Nonpharmacological Interventions for Insomnia: A Meta-Analysis of Treatment Efficacy

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Objective: *Because of the role of psychological factors in insomnia, the shortcomings of hypnotic medications, and patients' greater acceptance of nonpharmacological treatments for insomnia, the authors conducted a meta-analysis to examine the efficacy and durability of psychological treatments for the clinical management of chronic insomnia.* *Method:* *A total of 59 treatment outcome studies, involving 2,102 patients, were selected for review on the basis of the following criteria: 1) the primary target problem was sleep-onset, maintenance, or mixed insomnia, 2) the treatment was nonpharmacological, 3) the study used a group design, and 4) the outcome measures included sleep-onset latency, time awake after sleep onset, number of nighttime awakenings, or total sleep time.* *Results:* *Psychological interventions, averaging 5.0 hours of therapy time, produced reliable changes in two of the four sleep measures examined. The average effect sizes (i.e., z scores) were 0.88 for sleep latency and 0.65 for time awake after sleep onset. These results indicate that patients with insomnia were better off after treatment than 81% and 74% of untreated control subjects in terms of sleep induction and sleep maintenance, respectively. Stimulus control and sleep restriction were the most effective single therapy procedures, whereas sleep hygiene education was not effective when used alone. Clinical improvements seen at treatment completion were well maintained at follow-ups averaging 6 months in duration.* *Conclusions:* *The findings indicate that nonpharmacological interventions produce reliable and durable changes in the sleep patterns of patients with chronic insomnia.*

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Insomnia is among the most frequent health complaints brought to the attention of health care practitioners. Epidemiological surveys suggest that 10%–15% of adults complain of chronic insomnia (1, 2), and the prevalence estimates are higher among women, older adults, and patients with medical (3) or psychiatric disorders. Chronic insomnia is not a benign problem as it can adversely affect a person's life by causing substantial psychosocial, occupational, health, and economic repercussions (4). For example, individuals with chronic sleep disturbances experience more psychological distress, report greater impairments of daytime functioning, take more sick leave, are more preoccupied with somatic problems, and utilize health care resources more often than good sleepers (1, 2, 5, 6).

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Pharmacotherapy is the most frequently used method for treating insomnia. The National Institute of Mental Health survey of psychotherapeutic drug use indicated that 7.1% of adults have used either prescribed or over-the-counter sleeping aids in the course of a year and 11% of the users of hypnotics have used their medication regularly for more than a year (1). Benzodiazepine hypnotics, the most commonly prescribed sleeping aids, are efficacious on a short-term basis in reducing sleep latency, decreasing the number and duration of nocturnal awakenings, and increasing total sleep time and sleep efficiency (7, 8). The short-term use of hypnotic medications may be clinically indicated for selected subtypes of situational insomnia caused by acute stress, jet lag, or the like. There are few data, however, on their long-term efficacy, and their usefulness in the management of chronic insomnia is unclear (9). Furthermore, several problems are likely to arise either during the course of treatment or after its discontinuation: alteration of sleep stages, daytime residual effects, tolerance, dependence, and rebound insomnia (7, 8, 10). Because of reduced metabolic functioning with aging, their clinical use in geriatric patients warrants special cautions (11).

Recognition of the mediating role of psychological fac-

tors in insomnia, combined with the shortcomings associated with long-term use of hypnotics, have prompted the development of alternative nonpharmacological interventions for the management of chronic insomnia (4, 12–14). These treatment modalities are typically aimed at modifying maladaptive sleep habits, reducing autonomic and cognitive arousal, altering dysfunctional beliefs and attitudes about sleep, and educating patients about healthier sleep hygiene practices. As such, these cognitive, behavioral, and educational interventions are fairly practical, time limited, and sleep focused. Controlled evaluations of these treatment modalities have yielded promising results with a variety of clinical populations (15, 16). Nevertheless, nonpharmacological interventions are still underutilized by health care practitioners even though they are more acceptable to prospective insomnia patients than is pharmacotherapy (17).

Several review papers have contained descriptions of nonpharmacological clinical procedures, explanations of the rationale for using them, and discussions of their effectiveness in the management of disorders of initiating and maintaining sleep (8, 15, 18, 19). In the present paper we report on a meta-analysis of treatment outcome studies that quantifies the magnitude and durability of sleep pattern changes produced by more than a dozen psychological interventions evaluated with chronic insomnia sufferers.

METHOD

Selection of Studies

A total of 59 outcome studies conducted over 20 years (1974–1993) were selected from a pool of over 100 reports. (A list of those references is available on request from C.M.M.) The studies were identified through a computer search, bibliographies in previous reviews, and references cited in the reports themselves. The criteria for inclusion of a study in the meta-analysis were as follows: 1) the primary target problem was sleep-onset, maintenance, or mixed insomnia, 2) the treatment was nonpharmacological, 3) the study used a group design, and 4) the outcome measures consisted of one or more of the following: sleep-onset latency, time awake after sleep onset, number of awakenings, and total sleep time. Case reports or studies based on a single-subject design and studies evaluating pharmacological treatments were excluded.

Classification and Coding Systems

The studies were coded for several variables: sleep/wake measures, treatment modality, patients' characteristics, design quality, and several ancillary measures.

Each study was coded for a maximum of four dependent sleep/wake measures: sleep-onset latency, time awake after sleep onset, number of awakenings, and total sleep time. Means and standard deviations at baseline, posttreatment, and follow-up were compiled for each condition separately. When more than one follow-up was conducted, the data from the latest one were retained. Data for each of the sleep/wake measures were based on daily sleep diaries kept by the subjects, typically for 1 or 2 weeks before treatment and for a comparative duration after treatment and during follow-up. Polysomnographic data were available for nine of the 59 studies reviewed; these data are not reported here. Although sleep diary data do not reflect absolute values, as does polysomnography, they still yield a reliable and valid index of insomnia (20). Also, because the subjective complaint of poor sleep is an essential feature of insomnia, sleep perception is an important source of data in evaluating treatment outcome.

Each condition in a given study was classified according to a three-tiered system of increasing specificity. The first coding involved two broad categories used to identify whether a given condition was a treatment condition or a control condition. The second coding involved 11 categories, including one code for any control condition and 10 different codes for therapy types. The third coding involved 26 categories with more specific descriptors, including five codes for control conditions (e.g., waiting list, placebo) and 21 for therapies (e.g., stimulus control, sleep restriction, progressive muscle relaxation, biofeedback). A brief description of the most frequently used treatment procedures follows. A more detailed description of these treatments is available elsewhere (4, 12, 13).

Stimulus control therapy (21) consists of a set of instructional procedures designed to curtail sleep-incompatible behaviors and to regulate sleep-wake schedules. These procedures are 1) go to bed only when sleepy, 2) use the bed and bedroom only for sleep and sex (i.e., no reading, television watching, eating, or working during the day or at night), 3) get out of bed and go into another room whenever you are unable to sleep for 15–20 minutes, and return only when sleepy again, 4) arise in the morning at the same time regardless of the amount of sleep during the previous night, and 5) do not nap during the day.

Sleep restriction therapy consists of curtailing the amount of time spent in bed to the actual amount of sleep (22). For example, if a person reports sleeping an average of 5 hours per night out of 8 or 9 hours spent in bed, the initial prescribed sleep window (i.e., from initial bedtime to final arising time) would be 5 hours. The allowable time in bed is increased by 15–20 minutes for a given week when sleep efficiency—i.e., (total sleep/time in bed)×100%—exceeds 90%, decreased by the same amount of time when sleep efficiency is lower than 80%, and kept stable when sleep efficiency falls between 80% and 90%. Adjustments are made periodically until an optimal sleep duration is achieved.

Relaxation therapies are designed to alleviate somatic or cognitive arousal. Relaxation procedures, such as progressive muscle relaxation, autogenic training, and biofeedback, focus primarily on somatic arousal (e.g., muscle tension), whereas attention-focusing procedures, such as imagery training, meditation, and thought stopping, target cognitive arousal (e.g., intrusive thoughts, racing mind).

Paradoxical intention is a method that consists of persuading a patient to engage in his or her most feared behavior, i.e., staying awake. The basic premise is that performance anxiety inhibits sleep onset. Thus, if a patient stops trying to sleep and instead genuinely attempts to stay awake, performance anxiety will be alleviated and sleep may come more easily.

Sleep hygiene education is concerned with health practices (e.g., diet, exercise, substance use) and environmental factors (e.g., light, noise, temperature) that may be either detrimental or beneficial to sleep. It also involves basic information about sleep and changes in sleep patterns over the course of the life span.

The studies were coded according to whether treatment was delivered individually, delivered in a group, or self-administered (e.g., with a self-help manual). Treatment was also coded for type of therapist, i.e., professional, trainee, or automated (e.g., audiotape). Treatment duration was coded for the number of actual contact hours and for its duration in weeks.

In addition to the number of subjects, several demographic and clinical characteristics were coded. Depending on the available data, this information was entered either for each individual condition or for the total study group. The demographic variables included gender and age. The clinical characteristics included insomnia duration, the nature of the primary complaint (onset, maintenance, or mixed insomnia), and whether the patients were free from sleep medications upon entering the study. The nature of the study groups was coded along two dimensions, i.e., whether the patients were solicited or unsolicited for the study and the extent to which they represented an analog, community-recruited, clinical, or mixed group.

Each study was also coded for the quality of its design. The following six dimensions were rated on a scale of 1 to 3 to yield a composite score ranging from 6 to 18: a) treatment integrity and reproducibility (1=minimal description, 3=full description of treatment), b) mode of subjects' assignment (1=nonrandomized, 3=randomized with match-

ing), c) single versus multiple therapists, d) attrition rate, e) baseline group equivalency, and f) degree of control for nonspecific effects.

Calculation of Effect Sizes and Improvement Rates

Effect sizes were calculated by subtracting the mean of the control group from the mean of the treated group at posttreatment and dividing by the pooled standard deviations of the two groups (23). When means and standard deviations were not available, effect sizes were estimated by using the procedures outlined by Smith et al. (24) and by Glass et al. (25). Approximately 80% of the effect sizes were calculated by using exact statistics (i.e., means and standard deviations), whereas the remainder were derived from means combined with F or t statistics. Effect sizes were computed only for the studies (N=45) that included a control condition. There were a total of 180 calculated effect sizes, including 91 for sleep-onset latency, 15 for time awake after sleep onset, 38 for number of awakenings, and 36 for total sleep time.

Effect sizes represent standardized z scores that can be interpreted as the distance, in standard deviation units, between the average insomnia patient treated nonpharmacologically and the average control patient. An effect size of zero would indicate that there was no difference between treated and untreated patients, whereas an effect size of 0.50 would indicate that the improvement of the average treated patient was one-half of a standard deviation greater than that of the average control patient. In behavioral science research, an effect size of 0.2 is considered small, one of 0.5 is considered medium, and one of 0.8 is considered a large effect (23).

Improvement rates for each of the four dependent measures were also calculated in absolute values (i.e., difference between pre- and posttreatment) and in percentages. Changes from pre- to posttreatment and from posttreatment to follow-up were calculated for all studies that included means, regardless of whether or not a control condition was available. Of the 59 studies, 14 did not have a control condition. Those studies typically evaluated the effect of a single treatment method or compared the relative efficacy of several therapies. In most cases, they involved clinical patients and precluded the use of a control group. We retained those studies in the present analysis because they provided useful information about patients most typically seen in clinical practice.

Reliability of Coding

All objective variables (e.g., age, gender, treatment duration, sleep measures) were coded by one of us (C.M.M.) and by a research assistant. Because those variables were objective and well-defined operationally, they were less subject to disagreements between raters. Other variables, however, were more subjective and more likely to produce disagreements among investigators. For example, disagreements may arise as to how a given intervention should be labeled or about the quality of a study. Accordingly, variables involving a subjective rating (therapy types, design quality, etc.) were coded by two independent raters who were not involved in calculating improvement rates (i.e., effect sizes, change scores). Disagreements over treatment coding were resolved by discussion, whereas ratings of design quality were averaged to yield a single score. Interrater reliability coefficients were computed for a random sample of 30% of those codings. The kappa values were 0.60 for treatment format (individual, group, self-administered), 0.46 for therapist (professional, trainee, automated), 0.72 for diagnosis (onset, maintenance, mixed insomnia), 0.83 for nature of study group (analog, community-recruited, clinical, mixed), and 0.84 for source of subjects (solicited versus unsolicited). The kappas for the three-tiered coding of therapy types were, respectively, 0.91 (tier 1), 0.87 (tier 2), and 0.88 (tier 3).

RESULTS

Characteristics of Subjects and Studies

A total of 2,102 patients with insomnia were involved in the 59 studies reviewed. The subjects were predominantly women (mean=59.9%, SD=18.9%), and their age averaged 44.2 years (SD=12.6). They were typically recruited from the community to participate in controlled trials (i.e., 51% of all studies). The remaining subjects were selected from college student populations (20%) (a practice more common in the early studies), were self-referred or referred by health care professionals (14%), or were mixed community/clinical groups (15%). The typical criteria for subjects' inclusion in the studies were chronicity of the insomnia (i.e., duration greater than 6 months), sleep-onset latency or time awake after sleep onset greater than 30 minutes per night, and total sleep time less than 6.5 hours per night for 3 nights or more per week. Those criteria are consistent with those of the *International Classification of Sleep Disorders* (26). The average insomnia duration for all participants was 11.23 years (SD=3.73).

There were a total of 183 conditions (126 treatment and 57 control conditions), for an average of 3.1 conditions per study and a mean of 11.5 subjects per condition (SD=7.8). The treated subjects received short-term, sleep-focused intervention. The average treatment time was 5.0 hours (SD=3.5, range=1–20), and it was provided over an average period of 5.0 weeks (SD=2.9, range=1–16). Follow-up lasted a mean of 25.9 weeks (SD=25.6).

Treatments Versus Control Conditions

The first question of interest is whether nonpharmacological interventions are effective for the management of insomnia and, if so, whether they are more effective than no treatment at all. Table 1 presents summary data for the four dependent measures of interest: sleep-onset latency, time awake after sleep onset, number of awakenings, and total sleep time. The data are combined to compare all treatment conditions, regardless of their nature, with all control conditions. At baseline, the subjects were taking over 1 hour to fall asleep and reported an average of 70 minutes of wakefulness after sleep onset. They also reported about two awakenings per night and less than 6 hours of total sleep time per night. We conducted a series of t tests for independent samples on the baseline sleep measures, and there was no significant group difference between treatment and control subjects in any of these measures ($p>0.1$ in all cases).

The average effect size for sleep-onset latency was 0.88. This number, which is based on 91 treatment-control comparisons, is a standardized z score and indicates that the average outcome for treated patients was 0.88 standard deviation superior to that for untreated patients. A corresponding percentile rank of 81 is obtained by looking under a normal curve distribution at 0.88 standard deviation above the mean. This 81st percentile rank indicates that the average treated patient was falling asleep faster after treatment than 81% of those who did not receive treatment. (Percentile ranks are approximate and only providing that the data

TABLE 1. Efficacy of Psychological Treatments and Control Conditions in Studies of Insomnia

Sleep Measure	Pretreatment Value		Posttreatment Value		Follow-Up			Pre- to Posttreatment Change				
								Difference			Effect Size	
	Mean	SD	Mean	SD	Number of Conditions	Value Mean	Value SD	Number of Conditions	Change* Value	Change* %	Number of Comparisons	z
Sleep-onset latency (minutes)											91	0.88
Treatments	64.3	23.2	36.6	15.8	80	32.5	16.6	116	-27.7	43.1		
Control conditions	63.9	24.5	55.9	25.7	17	57.5	32.0	53	-8.0	12.6		
Time awake after sleep onset (minutes)											15	0.65
Treatments	70.3	31.3	37.6	15.7	20	37.8	20.5	25	-32.7	46.4		
Control conditions	67.0	15.8	56.7	17.6	1	45.3	0.0	9	-10.2	15.3		
Number of awakenings per night											38	0.53
Treatments	1.9	0.6	1.3	0.6	31	1.8	1.0	52	-0.6	29.8		
Control conditions	1.7	0.7	1.5	0.7	5	1.8	0.5	23	-0.2	9.5		
Total sleep time (minutes)											36	0.42
Treatments	349.4	43.9	377.9	41.0	35	395.9	44.5	55	28.5	8.2		
Control conditions	357.2	38.8	361.4	36.1	4	366.2	44.3	19	4.2	1.2		

*Because of rounding errors, some of the values for pre- to posttreatment difference scores and percentage improvements may be slightly different from those obtained from the values in this table.

are normally distributed. Although normative data for various EEG sleep measures are available, no comparable self-reported data are.) The mean effect size for time awake after sleep onset was 0.65, which corresponds to a percentile rank of 74. This effect size, which was based on 15 treatment-control comparisons, shows that patients with sleep-maintenance insomnia were better off after treatment than 74% of the untreated subjects. The mean effect sizes for number of awakenings and for total sleep time were, respectively, 0.53 and 0.42, corresponding to percentile ranks of 70 and 66, respectively. All four effect sizes were reliably greater than zero ($p<0.001$ in all cases).

The average sleep-onset latency for the treated subjects after treatment (table 1) was significantly lower than that for the control subjects ($t=-5.98$, $df=171$, $p<0.0001$). The rate of pre- to posttreatment improvement in sleep-onset latency was significantly greater in the treated subjects than in the control subjects, whether analyzed in terms of difference scores ($t=7.28$, $df=171$, $p<0.0001$) or percentages ($t=8.72$, $df=171$, $p<0.0001$). Changes from pre- to posttreatment were analyzed separately for percentage improvements and difference scores. The patterns of results for the two variables were almost identical. Therefore, only statistics on percentage improvements are reported hereafter.

The posttreatment time awake after sleep onset (table 1) was also significantly lower in the treated subjects than in the control subjects ($t=-2.94$, $df=33$, $p<0.01$). The degree of change from pre- to posttreatment was significantly greater in the treated subjects than in the control subjects ($t=4.11$, $df=33$, $p<0.0001$).

There was no significant difference between the treated and control subjects in the absolute number of awakenings at posttreatment (table 1). However, the percentage improvement from pre- to posttreatment was significantly greater in the treated subjects than in the control subjects ($t=4.18$, $df=73$, $p<0.0001$).

The posttreatment total sleep time was not significantly different in the treated and the control subjects (table 1). However, the improvement rate was significantly greater in the treated subjects than in the untreated ones ($t=-4.24$, $df=75$, $p<0.0001$).

Durability of Treatment Gains

Follow-up data were available for 48 of the 59 studies reviewed (98 treatment and 33 control conditions). When more than one follow-up was conducted, data from the latest one were retained for the present analysis. The average duration of the follow-ups was 25.9 weeks (SD=25.6, range=3–156). The average effect sizes for the studies that still had a control group at follow-up were 0.92 for sleep-onset latency (20 comparisons), 0.58 for time awake after sleep onset (one comparison), 0.56 for number of awakenings (seven comparisons), and 0.51 for total sleep time (nine comparisons). Between-group comparisons with independent t tests revealed significantly shorter sleep-onset latency for the treated subjects than for the control subjects ($t=-4.60$, $df=96$, $p<0.0001$). Although the absolute values for two of the remaining three outcome measures (time awake after sleep onset and total sleep time) were more improved in the treatment groups than in the control groups, those differences were not statistically significant. This was mostly because of the small number of control conditions (one and four, respectively) for which follow-up data on these variables were available. Within-group comparisons (paired t tests) of posttreatment and follow-up revealed significant additional improvements in the treatment group for sleep-onset latency ($t=3.36$, $df=79$, $p<0.0001$) and total sleep time ($t=-3.22$, $df=35$, $p<0.005$). There were no significant changes in time awake after sleep onset and number of awakenings for the same period, suggesting that clinical gains achieved at posttreatment were well maintained over time. Within-group analyses for the control conditions revealed no significant

TABLE 2. Efficacy of Psychological Treatments for Reducing Sleep-Onset Latency in Studies of Insomnia

Treatment	Pretreatment Value		Posttreatment Value		Follow-Up			Pre- to Posttreatment Change				
								Difference			Effect Size	
					Number of Conditions	Value		Number of Conditions	Change		Number of Comparisons	
	Mean	SD	Mean	SD		Mean	SD		Value	%		z
Individual treatments												
Stimulus control	64.3	15.8	32.5	9.2	14	32.2	11.7	20	−31.8	49.5	15	0.81
Sleep restriction	50.7	4.2	21.4	3.2	2	27.0	5.7	3	−29.3	57.8	1	0.98
Relaxation, somatic	67.4	28.2	40.6	17.9	21	40.2	22.8	37	−26.8	39.8	32	0.83
Relaxation, cognitive	63.8	22.6	34.0	9.5	7	27.4	11.3	9	−29.8	46.7	7	1.20
Biofeedback	52.8	20.1	32.9	18.3	11	27.0	14.5	13	−19.9	37.7	7	1.00
Paradoxical intention	59.8	11.2	41.8	14.3	3	33.5	14.2	9	−18.0	30.1	9	0.63
Sleep hygiene education	86.1	40.3	62.8	24.6	3	52.9	16.3	3	−23.3	27.1	2	0.71
Multicomponent therapies	68.9	23.8	32.6	12.7	18	26.7	11.0	20	−36.3	52.7	15	1.05
All treatments combined[a]	64.3	—	36.6	—	80	32.5	—	116	−27.7	43.1	91	0.88
All control conditions combined	63.9	—	55.9	—	17	57.5	—	53	−8.0	12.6		

[a]Some totals differ from the sums of all conditions because some treatment conditions are not included in this table.

TABLE 3. Efficacy of Psychological Treatments for Reducing Time Awake After Sleep Onset in Studies of Insomnia

Treatment	Pretreatment Value		Posttreatment Value		Follow-Up			Pre- to Posttreatment Change				
								Difference			Effect Size	
					Number of Conditions	Value		Number of Conditions	Change		Number of Comparisons	
	Mean	SD	Mean	SD		Mean	SD		Value	%		z
Individual treatments												
Stimulus control	84.0	33.0	43.5	18.2	5	39.7	14.0	6	−40.5	48.2	5	0.70
Sleep restriction	109.0	55.4	32.9	9.7	2	45.7	14.6	3	−76.1	69.8	1	0.76
Relaxation, somatic	59.9	22.0	42.7	18.7	3	57.5	37.8	4	−17.2	28.7	1	0.06
Relaxation, cognitive	70.0	13.7	51.1	6.0	3	41.6	16.3	3	−18.9	27.0	2	0.28
Biofeedback	45.4	24.0	18.3	18.1	1	19.5	0.0	2	−27.1	59.7	2	0.70
Paradoxical intention	62.2	0.0	28.3	0.0	1	10.7	0.0	1	−33.9	54.5	1	0.81
Sleep hygiene education	81.1	0.0	59.0	0.0	1	50.5	0.0	1	−22.1	27.3	—	—
Multicomponent therapies	53.2	6.0	27.9	1.6	3	21.5	15.1	4	−25.3	47.6	3	0.92
All treatments combined[a]	70.3	—	37.6	—	20	37.8	—	25	−32.7	46.4	15	0.65
All control conditions combined	67.0	—	56.7	—	1	45.3	—	9	−10.2	15.3		

[a]Some totals differ from the sums of all conditions because some treatment conditions are not included in this table.

change in any of the measures from posttreatment to follow-up.

Comparative Efficacy of Treatment Modalities

More than a dozen interventions were evaluated, and eight of them were tested frequently enough to yield meaningful estimates of improvement rates. Table 2 summarizes the clinical benefits of those treatment modalities for sleep-onset latency. The effect sizes varied from 0.63 (paradoxical intention) to 1.20 (cognitive relaxation); those based on two or more studies were all significantly greater than zero ($p<0.05$ in all cases). A one-way analysis of variance (ANOVA) for percentage improvement in sleep-onset latency revealed a significant overall treatment effect ($F=12.37$, $df=8, 158$, $p<0.0001$). Post hoc comparisons using the Newman-Keuls procedures indicated that, except for paradoxical intention and sleep hygiene education, all individual treatment methods produced percentages of improvement that were significantly greater than those for the control subjects ($p<0.05$ in all cases). Sleep restriction, multicomponent therapies, stimulus control, and cognitive relaxation, respectively, yielded the largest percentage reductions in sleep-onset latency. With the exception of sleep hygiene education, most therapies lowered the absolute posttreatment value of sleep latency below or near the 30-minute cutoff criterion typically used to define insomnia. There was no significant difference between stimulus control, relaxation-based, and multicomponent interventions. Comparison of the three stress-reduction methods (i.e., relaxation procedures and biofeedback) revealed a slight superiority for relaxation procedures targeting cognitive arousal (e.g., meditation, imagery training), although this difference was not statistically significant. There was no significant difference between active biofeedback procedures, i.e., electromyogram (EMG) and EEG, and placebo biofeedback procedures ($F=1.64$, $df=2, 12$).

Table 3 presents data on the comparative efficacy of therapies for reducing time awake after sleep onset, the most commonly targeted symptom among patients

TABLE 4. Efficacy of Psychological Treatments for Reducing Number of Night Awakenings in Studies of Insomnia

Treatment	Pretreatment Value		Posttreatment Value		Follow-Up			Pre- to Posttreatment Change				
								Difference			Effect Size	
	Mean	SD	Mean	SD	Number of Conditions	Value Mean	Value SD	Number of Conditions	Change Value	Change %	Number of Comparisons	z
Individual treatments												
Stimulus control	1.9	0.5	1.4	0.5	7	1.5	0.3	9	−0.5	26.3	11	0.59
Relaxation, somatic	1.7	0.8	1.0	0.5	5	2.3	2.4	17	−0.7	41.2	13	0.56
Relaxation, cognitive	2.1	0.8	1.7	0.9	3	2.1	0.5	4	−0.4	19.1	3	0.56
Biofeedback	2.1	0.5	1.6	0.7	6	2.0	0.7	7	−0.5	23.8	2	0.97
Paradoxical intention	1.8	0.8	1.0	0.9	1	1.9	0.0	4	−0.8	44.4	4	0.73
Sleep hygiene education	1.7	0.2	1.5	0.1	3	1.3	0.2	3	−0.2	11.8	1	−0.12
Multicomponent therapies	1.9	0.5	1.6	0.6	5	1.6	0.7	6	−0.3	15.8	4	−0.05
All treatments combined[a]	1.9	—	1.3	—	31	1.8	—	52	−0.6	29.8	38	0.53
All control conditions combined	1.7	—	1.5	—	5	1.8	—	23	−0.2	9.5		

[a]Some totals differ from the sums of all conditions because some treatment conditions are not included in this table.

with sleep-maintenance insomnia. Substantially fewer studies (N=14) focused on this population. The effect sizes varied between 0.06 (somatic relaxation) and 0.92 (multicomponent therapies). Of the procedures tested in more than one study, stimulus control, biofeedback, and multicomponent therapies yielded effect sizes that were significantly greater than zero ($p<0.05$). A one-way ANOVA for percentage improvement at posttreatment showed a significant effect for treatment ($F=5.76$, $df=8, 24$, $p<0.001$). Post hoc comparisons indicated that stimulus control, sleep restriction, multicomponent procedures, and biofeedback produced improvement rates significantly greater than that of the control group ($p<0.05$ in all cases). Sleep restriction yielded the highest improvement rate, followed by stimulus control and multicomponent therapies. Biofeedback and paradoxical intention produced substantial benefits as well, but these methods were tested in only two and one studies, respectively. Sleep restriction and multifocused interventions reduced time awake after sleep onset to near the 30-minute cutoff criterion used to define sleep-maintenance insomnia. Clinical gains were particularly well maintained in the multifocused intervention conditions.

There were few notable differences between therapies in the remaining two outcome variables, even though there was a significant overall treatment effect for both number of awakenings ($F=4.44$, $df=7, 64$, $p<0.001$) and total sleep time ($F=3.41$, $df=8, 65$, $p<0.005$). The effect sizes for number of awakenings (table 4) ranged from −0.12 to 0.97, and only stimulus control and somatic relaxation produced effect sizes that were reliably greater than zero ($p<0.05$). The effect sizes for total sleep time (table 5) showed large variability across treatment methods, ranging from −1.06 (sleep restriction) to 1.16 (sleep hygiene education). Three treatment methods yielded effect sizes reliably greater than zero: multicomponent therapies, stimulus control, and paradoxical intention. The absolute increase in total sleep time from pre- to posttreatment was fairly modest, averaging less than half an hour, although by follow-up sleep duration had increased from pretreatment by more than 45 minutes.

Variables Moderating Treatment Efficacy

One-way ANOVAs were computed to determine whether individual, group, and self-administered treatments produced different outcomes. There was only one significant difference, in number of awakenings ($F=10.39$, $df=2, 35$, $p<0.0005$). Post hoc comparisons using Newman-Keuls tests revealed that individual treatment yielded a greater effect size than did either group or self-administered treatment, whereas group therapy was more effective than the self-administered format ($p<0.05$ in all cases). For all three other variables, there was a nonsignificant trend for this same pattern of results in that individual and group therapy produced better outcomes than self-administered therapy.

A one-way ANOVA was computed for the data on sleep-onset latency to examine whether treatment administered by a professional was more effective than that administered by a trainee or treatment that is automated (e.g., manual, tape). A significant overall effect was obtained ($F=3.42$, $df=2, 106$, $p<0.05$), but post hoc comparisons failed to yield significant differences between any two groups. Because there were only one or two conditions involving self-administered treatments for the remaining three variables, two-tailed t tests for independent samples (i.e., professional versus trainee) were conducted. Treatment delivered by a professional yielded higher improvement rates for time awake after sleep onset than treatment delivered by a trainee ($t=2.44$, $df=23$, $p<0.05$). There was no significant difference for either number of awakenings or total sleep time.

Significant effects of subject type on sleep-onset latency were obtained at both baseline ($F=6.80$, $df=3, 115$, $p<0.001$) and posttreatment ($F=4.29$, $df=3, 115$, $p<0.01$). Groups composed of community-recruited subjects, patients referred by health care professionals, or mixed groups made up of a combination of those two had more severe sleep-onset insomnia than did

TABLE 5. Efficacy of Psychological Treatments for Increasing Total Sleep Time in Studies of Insomnia

Treatment	Pretreatment Value		Posttreatment Value		Follow-Up			Pre- to Posttreatment Change				
								Difference			Effect Size	
	Mean	SD	Mean	SD	Number of Conditions	Value Mean	Value SD	Number of Conditions	Change Value	Change %	Number of Comparisons	z
Individual treatments												
Stimulus control	333.9	32.5	371.3	20.2	3	380.7	69.2	8	37.4	11.2	7	0.41
Sleep restriction	303.7	45.8	317.5	45.6	2	370.9	29.5	3	13.8	4.5	1	−1.06
Relaxation, somatic	338.0	45.0	375.8	41.5	7	384.3	59.4	15	37.8	11.2	10	0.25
Relaxation, cognitive	351.1	0.0	360.6	0.0	1	336.7	0.0	1	9.5	2.7	1	0.28
Biofeedback	366.4	55.0	390.3	33.9	8	398.7	26.2	10	23.9	6.5	4	0.38
Paradoxical intention	350.7	24.0	378.4	10.4	2	386.9	12.9	5	27.7	7.9	5	0.46
Sleep hygiene education	371.0	30.7	388.8	60.4	3	401.3	67.7	3	17.8	4.8	2	1.16
Multicomponent therapies	369.3	39.5	393.7	49.9	9	418.2	41.7	10	24.4	6.6	6	0.75
All treatments combined[a]	349.4	—	377.9	—	35	395.9	—	55	28.5	8.2	36	0.42
All control conditions combined	357.2	—	361.4	—	4	366.2	—	19	4.2	1.2		

[a]Some totals differ from the sums of all conditions because some treatment conditions are not included in this table.

groups made up of analog subjects recruited from college student populations ($p<0.05$). However, there were no differences in improvement in sleep-onset latency among the various groups. Of the 15 studies investigating sleep-maintenance insomnia, none used analog subjects and there were no group differences among community, clinical, or mixed groups either in terms of baseline symptom severity or rate of improvement in time awake after sleep onset. There was a significant group difference in the number of awakenings at both pretreatment ($F=5.35$, $df=3, 48$, $p<0.005$) and posttreatment ($F=8.99$, $df=3, 48$, $p<0.001$). Community, clinical, and mixed groups all had more night awakenings than did the analog groups ($p<0.05$ in all cases) but did not differ among themselves. There was also a significant group effect for percentage improvement ($F=4.21$, $df=3, 48$, $p<0.01$). The percentage reduction in number of awakenings was significantly higher in the analog groups than in the other three subtypes ($p<0.05$ in all cases), but those latter types did not differ among themselves. Of the studies for which total sleep time was reported, none used analog subjects. For the remaining three groups, there was a significant effect at both pretreatment ($F=4.11$, $df=2, 54$, $p<0.05$) and posttreatment ($F=3.38$, $df=2, 54$, $p<0.05$). In both cases, the clinical subjects reported more total sleep time than either the community-recruited or mixed groups ($p<0.05$), but the last two groups did not differ from each other. Although there were no significant differences for percentage improvement, there was a significant effect for effect size ($F=17.47$, $df=2, 33$, $p<0.0001$); the clinical groups produced more favorable outcomes than either of the other two group types ($p<0.05$).

There were no group differences in any of the outcome measures between subjects who were drug free upon entering the studies and those who were permitted to continue using hypnotic medication. The only finding that approached significance ($p=0.06$) was that the medication-free subjects experienced a larger effect (1.00) on sleep-onset latency than did those allowed to use medication (0.73).

A series of analyses were conducted to determine whether patients with a single problem (onset versus maintenance insomnia) responded to treatment differently from those with mixed insomnia. For patients with sleep-onset insomnia, the primary outcome measure of interest is sleep-onset latency. A series of t tests for independent samples (onset versus mixed) revealed that the patients with sleep-onset insomnia responded better to treatment than did the patients with mixed insomnia in terms of percentage improvement ($t=1.92$, $df=126$, $p<0.05$). Both the number of awakenings and the duration of wakefulness after sleep onset are of clinical interest for patients with sleep-maintenance insomnia. There was no significant difference in time awake after sleep onset between the patients with maintenance-only insomnia and those with mixed insomnia. However, there was a greater reduction in night awakenings among patients with mixed insomnia than among those with maintenance insomnia only ($t=-2.13$, $df=30$, $p<0.05$). A one-way ANOVA for total sleep time revealed a significant group difference at both pretreatment ($F=4.30$, $df=2, 53$, $p<0.02$) and posttreatment ($F=9.85$, $df=2, 53$, $p<0.001$). At both points, the patients with sleep-onset insomnia reported significantly more sleep time than the patients with either maintenance or mixed insomnia ($p<0.05$ in all cases), but there was no difference in improvement rate over time.

We computed correlations between outcome and several moderating variables, including age, gender, insomnia chronicity, treatment duration, and quality of the study design. These analyses were conducted for all treatment conditions combined. There was no significant correlation between age or gender and any of the outcome measures. Insomnia duration was negatively correlated with effect size for sleep-onset latency ($r=-0.49$, $df=47$, $p<0.001$) and total sleep time ($r=-0.42$, $df=24$, $p<0.05$). Therapy time (in hours) was significantly correlated with effect size for frequency of night

awakenings (r=−0.43, df=38, p<0.01) and total sleep time (r=0.57, df=39, p<0.001), and the correlation with time awake after sleep onset approached significance (r=0.42, df=15, p=0.12). Treatment duration (in weeks) was positively correlated with effect size for total sleep time (r=0.51, df=39, p<0.002). Design quality was negatively correlated with effect size for sleep-onset latency (r=−0.24, df=91, p<0.03) but for no other outcome measure.

DISCUSSION

The findings from this meta-analysis indicate that nonpharmacological interventions produce reliable and durable clinical benefits in the treatment of sleep-onset and maintenance insomnia. Treated insomnia patients improved significantly more on the two main target symptoms than did patients who received attention, placebo, or no treatment. For sleep-onset latency, the treated patients were better off than 81% of the untreated patients, whereas for time awake after sleep onset the treated patients were more improved than 74% of the control subjects. The effect sizes for the number of awakenings and total sleep time were more modest but still reliably greater than zero. The clinical significance of these results is illustrated by the absolute values for the two main target symptoms, sleep latency and time awake after sleep onset, at posttreatment. Although improvement rates of 50%–60% are still far from an ideal outcome, the end point status of patients may be a better indicator of clinical significance. In that regard, the posttreatment values for sleep-onset latency (36.6 minutes) and time awake after sleep onset (37.6 minutes) were close to the cutoff value (i.e., 30 minutes) typically used to define sleep-onset and sleep-maintenance insomnia.

The clinical gains achieved by the end of treatment were well maintained at the follow-ups, which averaged 6 months in duration. These results are particularly encouraging given the chronic nature of insomnia (average duration, 11.23 years) and the relatively short duration of treatment (average, 5.0 weeks). The temporal stability of the therapeutic changes is perhaps the greatest strength of behavioral interventions for insomnia. Although the overall magnitude of improvement at posttreatment was moderate, these changes were well maintained over time, and for some measures (sleep latency and total sleep time), additional improvements were made from posttreatment to follow-up.

Comparison of the treatment procedures indicated that stimulus control was the most effective single therapy for either sleep-onset or maintenance insomnia. Sleep restriction produced even greater benefits, but this procedure has been used in a limited number of clinical trials thus far. Of the various relaxation-based methods, procedures aimed at reducing cognitive arousal (intrusive thoughts, racing mind) were slightly superior to those targeting physiological arousal (muscle tension). Comparison of biofeedback procedures showed no difference between methods providing accurate feedback of EMG or EEG activity and placebo conditions providing bogus feedback. This finding suggests that it is the perception of control, rather than actual control, that is important in overcoming chronic insomnia. Multicomponent interventions have produced results that are comparable, but not always superior, to the most effective single-therapy components (i.e., stimulus control and sleep restriction). Earlier studies sometimes combined various procedures in a hit-or-miss fashion and without much of a rationale for doing so (15). There is a need for multiperspective approaches, but these must be planned to target the various facets of insomnia.

The data on potential moderating factors in treatment outcome indicate that treatment provided by a professional therapist either individually or in a group yields a more favorable outcome than treatment that is self-administered, automated, or delivered by a trainee. Although 50% of the subjects included in the studies were recruited from the community, the finding of no difference in improvement between these solicited patients and patients seeking treatment suggests that clinical outcome obtained in research studies may well generalize to a clinical context. Gender and age were unrelated to outcome, a finding of particular importance given that older women are the group consuming most hypnotic medications (1). Insomnia duration was negatively correlated with outcome for sleep latency and total sleep time. These results, combined with other findings (2) suggesting that insomnia may increase the vulnerability to major depression, point to the need for early interventions.

The present results must be interpreted with some cautions because they are based on sleep diary data. The discrepancy between subjective and objective measures of sleep measures is well documented. Patients with insomnia tend to overestimate sleep latency and time awake after sleep onset and to underestimate the number of awakenings and sleep time (20). Nevertheless, the few studies that have used polysomnography to evaluate outcome have shown that psychological interventions are effective not only in altering sleep perceptions but also in improving objective sleep measures (27–30). The magnitude of recorded improvements in polysomnographic measures is somewhat smaller but in the same direction as the magnitude of changes shown in sleep diaries. Subjective improvements in sleep patterns have also been paralleled by data obtained from behavioral assessment devices (31–34) and by collateral reports from significant others (27, 34, 35). Finally, because the subjective complaint of poor sleep is an essential feature of insomnia, subjective perception of sleep improvements may be necessary in alleviating the psychological distress often underlying this complaint.

These findings have several implications for the clinical management of insomnia. First, although psychological treatment may be more expensive and time consuming than pharmacotherapy, the current data indicate that it may prove more cost-effective in the long

run. Second, the effective management of insomnia does not require long-term psychotherapy. In fact, there is no empirical evidence that psychotherapy alone is effective for insomnia. Instead, treatment must focus on sleep and target perpetuating factors, such as maladaptive sleep habits and dysfunctional sleep cognitions (4). Third, general sleep hygiene recommendations about caffeine, exercise, and diet are unlikely to be sufficient for treating chronic insomnia. Although sleep hygiene is usually incorporated into multicomponent interventions, simply asking patients to avoid alcohol and to exercise is unlikely to be successful.

Despite the widespread prevalence of sleep complaints, fewer than 15% of insomnia sufferers ever seek professional help (1), and the vast majority of those who do never receive treatment from a mental health professional (36). Of the patients seeking insomnia treatment from physicians, one-half are given prescriptions for sleep medications (37). Psychosocial interventions are underused in the management of insomnia, even though recent evidence suggests that they are more acceptable than pharmacotherapy to prospective patients seeking professional help (17). The present results, which are based on over 2,000 insomnia patients treated with psychological interventions, are promising. A similar meta-analysis would be helpful in evaluating the efficacy of pharmacotherapy.

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