to measure sleep. Participants completed diaries each morning for 2-week blocks during baseline, posttreatment, and follow-up. The diary yields a number of sleep measures: time spent napping during the day, sleep onset latency, number of awakenings during the night, wake time after sleep onset, total sleep time, and sleep efficiency percentage (SEP; the ratio of total time slept to total time in bed × 100). The diary also asks individuals to rate their overall quality of sleep on a 5-point scale (1 = very poor, 5 = excellent), termed sleep quality rating. Finally, the diary asks individuals to report any medication, alcohol, and so forth taken at bedtime.

**Insomnia Impact Scale (IIS).** The IIS contains 40 negative statements about the daytime impact of sleep (Hoelscher, Ware, & Bond, 1993). These statements sample five areas of impairment: physical, cognitive, emotional, social, and occupational. Respondents rate each item on a 5-point scale to register their degree of agreement. A maximum score of 200 indicates the greatest impairment. In the validating study (Hoelscher et al., 1993), the IIS discriminated these two groups as well, suggesting that it is sensitive to degree of insomnia.

**Geriatric Depression Scale (GDS).** The GDS (Yesavage et al., 1983) was developed and validated on geriatric samples. This instrument consists of 30 questions, each asking whether a symptom of depression is present. Items require a yes–no response, and scores vary between 0 and 30. The GDS shows test–retest reliability exceeding .8 and reliably distinguishes normal, mildly depressed, and severely depressed groups.

**State-Trait Anxiety Inventory—Form Y Trait Scale (STAI).** The STAI is a self-report questionnaire designed to measure anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The trait scale measures enduring anxiety states rather than transient anxiety. The STAI consists of 20 self-descriptive statements that are rated on a 4-point scale indicating how often the statement is true. Scores range from 20 to 80. The STAI shows test–retest reliability exceeding .7 and reliably distinguishes patient and normal groups.

**Adherence logs.** Participants in the treatment condition were given two logs designed to conveniently track relaxation and stimulus control home practice. The relaxation log provides tables to record amount of time spent practicing relaxation, prepractice and postpractice ratings of relaxation (1 = very aroused and upset, 10 = completely and deeply relaxed), and prepractice and postpractice pulse rates (this form is given in Lichstein, 2000a). Participants were trained to take their own pulse rates at their wrist or their neck. The stimulus control log listed the six parts to the stimulus control instructions and provided seven columns to record adherence for a week. The participant simply marked yes or no in each box to indicate whether the instruction was followed. Participants turned in completed logs (beginning with Session 2) and picked up blank ones at each treatment session. Participants were instructed to record pulse rate only at the end of relaxation practice session, because doing this at bedtime was arousing. Similarly, bedtime relaxation ratings could be recorded the following morning to avoid disrupting sleep onset.

**Procedure**

All participants were screened over the telephone to make a preliminary determination that they met inclusion and exclusion criteria. Participants who appeared to qualify were asked to come to the Department of Psychology at The University of Memphis for a follow-up screening interview. Participants who continued to satisfy admission criteria were given a detailed description of the study and were asked to sign a consent form. Satisfaction of the screening criteria was determined jointly by the therapist assigned to the case and the supervising clinical psychologist (Kenneth L. Lichstein).

Participants were given a questionnaire packet composed of a 2-week supply of sleep diaries and one each of the IIS, GDS, and STAI. Participants were instructed in their use and were told to begin completing the sleep diaries immediately and to complete the three other questionnaires at the end of the 2 weeks. Participants were also given a franked envelope to return the set of questionnaires.

The therapist assigned to the case and the supervising clinical psychologist jointly made the final decision on acceptance of the participant into the study after reviewing the baseline sleep diaries. These diaries had to show an insomnia sleep pattern according to our screening criteria regarding frequency and severity of latency to sleep, wake time during the night, or early morning awakening.

Participants accepted into the study were randomly assigned to either the treatment group or a delayed treatment control condition. The same questionnaire packet used at baseline was administered at posttreatment and the 3-month follow-up. Participants in the control group were asked to complete the packet at times matching the schedule of the treated group.

We had no contact with control group participants during the treatment phase. At posttreatment, control participants were mailed an assessment packet. At follow-up, both groups were sent a packet. Posttreatment and follow-up assessment packets were returned in a franked envelope we provided. Control participants were offered treatment after follow-up was completed.

**Treatment**

Treatment consisted of four weekly 1-hr individual treatment sessions conducted at the Psychological Services Center in the Department of Psychology. This is a well-appointed treatment facility that offers services to the general community and serves as a training and research laboratory. We used a treatment package composed of sleep hygiene instructions, stimulus control, and relaxation.

The therapists for this study were four graduate students in clinical psychology, each having 2–3 years of clinical experience. All of these students had prior experience in sleep research and treatment. Training specific to this study included studying a detailed treatment manual, mock therapy sessions, observing experienced therapists, and discussion. Weekly therapy supervision was provided by Kenneth L. Lichstein, a clinical psychologist experienced in sleep treatment.

**Sleep hygiene.** This refers to a collection of guidelines on how best to arrange one’s daytime activities to minimize impediments to nighttime sleep and maximize sleep facilitation (Riedel, 2000). For the present study, the sleep hygiene guidelines were as follows: avoid caffeine after noon, avoid exercise within 2 hr of bedtime, avoid nicotine within 2 hr of bedtime, avoid alcohol within 2 hr of bedtime, avoid heavy meals within 2 hr of bedtime. Participants were given a list of these instructions to promote adherence.

**Stimulus control.** This procedure consists of giving the patient the following instructions (Bootzin et al., 1991).

1. Go to sleep only when sleepy.
2. Do not use your bed for anything except sleep (and sex); do not read, watch television, eat, or worry in bed.
3. If you do not fall asleep within 15–20 minutes, get up and go into
another room. Stay up as long as you wish and return to the bedroom only when you feel sleepy.
4. If you still can't fall asleep, repeat Step 3. Do this as often as necessary throughout the night.
5. Set your alarm and get up at the same time every morning.
6. Do not nap during the day.

Stimulus control is designed to strengthen the association between the bedroom and sleep so that the individual is more likely to fall asleep when in that environment. Each of the steps contributes to this goal, and the therapist carefully explained the role of each component so that the participant understood the rationale of the stimulus control parts and would be more likely to comply (Sloan et al., 1993).

During treatment sessions, the therapist encouraged the participant to follow the instructions and helped troubleshoot any problems that arose. Participants were given a list of stimulus control instructions to foster adherence.

Relaxation. We used a hybrid relaxation procedure consisting of (a) emphasizing a relaxed attitude; (b) taking five slow, deep breaths including a softly spoken “relax” self-instruction with each exhale; (c) slowly reviewing the body in sequential parts while focusing on relaxed sensations, sometimes termed passive relaxation; and (d) repeating, slowly and silently, the autogenic phrase “I am at peace, my arms and legs are heavy and warm.” The procedure requires 10 min, has been used successfully with older adults with insomnia (Lichstein & Johnson, 1993), and is given verbatim in Lichstein (2000a).

Progressive relaxation, the most common method of relaxation used with insomnia, may be less suitable for older adults, particularly those with medical illness, because of its procedural complexity and physical exertion. The present method avoided these potential pitfalls and also had the advantage of being composed of discreet components. Thus, the participant was presented with a menu of techniques and can emphasize those parts found most appealing.

Participants were introduced to relaxation during the first session. Sessions 2, 3, and 4 were used to refine the participant’s technique, tailor the technique to the individual participant, and troubleshoot any problems the participant might have. Participants were strongly encouraged to practice relaxation at home twice a day: once at any time and once at bedtime. They were given a handout describing the technique to help facilitate home practice.

Results

Description of Participants

Forty-four individuals completed all phases of data collection, 23 in the treatment condition and 21 in the control condition. Among treated participants, 11 had PSI and 12 had MSI. Among control group participants, 9 had PSI and 12 had MSI. For the overall sample, PSI participants were divided between 8 with an anxiety disorder (4 each in the treatment and control groups) and 12 with some form of depression (7 treatment and 5 control). MSI participants were divided among 10 whose insomnia was primarily due to chronic pain (6 treatment and 4 control; arthritis, physical trauma, or neuropathy), 7 with prostate disease (3 treatment and 4 control; sleep disrupted by frequent urination), 5 with neurologic disorders (1 treatment and 4 control; stroke, Parkinson’s, or epilepsy), and 2 with chronic respiratory disease (both in the treatment group; asthma or chronic obstructive pulmonary disease).

Five individuals dropped out of the study before completing their participation. Only one of these individuals was from the treatment group, and this person died. Four participants in the control group lost interest in waiting and declined to complete assessment data at either posttreatment or follow-up.

During screening interviews, participants provided data on a number of personal characteristics (gender, age, and body mass index [BMI] and symptoms (insomnia type, number of nights per week insomnia is typically experienced, duration of insomnia, prior sleep treatment, sleep medications taken at time of initial interview, and total number of medications taken). The last two variables require additional explanation. A number of participants who otherwise qualified for the study were taking sleep medication. They were told to consult with their physician about the advisability of discontinuing the hypnotic. If they were hypnotic free for 1 month, we reconsidered them for this study, but they had to agree not to resume sleep medication consumption during the course of the study. The last variable was simply a count of the number of medications the participant was taking at the time of entering the study, which could serve as a crude index of general health.

We performed a two-way analysis of variance (ANOVA) comparing treatment groups (treatment or control) and type of SI (PSI or MSI). The variables were age, BMI, number of insomnia nights per week, insomnia duration, and total number of medications currently being taken. There were no significant main or interaction effects. We performed chi-square tests of independence on the four nominal variables (gender, insomnia type, prior sleep treatment, and sleep medications taken at time of initial interview). These tests first compared treatment groups and then compared type of SI. All eight chi-square tests were nonsignificant.

A breakdown by group on these variables is presented in Table 1. Ranges for the parametric variables were as follows: age, 58 to 85 years; BMI, 19.8 to 40.4 kg/m²; number of insomnia nights, 2 to 7; insomnia duration, 0.5 to 58 years; and number of medications taken, 0 to 9.

Table 1

<table>
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<tr>
<th>Characteristic</th>
<th>Treatment group</th>
<th>Control group</th>
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<tr>
<td>Age (years)</td>
<td>67.1 6.1</td>
<td>70.1 6.8</td>
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<tr>
<td>Body mass index (kg/m²)</td>
<td>27.9 5.6</td>
<td>25.4 3.7</td>
</tr>
<tr>
<td>Number of insomnia nights per week</td>
<td>5.3 1.9</td>
<td>6.3 1.4</td>
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<tr>
<td>Duration of insomnia (years)</td>
<td>10.3 12.5</td>
<td>9.4 11.7</td>
</tr>
<tr>
<td>Total number of medications taken</td>
<td>3.2 2.0</td>
<td>3.5 2.5</td>
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<tr>
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<tr>
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<tr>
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<tr>
<td>No</td>
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<td>16</td>
</tr>
<tr>
<td>Sleep medications taken at time of initial interview*</td>
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<td>6</td>
</tr>
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</table>

* Counts do not sum to 44 participants because data were missing on a few individuals.
Treatment Implementation

As with any disorder, adherence to treatment for insomnia is a complex, crucial issue. Traditionally, adherence concerns have been limited to the degree to which the participant complies with therapeutic instructions, but this view is prone to overestimate adherence because it ignores other salient factors that may also affect adherence. We have proposed a treatment implementation model as one way of understanding adherence (Lichstein, Riedel, & Grieve, 1994). Independent treatment components termed delivery, receipt, and enactment must be adequately represented to conclude that proper treatment occurred. The delivery component refers to the accuracy of treatment presentation (also called treatment integrity), receipt refers to the accuracy of the participant’s comprehension of or mastery of treatment, and enactment refers to the extent of out-of-session application initiated by the participant (this component is equivalent to the usual meaning of adherence). This section presents the results of our efforts to evaluate the degree of treatment implementation.

Delivery. As a means of increasing the likelihood that treatments were delivered as intended, therapists were carefully trained (see the Treatment section) and had to complete a checklist for each session. This checklist served as a reminder of all parts of the session that had to be covered and were tailored for each session.

We performed only qualitative assessment of the delivery component in weekly clinical supervision. In the process of reviewing clinical procedures, the supervisor also checked to determine that all parts of treatment were presented as intended and extraneous treatments were not introduced. This procedure failed to reveal a single instance of serious departure from the standardized protocol. Examples of serious departure are omitting an important part of a treatment and introducing a treatment that was not part of the protocol. We did observe occasional minor departures such as rushing the rationale for a part of the stimulus control protocol.

Receipt. As a means of determining that relaxation inductions conducted in the four treatment sessions were successful (i.e., treatment was properly received), participants provided relaxation ratings and pulse rates before and after each induction (see the Adherence logs section for a description of the rating scale). Participants reported an average preinduction rating of 4.9 (SD = 1.4) and an average postinduction rating of 7.2 (SD = 1.0), t(20) = 7.91, p < .01. Preinduction pulse rates averaged 74.1 (SD = 13.7), and postinduction pulse rates averaged 68.8 (SD = 11.0), t(19) = 4.50, p < .01. Changes in ratings and pulse rates were consistent with successful relaxation inductions.

Satisfactory mastery of the stimulus control procedures was assessed via a homemade quiz consisting of 10 true–false questions inquiring about the procedures of stimulus control. This quiz was administered at the beginning of the second treatment session. Number correct averaged 8.8 (SD = 1.5), scores ranged from 4 to 10, and the mode was 10. Subsequent to its administration, the test was reviewed with each participant to clarify any questions. The therapist provided careful explanations in response to every incorrect answer until the participant could verbally demonstrate knowledge of that point.

Enactment. Adherence logs were used to self-monitor home practice of relaxation and stimulus control. Participants practiced relaxation an average of 12.6 times per week (SD = 1.8), and the duration of a practice averaged 10.6 min (SD = 2.0). Relaxation ratings improved from preinduction (M = 5.0, SD = 1.4) to postinduction (M = 7.0, SD = 1.2), as did preinduction (M = 72.8, SD = 13.2) to postinduction (M = 67.5, SD = 12.0) pulse rates. For stimulus control, participants averaged 85.6% adherence (SD = 9.2%).

Sleep

We performed a series of 2 (types: PSI vs. MSI) X 2 (groups: treatment vs. control) X 3 (times: baseline vs. posttreatment vs. follow-up; repeated measures) ANOVAs on the sleep measures. To avoid inflated Type I error rates occurring in repeated measures ANOVA when the assumption of sphericity is violated, we used a multivariate approach to evaluate the repeated measures variable and interactions to which it contributed. Similarly, in analyses of simple effects of time, we used Bonferroni adjusted paired t tests.

Group means on the sleep variables are presented in Table 2. Because type of SI did not prove to be a discriminating factor, Table 2 identifies only group membership and time. Reference to this table will help clarify the statistical results provided subsequently.

There were no significant main or interaction effects associated with number of awakenings. Three variables yielded only significant time main effects: sleep onset latency, total sleep time, and naps. For two of these variables, sleep onset latency and total sleep time, the pattern was the same. Sleep significantly improved from baseline to posttreatment and from baseline to follow-up, but there was no significant change from posttreatment to follow-up. For naps, significant change occurred only from baseline to posttreatment, although the comparison of baseline and follow-up approached significance. Inspection of the means of these three variables (see Table 2) reveals that greater improvement always occurred in the treated group, but large error variance prevented significant interaction effects.

Significant Group × Time interactions were found for three variables: wake time after sleep onset, Wilk’s Λ = 0.85, F(2, 39) = 3.46, p < .05; SEP, Wilk’s Λ = 0.84, F(2, 39) = 3.63, p < .05; and sleep quality rating, Wilk’s Λ = 0.83, F(2, 39) = 3.90, p < .05. Simple effects tests for SEP and sleep quality rating yielded similar results. There was no significant difference between groups at baseline, but the treated group had a significantly higher SEP and a significantly higher sleep quality rating than the untreated group at follow-up. For the sleep quality rating, there was also a significant group effect at posttreatment. For time, significant changes occurred only among the treated group. A comparison of baseline with posttreatment and follow-up showed that significant improvement occurred for both measures. There was no significant change from posttreatment to follow-up. Simple effects results for wake time after sleep onset differed. There were no significant group differences at any of the time points. As with SEP and sleep quality rating, improvement occurred over time only for the treated group and in the same pattern. A comparison of baseline with posttreatment and follow-up showed that significant improvement occurred for wake time after sleep onset. There was no significant change from posttreatment to follow-up.

There was one additional interesting outcome that should be noted. Significant change never occurred from posttreatment to follow-up on any of the seven sleep measures, indicating an absence of either improvement or deterioration during this period.

This section presents the results of our efforts to evaluate the extent of out-of-session application initiated by the participant (this component is equivalent to the usual meaning of adherence). This section presents the results of our efforts to evaluate the degree of treatment implementation.

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We performed a series of 2 (types: PSI vs. MSI) X 2 (groups: treatment vs. control) X 3 (times: baseline vs. posttreatment vs. follow-up; repeated measures) ANOVAs on the sleep measures. To avoid inflated Type I error rates occurring in repeated measures ANOVA when the assumption of sphericity is violated, we used a multivariate approach to evaluate the repeated measures variable and interactions to which it contributed. Similarly, in analyses of simple effects of time, we used Bonferroni adjusted paired t tests.

Group means on the sleep variables are presented in Table 2. Because type of SI did not prove to be a discriminating factor, Table 2 identifies only group membership and time. Reference to this table will help clarify the statistical results provided subsequently.

There were no significant main or interaction effects associated with number of awakenings. Three variables yielded only significant time main effects: sleep onset latency, total sleep time, and naps. For two of these variables, sleep onset latency and total sleep time, the pattern was the same. Sleep significantly improved from baseline to posttreatment and from baseline to follow-up, but there was no significant change from posttreatment to follow-up. For naps, significant change occurred only from baseline to posttreatment, although the comparison of baseline and follow-up approached significance. Inspection of the means of these three variables (see Table 2) reveals that greater improvement always occurred in the treated group, but large error variance prevented significant interaction effects.

Significant Group × Time interactions were found for three variables: wake time after sleep onset, Wilk’s Λ = 0.85, F(2, 39) = 3.46, p < .05; SEP, Wilk’s Λ = 0.84, F(2, 39) = 3.63, p < .05; and sleep quality rating, Wilk’s Λ = 0.83, F(2, 39) = 3.90, p < .05. Simple effects tests for SEP and sleep quality rating yielded similar results. There was no significant difference between groups at baseline, but the treated group had a significantly higher SEP and a significantly higher sleep quality rating than the untreated group at follow-up. For the sleep quality rating, there was also a significant group effect at posttreatment. For time, significant changes occurred only among the treated group. A comparison of baseline with posttreatment and follow-up showed that significant improvement occurred for both measures. There was no significant change from posttreatment to follow-up. Simple effects results for wake time after sleep onset differed. There were no significant group differences at any of the time points. As with SEP and sleep quality rating, improvement occurred over time only for the treated group and in the same pattern. A comparison of baseline with posttreatment and follow-up showed that significant improvement occurred for wake time after sleep onset. There was no significant change from posttreatment to follow-up.

There was one additional interesting outcome that should be noted. Significant change never occurred from posttreatment to follow-up on any of the seven sleep measures, indicating an absence of either improvement or deterioration during this period.
We compared our data with previously reported rates before and after practice) and one measure of stimulus control (proportion of stimulus control instructions followed). These measures did not occur over time in either group, particularly for the GDS and STAI. Analyses, a main effect for naps, F(1, 40) = 5.61, p < .05. MSI participants showed significantly more clinical improvement than untreated participants, \( \chi^2(1, N = 44) = 7.24, p < .01 \).

### Daytime Functioning

The same statistical model used with the sleep measures was applied to the three measures of daytime functioning (GDS, STAI, and IIS). As can be seen in Table 2, large changes in these measures did not occur over time in either group, particularly for the GDS and STAI. All main effects tests on time and all interactions involving time were nonsignificant.

Some of the main effects tests for type of SI were significant, and most of these results reflect participant selection biases. Not surprisingly, PSI participants reported more depression on the GDS (\( M = 15.1, SD = 8.0 \)) than did MSI participants (\( M = 9.1, SD = 7.3 \)). Similarly, PSI participants reported more anxiety on the STAI (\( M = 48.3, SD = 11.5 \)) than did MSI participants (\( M = 36.5, SD = 11.6 \)). On the IIS, PSI participants reported greater impact of their insomnia (\( M = 120.8, SD = 22.4 \)) than did MSI participants (\( M = 105.8, SD = 19.4 \)). In a review of the means for the IIS, the treatment group did show improvement (vs. no change in the control group), but this interaction failed to achieve significance.

### Relating Treatment Implementation to Outcome

We considered four indexes of adherence: three measures of home relaxation practice (minutes of relaxation practice, change in relaxation ratings before and after practice, and change in pulse rate before and after practice) and one measure of stimulus control (proportion of stimulus control instructions followed). These mea-

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**Table 2**

<p>| Sleep and Daytime Functioning Variables, by Group, Over Treatment Phases |
|-------------------|-----------------|-----------------|-----------------|
|                    | Baseline        | Posttreatment   | Follow-up       |</p>
<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep latency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>48.0</td>
<td>42.4</td>
<td>30.8</td>
<td>23.6</td>
<td>26.9</td>
<td>19.3</td>
</tr>
<tr>
<td>Control</td>
<td>54.9</td>
<td>41.0</td>
<td>41.9</td>
<td>25.1</td>
<td>50.2</td>
<td>36.8</td>
</tr>
<tr>
<td>Total sleep time</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>328.8</td>
<td>86.4</td>
<td>374.3</td>
<td>90.9</td>
<td>373.3</td>
<td>66.5</td>
</tr>
<tr>
<td>Control</td>
<td>343.1</td>
<td>99.4</td>
<td>374.4</td>
<td>114.6</td>
<td>359.9</td>
<td>103.4</td>
</tr>
<tr>
<td>Number of awakenings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>2.7</td>
<td>1.5</td>
<td>2.4</td>
<td>1.1</td>
<td>2.1</td>
<td>1.3</td>
</tr>
<tr>
<td>Control</td>
<td>2.0</td>
<td>1.1</td>
<td>1.9</td>
<td>1.1</td>
<td>2.1</td>
<td>1.3</td>
</tr>
<tr>
<td>Wake time after sleep onset</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>87.3</td>
<td>61.3</td>
<td>60.9</td>
<td>64.2</td>
<td>56.4</td>
<td>40.8</td>
</tr>
<tr>
<td>Control</td>
<td>68.3</td>
<td>57.1</td>
<td>69.2</td>
<td>53.2</td>
<td>60.8</td>
<td>54.5</td>
</tr>
<tr>
<td>Sleep efficiency percentage</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>66.7</td>
<td>17.1</td>
<td>77.8</td>
<td>15.0</td>
<td>77.7</td>
<td>10.8</td>
</tr>
<tr>
<td>Control</td>
<td>65.5</td>
<td>14.1</td>
<td>69.1</td>
<td>14.8</td>
<td>68.1</td>
<td>14.4</td>
</tr>
<tr>
<td>Naps (minutes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>15.3</td>
<td>20.0</td>
<td>6.0</td>
<td>12.3</td>
<td>5.9</td>
<td>7.6</td>
</tr>
<tr>
<td>Control</td>
<td>13.2</td>
<td>14.6</td>
<td>11.6</td>
<td>15.6</td>
<td>12.7</td>
<td>15.2</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>2.7</td>
<td>0.7</td>
<td>3.2e</td>
<td>0.6</td>
<td>3.2e</td>
<td>0.6</td>
</tr>
<tr>
<td>Control</td>
<td>2.6</td>
<td>0.6</td>
<td>2.7</td>
<td>0.6</td>
<td>2.6</td>
<td>0.7</td>
</tr>
</tbody>
</table>

* There was a significant main effect for time showing significant change from baseline to this treatment period (p < .05). b There was a significant change from baseline to this treatment period within this group (p < .05).

Type of SI contributed only one significant result in the set of analyses, a main effect for naps, \( F(1, 40) = 5.61, p < .05 \). MSI participants napped an average of 14.4 min (SD = 15.0), as compared with 5.8 min (SD = 6.1) for PSI participants.

### Clinical Significance

Statistical significance informs one little on the clinical meaningfulness of change. One can establish standards of change that represent clinical meaningfulness, and these standards can be on a gradient of magnitude of improvement (Kazdin, 1999).

We used SEP to measure clinical significance for two reasons: It responded well to therapy, and it is recognized as a good general index of sleep. We compared our data with previously reported norms for self-reported SEP among normal-sleeping older adult individuals (i.e., \( M = 86.1, SD = 9.7 \); Lichstein, 1997). We constructed three criteria to judge clinical significance.

First, achieving an SEP equal to or greater than the mean of the normative sample signified clinically significant improvement. Second, achieving an SEP within 1 SD of the mean of the normative sample signified moderately clinically significant improvement. For each of these two criteria, we wanted to eliminate individuals who had high SEP values at baseline, in which case satisfying these criteria might not signify substantial change. Therefore, if a participant was to qualify under either of these criteria, there had to be an improvement of at least 0.5 SD. Third, some individuals had such a low SEP at baseline that they had little hope for improving into the normal range but still may have achieved substantial improvement. To recognize these individuals, we added the criterion that an improvement of 2 SD would be judged as substantial improvement.

Comparisons of baseline and follow-up SEP change for the treated group indicated that 4 participants registered clinically significant improvement, 6 participants registered moderately clinically significant improvement, and 3 participants registered substantial improvement. Of 23 treated participants, 13 (57%) showed some measure of clinical improvement.

In the control group, there were 2 participants who registered clinically significant improvement, 2 participants who registered moderately clinically significant improvement, and no participants who registered substantial improvement. Of 21 control participants, 4 (19%) showed some measure of clinical improvement. In a comparison of the total count in the two groups, treated participants showed significantly more clinical improvement than untreated participants, \( \chi^2(1, N = 44) = 7.24, p < .01 \).

The same statistical model used with the sleep measures was applied to the three measures of daytime functioning (GDS, STAI, and IIS). As can be seen in Table 2, large changes in these measures did not occur over time in either group, particularly for the GDS and STAI. All main effects tests on time and all interactions involving time were nonsignificant.

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ures were converted to z scores and averaged to yield a single z score representing an adherence index.

When a treatment package is used, a summary measure of adherence is likely to be more informative than separate measures of individual components. We reasoned that if adherence was high in some areas and low in others, treatment outcome was not likely to be related to any of the measures. By joining the several measures into one index, we obtained an overall measure of adherence that should better relate to treatment impact.

The adherence index was correlated with change in the three measures of sleep that showed the strongest treatment response: wake time after sleep onset, SEP, and sleep quality rating. Only one significant result was found. The adherence index was related to improvement in sleep quality rating from baseline to follow-up (r = .44, p < .05). Further analyses showed that this association was stronger than that of any of the adherence measures taken separately with outcome.

Similar analyses were conducted to assess the relationship between the in-session experience and treatment outcome. Measures of treatment receipt (in-session change in relaxation rating and pulse rate, along with sleep quiz score) were correlated with the same outcome measures, as a result index derived from the mean of z scores. There were no significant correlations.

Discussion

SI showed a strong response to psychological intervention. Sleep improvement was substantial and sustained over the follow-up period. We found no evidence to suggest that one type of SI showed a greater treatment response than another.

Of the few extant treatment studies of SI, only one was a randomized trial, and it focused on a narrow subset of SI patients (cancer patients; Cannici et al., 1983). Ours is the first randomized study involving a mixed sample of people with SI and the first to focus on older adults. Because SI is likely the predominant type of insomnia in older adults (Lichstein, 2000b), it is noteworthy that we have demonstrated for the first time that this needy subset is treatment responsive.

However, we must also acknowledge methodological weaknesses in the present research. The use of a no-treatment control group is less satisfactory than a placebo-control group, and therefore the current design cannot rule out the role of placebo effects in the treatment group. In defense of the current design, the insomnia treatment literature has accumulated a substantial amount of information on the role of placebo in psychological treatments, and this archival data can inform researchers as to what to expect of placebo factors. On the average, effect sizes associated with insomnia treatments more than double those of placebo groups, suggesting that placebo factors do not account for a substantial portion of outcome (Murtagh & Greenwood, 1995).

Measuring sleep by self-report does not attain the level of specificity and validity yielded by objective evaluations of sleep, such as polysomnography (PSG). Alternatively, the issue of self-report versus PSG sleep data is not as simple as it might appear, and self-report sleep data should be construed not as a weak substitute for PSG but, rather, as a unique view of sleep. PSG is not without its own shortcomings, including frequent correspondence between Sleep Stages 1 and 2 and experiential awareness (Antrobus & Saul, 1980; Bonnet & Moore, 1982), high night-to-night sleep variability that may cause 2 or even 3 nights of PSG data to be an unrepresentative sample of sleep (Edinger, Marsh, McCall, Erwin, & Lininger, 1991), and observation of sleep in an artificial environment. Such flaws may introduce error into "objective" PSG findings. Self-reported sleep represents a view of sleep not obtainable from PSG, the subjective experience of the individual, and this is an important perspective. Finally, the sleep patterns derived from self-reports do inform researchers of objective sleep patterns in that the two are not wholly unrelated. Numerous studies of people with insomnia, including older adults with insomnia, have shown that these individuals often report more disturbed sleep than is found in PSG records, but the two are moderately to highly correlated (Carskadon et al., 1976; Jacobs, Benson, & Friedman, 1993; Morin, Kowatch, Barry, & Walton, 1993). Thus, there often is a "constant" offset, but change over time usually reflects objective change.

The present treatment outcome was strong, and it was accomplished with a minimum number of treatment sessions (four). This predicts well for the future of treatment in this domain, because we probably did not deliver a full therapeutic dose to some participants. Future research with this population should double the number of treatment sessions (Lichstein & Morin, 2000), and perhaps one could look forward to even stronger outcomes.

As sleep changed, there was no evidence that improved daytime functioning followed. The variety of types of MSI made it difficult to monitor daytime changes in these participants, but we probably could have done a better job of that. About half of our MSI participants had chronic pain, and we should have collected pain ratings. It is unlikely that the other types of MSI in our sample—prostate, neurological, and pulmonary disease—would have involved daytime benefits from sleep improvement. However, we did measure anxiety and depression, and we could not discern daytime improvement in our participants with PSI.

Two recent studies from our laboratory treating primary insomnia in young adults (Means, Lichstein, Epperson, & Johnson, in press) and older adults (Lichstein, Riedel, Wilson, Lester, & Aguillard, 1999) also showed that daytime functioning change did not accompany sleep improvement. The latter study included a 1-year follow-up, undermining one plausible explanation that daytime changes occur at a slower pace than nighttime changes. One hypothesis worth considering is that nighttime improvement primes daytime functioning for change, but direct daytime intervention is needed to instigate change. We think the correspondence between sleep and daytime functioning is a clinically significant subject, and future research should continue to investigate the nature of this relationship and strategies to promote therapeutic generalization from night to day.

We have introduced the adherence index, a novel method of representing home treatment practice when treatment is multicomponent rather than a unitary intervention (see the Relating Treatment Implementation to Outcome section). Our data suggest that a summary measure of adherence relates more strongly to outcome than any of the separate adherence measures of the treatment package components. Nevertheless, adherence analyses did not reveal a strong connection between treatment and outcome despite strong sleep effects associated with the treated group. Three explanations can be offered for this. First, the treated group included 23 participants, which limited the power of the statistical...
tests. Many of the correlations were in the .3 range but failed to reach significance because of the small sample size.

Second, our ability to identify and measure the key therapeutic ingredients in the treatments was limited. Measures such as number of minutes of relaxation practice and number of stimulus control instructions followed may fail to capture the essence of the therapeutic experience for many participants. They may find value in other treatment nuances, be highly compliant with these treatments, and attend less diligently to the obvious indexes identified by researchers, thus weakening the apparent relationship between practice and outcome.

Third, our measures of both adherence and outcome were self-report measures. Each measure introduces psychometric noise, and when related to each other, error variance thrives and statistical power suffers. For example, we have shown that objective measures of relaxation adherence are significantly related to treatment outcome when self-report measures are not (Hoelscher, Lichstein, & Rosenthal, 1984).

How shall one understand the responsibility of individuals with SI to psychological treatment? By definition, SI cannot as long as its primary cause persists. According to this view, as psychological treatment abates SI, the primary cause replenishes it, and there is little net change in the sleep disturbance. This reasoning assumes that satisfactory diagnosis of SI has occurred, and the relationship between the insomnia and its cause is constant. These assumptions are probably unfounded much of the time.

Three types of SI have been identified (Lichstein, 2000b). Absolute SI refers to the conventional definition of SI. Partial SI asserts that the primary disorder affects sleep but does not control 100% of the variability of the insomnia. The insomnia may have predated the primary medical-psychiatric condition that serves to worsen the insomnia, or, once the insomnia is instigated by the medical-psychiatric condition, the sleep disturbance projects its own course. Specious SI specifies that there is the appearance of a causal link between a primary disorder and insomnia, but in fact no such relationship exists. When insomnia occurs in the absence of a comorbid disorder, one can be reasonably confident that the insomnia is primary rather than secondary. However, when a comorbid disorder is present, the determination of its relationship with observed insomnia is usually equivocal. Often (perhaps most of the time) when SI is diagnosed, one is in fact treating primary insomnia, at least in part.

The only study to our knowledge that measured the reliability of the SI diagnosis highlighted the difficulty inherent in this process (Buysse et al., 1994). Sleep specialists and general clinicians rendered independent diagnoses on a series of individuals reporting insomnia. Agreement between them in conferring the diagnosis of insomnia secondary to a mental disorder was at the lower boundary of the moderately good range (median κ = .42). This modest reliability reflects the ambiguity inherent in asserting the presence of SI.

There are mainly three reasons why rendering an accurate diagnosis of SI is difficult. First, the diagnosis derives primarily from the patient’s historical accounting of the course of the primary condition and of the insomnia, and this information may be faulty. Second, even when high-quality data are obtained, the diagnosis of SI usually represents an educated guess. The presence of a correlated history is not reliable evidence of a causal relationship. Third, the relationship between the insomnia and the primary disorder may transform over time.

When newly dawned insomnia emerges in the immediate aftermath of acute illness, the diagnosis of (absolute) SI is valid, or at least it appears so. However, more typically, by the time the patient seeks treatment for supposed SI, the insomnia has attained chronic status, and even an experienced diagnostician would have difficulty discriminating among absolute, partial, and specious SI.

It is not uncommon for insomnia to be instigated by another disorder, but over time, the insomnia may become self-sustaining. Spielman and Glovinsky (1991) have discussed “perpetuating” factors not present at the time insomnia originates, such as excessive time in bed and anxiety associated with sleeplessness and daytime deficits, that emerge over time, exacerbate and sustain the insomnia, and promote the independence of the insomnia from its original cause. Furthermore, the insomnia may reciprocate and aggravate the primary disorder. For example, several of our pain patients reported that their daytime pain experience improved as insomnia lessened. Similarly, others have reported both positive and negative change in “secondary” insomnia prompting like change in the “primary” disorder (Morin et al., 1989, 1990; Paiva, Batista, Martins, & Martins, 1995).

In the present study, we attempted to be thorough in our efforts to diagnose SI. We do not claim that we have achieved greater success than others in rendering valid diagnoses, and we cannot rule out that there was a substantial presence of either partial or specious SI (i.e., comorbidity mistaken for SI). However, we view this conclusion not as an indictment of our methods but, rather, as a strong justification for aggressively treating “secondary” insomnia.

References


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