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Identifying Effective Psychological Treatments for Insomnia: A Meta-Analysis

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Insomnia is a debilitating and widespread complaint. Concern over the iatrogenic effects of pharmacological therapies has led to the development of several psychological treatments for insomnia. To clarify the effects of these treatments, 66 outcome studies representing 139 treatment groups were included in a meta-analysis. The results indicated that psychological treatments produce considerable enhancement of both sleep patterns and the subjective experience of sleep. In terms of enhancing sleep onset, active treatments were all superior to placebo therapies but did not differ greatly in efficacy. Greater therapeutic gains were available for participants who were clinically referred and who were not regular users of sedative hypnotics. Future research directions are suggested.

Insomnia, defined as the subjective inability to obtain adequate sleep (Gillin & Byerley, 1990), is a distressing and often debilitating condition that can affect health, daytime performance, relationships, mood, and psychological well being (Lacks, 1987; Sloan & Shapiro, 1993). Estimates of the prevalence of insomnia typically range between 15% to 20% for chronic insomnia and 30% to 40% for occasional or transient insomnia (Mellinger, Balter, & Uhlenhuth, 1985). Insomnia can therefore be regarded as a significant problem in the community.

The treatment of choice for insomnia, for practitioner and patient alike, has been the prescription of sedative hypnotics or other sleep-inducing agents (Bliwise, 1991). Pharmacological treatments may, however, involve a variety of iatrogenic effects, including poor-quality sleep (Kales & Kales, 1987), deterioration of daytime functioning (Johnson & Chernik, 1982), and, if used regularly, the development of psychological dependence, tolerance, and addiction (Espie, 1991). Withdrawal from the addiction cycle is made particularly difficult by the effects of "rebound insomnia" (Killen & Coates, 1979). A further consideration is that the habitual use of sleeping medication may involve substantial financial expense (Hauri, 1979).

Concern over the iatrogenic effects of pharmacological approaches has led researchers to explore alternative treatments for insomnia. These have included stimulus control (Bootzin, 1972); paradoxical intention (Frankl, 1955); sleep restriction therapy (Spielman, Saskin, & Thorpy, 1987); and relaxation-based therapies such as progressive muscle relaxation (Jacobson, 1938), meditation, systematic desensitization, imagery, autogenic training, and hypnosis. (For a detailed description of each treatment, see Espie [1991].)

Despite extensive research, the absolute and relative effects of these treatments are not clear. In general, qualitative reviews of the literature (Bootzin & Nicassio, 1978; Borkovec, 1982; Espie, 1991; Gillin & Byerley, 1990; Killen & Coates, 1979; Knapp, Downs, & Alperson, 1976; Montgomery, Perkin, & Wise, 1975; Ribordy & Denney, 1977; Turner & Di Tomasso, 1980) have been inconclusive. Although some reviewers have suggested that nonrelaxation treatments—in particular, stimulus control—are the most potent (e.g., Borkovec, 1982), others have concluded that the treatments do not differ in efficacy (e.g., Bootzin & Nicassio, 1978; Turner & Di Tomasso, 1980). Furthermore, it has been proposed that differences in efficacy may merely reflect the variable being measured and the time of measurement (Espie, 1991). For example, although stimulus control may cause greater improvements in sleep pattern measures such as sleep onset latency, particularly in the short term, relaxation-based approaches may be superior over the long term on subjective evaluations of sleep quality (Espie, Lindsay, Brooks, Hood, & Turvey, 1989).

It has been hypothesized that several treatment and patient characteristics may be important to therapeutic outcome, but they have been largely neglected as a subject for empirical investigation, and clarification of their effects has proved problematic for qualitative reviews. Identification of the conditions under which optimal effects are obtained may allow treatment success and efficiency to be maximized (Chambers, 1992; Sanavio, 1988).

Existing reviews of the literature may, however, have been compromised by their narrative approach (Cook & Leviton, 1980; Greenberg & Folger, 1988; Strube & Hartmann, 1983). Meta-analysis, a comprehensive form of review based on quantitative rigor and the statistical standards that are applied in primary data analysis, may be better able to exploit the information available within the research body on behavioral treatments of insomnia (Hunter, Schmidt, & Jackson, 1982; Wolf, 1986).

Although the majority of reviews have been narrative, four previous studies have applied quantitative approaches to the lit-

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erature field. Morin, Culbert, Kowatch, & Walton (1989) integrated 43 studies of nonpharmacological treatments for insomnia, using measures such as hours of sleep, time taken to fall asleep, and the number of awakenings during the night. A standard meta-analysis was also performed by Morin, Culbert, and Schwartz (in press), using 45 studies with matching control groups. This study investigated the influence of treatment modality, patient characteristics, and design characteristics on effect sizes, using the same sleep parameters as those of Morin et al. (1989). Morin, Culbert, and Schwartz also integrated an additional 15 studies without control groups by calculating average parameters at pretreatment, posttreatment, and follow-up and the percentage of improvement for each treatment modality. A similar strategy has been used in two other studies (Lacks & Morin, 1992; Lichstein & Fischer, 1985), although it is not analogous to standard meta-analysis.

In general, however, these studies only examined a small number of moderator variables, and the investigation of both these variables and the comparative efficacy of treatments was limited by the use of uncontrolled mean effect sizes. The present meta-analysis was unique in that we were able to use regression analyses to evaluate treatment effects independent from moderator variables. In addition, we were able to apply the Hunter et al. (1982) meta-analytic strategy of correcting observed variance for variance that is due to statistical artifacts. Furthermore, it allowed us to consider the effects of treatment on subjective ratings of sleep, in addition to sleep parameters, in both the long term and short term. Finally, by using unpublished studies and treatment groups from studies without control conditions, we were able to obtain the largest and most representative integration of studies to date.

The intention of the present meta-analysis was to provide practical directions for the treatment of insomnia. The following research questions were investigated: (a) How do psychological treatments differ among each other and from placebo treatment in their effectiveness? (b) Which treatments are most effective for the different symptoms of insomnia, over both the short term and long term? (c) Which treatment and patient variables are important to outcome? (d) How are effect sizes related to systematic differences in study methodology? The identification of effective psychological treatments for insomnia and of variables that are important to therapeutic outcome may lead to a greater capacity to relieve this significant problem in the community.

Method

Studies

Published studies were identified through searches (using the keyword "insomnia") of CD-ROM PsycLIT and MEDLINE for the period 1973–1993 and the reference lists of relevant review articles and books. Unpublished studies were identified from listings of dissertations and theses over the same period. Unfortunately, it was not possible to obtain all the unpublished studies that were identified. Written requests were sent to prominent researchers in a further attempt to obtain unpublished research reports. Unpublished studies were included because publication policy may often be biased toward the reporting of significant findings or findings that comply with popular theory (Strube & Hartmann, 1983), and therefore, effect sizes may be overestimated if based solely on published sources. Furthermore, publication status was able to be examined as a potential moderator variable to determine whether a bias existed.

Treatment groups from the studies were included in the meta-analysis if (a) they involved the use of a psychological treatment, or a combination of psychological treatments, for insomnia (treatments that required equipment that was considered to be impractical for general, wide-scale use were excluded; treatments that were evaluated by using two or fewer groups in the full set of obtained studies were excluded on the grounds that their inclusion would have been unsound and that combining their effects under the category of "other treatments" would have produced results lacking in conceptual clarity and clinical usefulness); (b) they involved at least five insomniacs whose health and background were described as normal or, through lack of reporting, were assumed to be normal (treatment groups that used specialized participants [e.g., prisoners, alcoholics] or insomniacs clinically diagnosed with other sleep disorders [e.g., with midwinter insomnia], psychological disturbances [e.g., anxiety, depression], or serious medical disorders [e.g., cancer] were excluded); or (c) the study was written in English.

The literature search yielded 139 treatment groups from 66 studies that met the inclusion criteria. One hundred twenty-five treatment groups were located from 58 published studies listed in PsycLIT or MEDLINE, 9 groups were from 5 theses from Australia, and 5 groups were extracted from 3 dissertations from North America. Using a random sample of 123 PsycLIT studies, we measured interrater agreement for the inclusion of treatment groups (according to the aforementioned criteria) at 100%.

Measures

Whenever studies used several dependent variables or posttherapy measurements, they produced multiple effect sizes for each treatment group. In an attempt to fully exploit the available information yet minimize the problems caused by including multiple effect sizes from single treatment groups (see Bangert-Drowns [1986] for a review), we conducted separate meta-analyses for four dependent variable constructs: sleep onset latency (SOL); total sleep time (TST); number of nocturnal awakenings (#A); and subjective evaluation of sleep quality (SQR). Other dependent variables may have provided further insight into treatment efficacy, but they were not measured frequently enough to permit inclusion. Note that effect sizes were calculated using sleep diary data only as there was an insufficient number of groups using objective physiological or observer measures of sleep patterns.

Treatment effects may vary over time and in different ways for each treatment (Espie, 1991). Therefore, two separate meta-analyses were performed for each dependent variable construct, one for "short-term" effect sizes, within 3 months of treatment (Lacks & Powlishta, 1989), and one for "long-term" effect sizes after that period. Long-term effect sizes were based on follow-up measures taken, on average, 8 months after treatment.

If a treatment group produced two or more effect sizes for a particular dependent variable construct within the same period of measurement, then they were combined by calculating the average. As a consequence, the maximum number of effect sizes a single treatment group could contribute to the overall analysis was limited to eight (4 [dependent variables] × 2 [times]).

Estimation of Effect Size and Effect Size Variance

The value d was used for meta-analysis, where d was the standardized, scale-invariant estimate of effect size. Where possible, effect sizes were calculated by dividing the difference between the treatment group pretreatment-to-posttreatment (pre–post) change and the control group pre–post change by the pooled standard deviation (see Glass, McGaw, & Smith, 1981). Where control group comparisons were not possible, effect sizes were calculated by dividing the treatment group pre–post

change by the pooled standard deviation. Fifty-eight (41.7%) of the treatment groups were taken from studies that did not use a control group. The inclusion of these treatment groups allowed for the investigation of a more comprehensive and representative selection of the research literature and permitted the use of regression analyses to examine moderator variable effects. Given that the participants used in the studies were generally chronic insomniacs and that untreated chronic insomniacs do not spontaneously improve (Cartwright & Weiss, 1975; Woolfolk, Carr-Kaffashan, & McNulty, 1976), it seemed acceptable to use pretreatment measures as a "no-treatment" comparison group.[1]

Effect sizes were calculated so that a positive effect size indicated a reduction in insomnia symptomology. The pooled standard deviation was used because it provides a more precise estimate of the population variance than the standard deviation of either pretreatment or posttreatment values (Hedges & Olkin, 1985). When means and standard deviations were not provided, written requests for additional information were sent to the authors of the studies. On the whole, however, these efforts were unsuccessful. In these cases, we estimated the effect sizes from appropriate *F*, *t*, or *p* values, using procedures described by Glass et al. (1981). Only 14.7% of the effect sizes were estimated in this way. Five studies were not able to be included in the meta-analysis because these values were unavailable.

Treatments were classified into one of six categories. Relaxation-based approaches were divided into progressive muscle relaxation (40 groups) and other relaxation (16), which included meditation (3), desensitization (1), imagery relaxation (8), hypnosis (2), and autogenic training (2). Grouping these techniques together was considered appropriate because they appear to share a similar mechanism (Bootzin & Nicassio, 1978). All relaxation-based treatment groups were also combined in comparisons with nonrelaxation treatment groups. Other treatment categories included stimulus control (25 groups), paradoxical intention (14), sleep restriction therapy (4), and combination treatments (23). Combination treatments consisted largely of composites of stimulus control and relaxation (18 groups).

We calculated placebo effect sizes by using the placebo control groups from the studies included in the meta-analysis (17 groups). The majority (12) of placebo approaches used Steinmark and Borkovec's (1974) quasi-desensitization therapy. Although quasi-desensitization resembles relaxation treatments, it has also been frequently used to estimate the influence of nonspecific treatment factors for nonrelaxation treatments. Placebo effect sizes should be subtracted from treatment group effect sizes to provide an estimation of active effects. This adjustment may be particularly important because measurement relied on self-report and may therefore have been particularly susceptible to demand effects.

Average effect sizes and variance of effect sizes were calculated. In an effort to ensure that these values were not distorted by the results from small-sample treatment groups, they were weighted by sample size. Following the recommendations of Hunter et al. (1982), effect size variance was corrected for artifactual variance. Unfortunately, because of limited reporting in the studies, corrections for measurement and range restriction error were not possible. However, we were able to calculate artifactual variance that was due to sampling error using the formula provided by Hunter et al. (1982, p. 102). On the basis of the corrected estimation of variance and the assumption that effect sizes were normally distributed about the mean, we calculated 95% confidence intervals for each effect size.

Although there are advantages to the use of effect size estimates, they may be limited by their lack of intuitive meaning (Rosenthal & Rubin, 1986). To assist in the interpretation of the findings, we applied effect sizes to the hypothetical "average" complaint of insomnia, constructed by calculating mean pretreatment SOL, TST, and #A values. We then calculated expected improvements in terms of actual sleep parameters using these pretreatment means and the appropriate pooled standard deviations. Consider, for example, an average posttreatment SOL effect size of .87. The pretreatment mean SOL was 61.41 min, and the pooled standard deviation was 27.68 min. The effect size represents .87 of a standard deviation improvement (i.e., reduction) in SOL (i.e., .87 × 27.68 min = 24.08 min). SOL may therefore be expected to decrease from 61 to 37 min for the hypothetical average case of insomnia.

Identification of Moderator Variables

Treatment groups were coded on 17 treatment, patient, and design characteristics (see Appendix) according to a priori identification of potential moderators. Design variables were investigated so that biases that were due to methodological features could be identified. A second researcher independently coded a random sample of 44 treatment groups, and intercoder agreement was reached for 94.8% of values overall. There was 100% agreement for seven of the variables, and the lowest agreement was 79.5% for the variable "comprehensive reporting of results."

Because of limited numbers of effect sizes, only posttreatment SOL effect sizes were able to be examined for moderating influences. Although this represented a limitation of the analysis, it is generally recognized that SOL is the primary index of insomnia (Borkovec, 1982). Hunter et al. (1982) proposed that effect sizes should be examined for moderating variables only if approximately 20% or more of the observed variance remains after removal of sampling error variance. In the present analysis, sampling error variance accounted for 81.6% of the overall variance in posttreatment SOL effect sizes. However, given the large variance of effect sizes and the limitations of the sampling error formula when applied to small sample sizes (Hedges & Olkin, 1985), some liberty was taken with the aforementioned decision rule and potential moderator variables were investigated using regression analyses.

Results

A total of 381 effect sizes were calculated on the combined population of 1,538 experimental and 369 no-treatment control participants. The mean age of participants was 41.65 years (SD = 12.65), with a reported range of 17 to 79 years. Treatment groups predominantly consisted of female participants (61.54%) who were solicited volunteers (84.52%) from the general community (84.33%) with a mean duration of chronic insomnia of 11.14 years (SD = 4.01), mean SOL of 61.41 min (SD = 31.38), mean total sleep time of 5.65 hr (SD = 1.12), and 1.63 awakenings per night (SD = 1.94).

Treatments were initially compared by calculating the average effect size for each approach. The sample-size-weighted mean effect size ($\bar{d}$), the number of effect sizes (#d), improvement expected for an average complaint of insomnia, and the 95% confidence interval around $\bar{d}$ are presented for each treatment category in Table 1. Note, however, that comparison of treatments based on these uncontrolled average effect sizes may be a hazardous procedure, particularly when there are small numbers of d values. The mean SOL effect size at posttreatment was .87, representing a reduction of 24 min (39.5%) from pretreatment levels for the hypothetical average insomniac. At follow-up, improvements in SOL were further enhanced ($\bar{d}$ = 1.01). This trend was largely due to the enhancement of relax-

[1] The different d formula used for groups from studies without a control group may have led to distorted results, especially if the variance of sleep measures was reduced after treatment. To evaluate this possibility, we repeated d calculations, excluding groups from studies that did not use a control group. No significant differences were found, and therefore data from all studies were combined.

Table 1
Results for SOL, TST, #A, and SQR as a Function of Treatment Approach

Treatment	$\bar{d}$	#*d*	Improvement	95% CI
SOL at posttreatment				
Overall	0.87	116	24	0.58–1.16
Progressive muscle relaxation	0.81	36	22	0.81–0.81
Other relaxation	0.93	13	26	−0.19–2.05
Stimulus control	1.16	20	32	0.50–1.82
Paradoxical intention	0.73	12	20	0.11–1.35
Sleep restriction	0.85	4	24	0.85–0.85
Combinations	1.00	18	28	0.24–1.76
Placebo	0.46	13	13	0.46–0.46
Relaxation	0.84	49	23	0.84–0.84
Nonrelaxation	0.97	36	27	0.50–1.44
SOL at follow-up				
Overall	1.01	53	28	1.01–1.01
Progressive muscle relaxation	0.97	18	27	0.97–0.97
Other relaxation	2.04	7	57	2.04–2.04
Stimulus control	1.14	8	32	1.14–1.14
Paradoxical intention	0.91	2	25	0.91–0.91
Sleep restriction	0.57	2	16	0.57–0.57
Combinations	0.96	9	27	0.61–1.31
Placebo	0.43	7	12	0.43–0.43
Relaxation	1.25	25	35	1.25–1.25
Nonrelaxation	0.95	12	26	0.95–0.95
TST				
Posttreatment overall	0.49	60	32	0.49–0.49
Progressive muscle relaxation	0.52	16	34	−1.04–2.08
Other relaxation	0.57	5	37	0.57–0.57
Stimulus control	0.38	6	25	−1.10–1.86
Paradoxical intention	0.10	6	7	−0.74–0.94
Sleep restriction	0.37	4	24	−0.51–1.25
Combinations	0.78	18	51	−1.89–3.45
Placebo	0.10	5	7	0.10–0.10
Relaxation	0.53	24	35	−0.70–1.76
Nonrelaxation	0.28	16	18	−0.83–1.39
Follow-up	0.54	19	37	0.54–0.54
No. of nocturnal awakenings				
Posttreatment overall	0.63	55	1.2	0.63–0.63
Progressive muscle relaxation	0.57	15	1.1	0.57–0.57
Other relaxation	0.37	5	0.7	0.37–0.37
Stimulus control	0.61	12	1.2	0.61–0.61
Paradoxical intention	1.00	6	1.9	1.00–1.00
Combinations	0.84	10	1.6	−0.86–2.54
Placebo	0.41	7	0.8	0.41–0.41
Relaxation	0.52	20	1.0	0.52–0.52
Nonrelaxation	0.73	18	1.4	0.73–0.73
Follow-up	0.78	16	1.5	−0.08–1.64
Sleep quality ratings				
Posttreatment overall	0.94	53	—	0.28–1.60
Progressive muscle relaxation	0.97	15	—	0.97–0.97
Other relaxation	1.08	1	—	1.08–1.08
Stimulus control	1.30	6	—	1.30–1.30
Paradoxical intention	0.77	8	—	−0.79–2.33
Combinations	1.12	17	—	1.12–1.12
Placebo	0.21	6	—	−0.08–0.50
Relaxation	0.98	16	—	0.98–0.98
Nonrelaxation	1.00	14	—	−0.17–2.17
Follow-up	1.30	9	—	1.30–1.30

Note. SOL = sleep onset latency; TST = total sleep time; #A = number of nocturnal awakenings; SQR = subjective evaluation of sleep quality; CI = confidence interval; $\bar{d}$ = sample-size-weighted mean effect size; #*d* = number of effect sizes.

ation treatment effect sizes, whereas nonrelaxation treatments essentially maintained posttreatment gains. Over posttreatment and follow-up, stimulus control appeared to be the most effective therapy ($\bar{d}$ = 1.16 and $\bar{d}$ = 1.14, respectively), although the miscellaneous grouping of relaxation treatments clearly produced the largest effect size at follow-up ($\bar{d}$ = 2.04).

Treatment gains for TST, when interpreted in terms of sleep parameters, were similar to those reported for SOL. Although

the mean TST effect size was comparatively small ($\bar{d}$ = .49), it nonetheless represented an increase of 32 min (9.4%) in total sleep time from pretreatment levels. Combination treatments produced clearly the largest mean effect size ($\bar{d}$ = .78), whereas nonrelaxation therapies—in particular, paradoxical intention ($\bar{d}$ = .10)—tended to have smaller effect sizes. There appeared to be substantial variation in effect sizes; with the exception of "other relaxation" approaches, all 95% confidence intervals overlapped with both the placebo group confidence interval and zero. At follow-up, treatment effects essentially appeared to be maintained ($\bar{d}$ = .54), however, only 19 effect sizes were able to be calculated.

The overall mean #A effect size at posttreatment was .63, representing a reduction of 1.2 awakenings per night (73%) from pretreatment levels. Paradoxical intention ($\bar{d}$ = 1.00) and combined treatments ($\bar{d}$ = .84) were associated with the largest reductions in the number of nightly awakenings. "Other relaxation" approaches were particularly ineffective, with an average effect size smaller than that of placebo groups ($\bar{d}$ = .37 vs. $\bar{d}$ = .41). At follow-up, treatment effects appeared to be enhanced ($\bar{d}$ = .78), however, only 16 effect sizes were able to be calculated.

The mean SQR effect size at posttreatment was .94. Stimulus control ($\bar{d}$ = 1.30) appeared to be a particularly effective treatment approach. At follow-up, treatment effects appeared to be considerably enhanced ($\bar{d}$ = 1.30), however, only nine effect sizes were able to be calculated.

SOL effect sizes at posttreatment were investigated through regression analyses to (a) detect the presence of moderator variables and (b) determine whether treatments differed in efficacy after moderator variables had been statistically controlled. SOL effect sizes at posttreatment were transformed to give a normal distribution by taking the square root of sample-size-weighted effect sizes. Treatment methods and potential moderating variables (where necessary) were dummy coded. To avoid the problems caused by missing data, we used measures of central tendency and regression analyses to estimate missing values for the variables sex (for 22.3% of the treatment groups), age (15.1%), duration of insomnia (35.3%), severity of insomnia (16.5%), and total minutes of treatment (25.2%).

We performed a stepwise regression with the aim of identifying the potential moderating variables that were significant predictors of effect size. The following were found to be significant predictors at the .05 level: source of the participants (β = .39, where solicited volunteers = 0 and clinically referred patients = 1); hypnotic drug use (β = .25, where drug users = 0 and nondrug users = 1); year of the study (β = .21, measured as years since 1972); and home practice (β = −.19, where no practice = 0 and practice = 1). R for the regression after the final step was significantly different from zero, $F(4, 100)$ = 9.97, $p < .0001$, and in combination, the four variables predicted 28.5% (25.6% adjusted) of the variance.

Hierarchical regression was used to determine whether the treatment methods predicted effect sizes after controlling for differences in patient characteristics, treatment setting, and methodological features. Only the potential moderating variables identified as being significant through the stepwise regression were entered (Step 1). Excluding the nonsignificant variables maximized the ratio of cases to independent variables and, therefore, protected the value of adjusted R^2. Table 2 displays the unstandardized regression coefficients (B) and intercept, the standardized regression coefficients (β) and their corresponding p values, R, R^2, and adjusted R^2 after entry of the independent variables (Step 2). Addition of the six treatment method variables to the equation resulted in a nonsignificant increase in R^2, $F_{inc}(6, 94) = 1.76, p > .05$; R for regression was significantly different from zero, $F(10, 94) = 5.22, p < .0001$, and R^2 = 35.7% (28.9% adjusted). The source of the participants, nonuse of drugs, and placebo treatment contributed significantly to the prediction of the dependent variable.

Table 2
Hierarchical Regression of Potential Moderating and Treatment Variables on Sleep Onset Latency Effect Sizes at Posttreatment

Variable	B	β	p
Nonuse of drugs	0.77*	0.29	.00
Home practice	−0.13	−0.06	.70
Source of the participants	1.20*	0.35	.00
Year	0.03	0.17	.09
Other relaxation	0.24	0.07	.45
Sleep restriction	0.78	0.10	.31
Placebo	−0.80*	−0.24	.01
Paradoxical intention	0.18	0.05	.69
Combination	−0.02	−0.01	.96
Stimulus control	0.33	0.11	.43
Intercept	2.43		

Note. $R^2 = .36, p < .05$. Adjusted $R^2 = .29, p < .05$. $R = .59, p < .05$.
* $p < .05$.

Discussion

The meta-analysis of the research literature indicated that psychological treatments of insomnia produce considerable enhancement of sleep patterns and the subjective experience of sleep. The majority of effect sizes exceeded .80, which has been defined as a "large effect" (Cohen, 1977). Average treatment effects may be expected to reduce sleep onset latency from 61 to 37 min, increase total sleep time from 5.65 to 6.18 hr, and decrease the number of nightly awakenings from 1.63 to .44 for the hypothetical average case of insomnia. Furthermore, the majority of active treatment effect sizes were considerably greater than corresponding placebo effect sizes, indicating that improvements were only partially mediated by expectation of therapeutic gain, demand characteristics, or other nonspecific influences.

At follow-up, effects generally appeared to be maintained and, in most cases, slightly enhanced. However, given that there was greater than 50% attrition from posttreatment to follow-up in the number of effect sizes for each dependent variable, a reporting bias possibly contributed to this finding.

In general, this pattern of results converges with those of other quantitative reviews of the literature (Lacks & Morin, 1992; Lichstein & Fischer, 1985; Morin et al., 1989; Morin et al., in press).

Sleep Onset Latency at Posttreatment

When the influence of potential moderator variables was statistically controlled, placebo was the only treatment method

that was a significant predictor of outcome. This result indicated that, although all active treatments were superior to placebo, they did not differ significantly among each other in efficacy. The apparent superiority of stimulus control (as demonstrated by average effect sizes in Table 1) appears to have been largely a function of its association with other variables.

This finding is in disagreement with most previous reviews (e.g., Borkovec, 1982; Lacks & Morin, 1992; Morin et al., in press). These reviews have often, however, been based on the use of uncontrolled averages for comparing treatments and, therefore, have failed to control for moderating influences. Furthermore, the differences that have been demonstrated have often been marginal. For example, Lacks and Morin concluded that stimulus control, which produced average improvements on SOL of 58%, was the superior approach. Their integration showed, however, that 7 of the 10 remaining treatment modalities led to improvements of greater than 50%. It is reasonable to believe that these differences could be attributable to moderator variables. Alternatively, the results may be a function of the studies that have been integrated. A final consideration is that the present analysis may have been limited in its power to detect the superiority of stimulus control. Nonetheless, this also suggests that any differences were not great. In summary, the results appear to offer support for the judgments of Lichstein and Fischer (1985) who concluded that, although stimulus control may have a slight advantage, there were no consistent differences between treatments.

Combination approaches, which some researchers have assumed to be superior, did not produce larger effect sizes than either stimulus control or relaxation alone. This result is in agreement with previous reviews (Lacks & Morin, 1992; Lichstein & Fischer, 1985).

Patients who were referred for treatment made greater treatment gains than solicited volunteers. One explanation for this may be that referred insomniacs had greater compliance with treatment instructions. Referred patients were more likely to have been treated by a qualified therapist in a clinical setting (r = .62) and, therefore, may have felt a greater obligation to follow directions. Furthermore, they suffered from more severe symptoms (r = .20) and, consequently, may have been more motivated to make treatment gains, as demonstrated by their help-seeking behavior. Compliance may be the most crucial factor in the success of psychological treatments for insomnia (Chambers, 1992), given that these treatments often involve instructions that are difficult to follow (Sloan & Shapiro, 1993) and require considerable investment of time and effort (Glovinsky & Spielman, 1991).

Insomniacs who were regular users of sleep medications benefited less from treatment. Given that both Lacks and Morin (1992) and Morin et al. (in press) have reached the same conclusion, there now appears to be a fair degree of consensus on this issue. It was possible that the drug users, because of their psychological reliance on drugs, had low self-efficacy for falling asleep naturally. This low self-efficacy may have reduced expectancy of improvement and consequently undermined treatment. It was also noteworthy that insomniacs who were regularly using drugs when recruited but who were required to withdraw from use before treatment began (typically 4 weeks before treatment) did not obtain better results than those allowed to continue drug use. This suggests that although withdrawal patients were no longer as dependent on medication as those continuing use, their self-efficacy for falling asleep without pharmacological assistance was still impaired. Alternatively, the effects of rebound insomnia, which may last for several months after withdrawal (Kales & Berger, 1970), may have masked treatment gains. Given that insomniacs normally suffer severe insomnia after withdrawal, the present results may actually represent a substantial treatment effect.

These results hold several implications for the clinician. Therapists working in a clinical setting may reasonably expect greater improvements than those generally reported in the research literature, which have been largely based on solicited volunteers. Moreover, their ability to present treatments in a credible manner that engenders patient belief, expectancy, and compliance may be crucial to therapeutic success. Treatment effects for drug users may be maximized by allowing sufficient time after withdrawal for the effects of rebound insomnia to pass or self-efficacy for falling asleep naturally to be enhanced. However, the therapist should also consider that many people find it difficult to cease drug use (Sanavio, Vidotto, Bettinardi, Rolletto, & Zorzi, 1990) and that providing people with techniques to combat rebound insomnia may be crucial in helping them withdraw.

No other patient, treatment, or design variables were found to be independently predictive of treatment outcome. This suggests that individual therapy involving extensive investment of therapist and patient time (including home practice) may not be necessary for substantial improvements to be achieved. More efficient self-help and group treatments appear to be equally effective. The results also suggest that, although sleep patterns may be more disturbed in older adults, these adults are capable of obtaining the same treatment gains available to younger insomniacs. Given the dangers associated with pharmacological treatments for older adults (Reynolds, Kupfer, Hoch, & Sewitch, 1985), this may be a particularly important finding. It was also encouraging to find that none of the design variables influenced effect size estimates. The decision to include all studies, regardless of methodological standard, therefore appears to have been justified. Finally, it appears unlikely that a publication bias seriously distorted the findings.

These null results must, however, be regarded with added caution, given the limited power of the analyses. Furthermore, given that other quantitative and qualitative reviews have not been able to reach consensus on the impact of variables such as treatment modality, therapist experience, or the source of subjects (Espie, 1991; Lacks & Morin, 1992; Lacks & Powlishta, 1989; Morin et al., in press), it appears that further research is required before definite conclusions can be made.

Sleep Onset Latency at Follow-Up

Effect sizes for SOL at follow-up provided some support for Espie's (1991) hypothesis that the full effects of relaxation treatments, in comparison with nonrelaxation approaches, are larger in magnitude but require more time to become evident. This may be an important consideration in selecting an appropriate treatment strategy.

Total Sleep Time

Relaxation appears to be important to the lengthening of sleep. Relaxation and combination approaches (which usually

incorporated relaxation) demonstrated a superiority over alternative treatments. However, there was a large amount of variation associated with TST effect sizes, and the results should, therefore, be regarded as tentative.

Number of Awakenings

In contrast to the results for TST, nonrelaxation approaches appeared to be superior in reducing the number of nightly awakenings. This was due largely to the contribution of paradoxical intention, which had the largest effect size of any separate approach.

Sleep Quality Ratings

The largest effect sizes were found for SQR, suggesting that treatments were particularly effective at enhancing the experience of sleep. This result represents a strong endorsement of the treatments, given that enhancement of the sleep experience may help offset the psychological distress often caused by insomnia (Chambers, 1992). No evidence was found in support of Espie et al.'s (1989) hypothesis that relaxation treatments may be particularly effective at enhancing ratings of sleep quality.

Overall Efficacy

When all four dependent variable constructs were considered, combination approaches and stimulus control appeared to offer the largest improvements in general. Given that over three fourths of the combination approaches involved the use of stimulus control, this suggests that stimulus control may be the element responsible for superior treatment outcomes.

With the exception of reducing awakenings, the treatment gains reported for paradoxical intention generally did not match the effects of the other active treatments. Ascher and Turner (1980) reported that clients often mobilize a great deal of resistance against executing paradoxical instructions that are clearly counterintuitive and diametrically opposed to their true goals. Therefore, lack of compliance may be one explanation for the relatively poor gains produced by paradoxical intention. Further research may identify approaches through which the basic therapeutic elements can be delivered while simultaneously engendering the cooperation of the patient.

Effect sizes for sleep restriction therapy should be regarded with added caution, given that they were based on only four groups and that we were unable to calculate the effects on #A and SQR. Further research is required before the efficacy of sleep restriction therapy can be accurately quantified.

Clinical Significance

It has been suggested that the proportion of patients judged to have achieved "meaningful clinical improvement" may be a useful indicator of clinical efficacy (Kazdin, 1980). Coursey, Frankel, Gaardner, and Mott (1980) proposed that the reduction of SOL in daily logs by at least 33% from baseline levels and to less than 35 min absolutely can be used as one criterion for meaningful clinical improvement. In the present study, the hypothetical average insomniac would be expected to reduce SOL from 61 to 37 min (a 40% decrease) under average treatment effects. However, this average is based on all the treatment methods, including placebo. Therefore, if the participant received active treatment, this criterion would be met. Furthermore, assuming that effect size distributions were normal and that pretreatment SOL was not related to effect size (as indicated by the regression analyses), then approximately 50% of the treated insomniacs would be expected to meet the criterion. Other reviews have similarly concluded that treatments generally reduce symptoms to below or near the clinical criteria for a diagnosis of insomnia (Lacks & Powlishta, 1989; Morin et al., in press). This provides encouraging evidence supporting the clinical efficacy of the treatments.

Limitations and Future Research Directions

Several researchers have argued that there is a need to tailor treatments to match the etiology of each presenting case and the needs and preferences of the client (Hauri, 1991; Lacks & Morin, 1992; Morin & Kwentus, 1988). Successful treatment may depend on understanding and addressing each patient's special mix of contributing causes. Although we had hoped to identify guidelines for determining the appropriate treatment strategy for a given case of insomnia, because of the small numbers of effect sizes it was not possible to make comparisons within patient subgroups. Although analyses suggested that the treatments did not differ significantly in efficacy, there may have been differences for specific patient groups (e.g., older adults). Further research into this issue is required. For accurate reflection of the maximum therapeutic outcomes available, research should be based on tailored treatments (Sloan & Shapiro, 1993).

Although only three significant predictors of treatment outcome were identified, it was possible that other predictor variables were not detected. The ratio of cases to independent variables was only half of that recommended for standard and hierarchical multiple regression, and the number of cases only barely exceeded the criterion of 100 cases, below which the power would have been unacceptably low, regardless of the number of independent variables (Tabachnick & Fidell, 1989).

The meta-analysis provided a thorough description and critique of the methodology used in insomnia therapy research. Several problematic aspects were identified.

Long-term treatment effects were difficult to determine. Researchers frequently neglected to take follow-up measures, and existing follow-ups were taken, on average, only 8 months after treatment. However, one study, in which follow-up measures were taken 3 years after treatment, found effects to be substantially enhanced from posttreatment (Sanavio et al., 1990). If demonstrated, long-term efficacy would represent a distinct advantage for psychological treatments over pharmacological approaches.

There were several limitations to the generalizability of the results. The meta-analysis excluded insomniacs with abnormal background characteristics. This approach, which mirrors that of most empirical research, may be criticized for its poor correspondence with clinical reality. It appears that the development of treatments for people with psychological disturbances, medical complaints, or other irregularities is in demand and may be an area worthy of further research (Lacks & Morin, 1992).

Although researchers have reported that compliance and attrition are problems for behavioral treatments of insomnia

(Hauri, 1979), attrition rates were only occasionally provided. The development of more flexible, user-friendly treatments may be a profitable area for future research to consider (Edinger, Hoelscher, Marsh, Lipper, & Ionescu-Pioggia, 1992), but only the consistent reporting of attrition rates will enable these factors to be considered.

Future research may benefit from greater consideration of clinical significance on a wider range of dependent variables. Demonstrating the capacity of treatments to meaningfully enhance daytime functioning, improve mood and health, help insomniacs withdraw from regular use of medications, and reduce the distress associated with chronic insomnia may represent a strong endorsement.

Although it is generally recognized that sleep diaries do not accurately measure sleep parameters, they appear to be reliable and, therefore, provide accurate estimations of changes in sleep patterns (Espie, 1991). Furthermore, the perception of change, regardless of actual effects, may be important to the insomniac. Nonetheless, objective physiological evidence demonstrating improvement in sleep parameters may enhance the credibility of insomnia therapy research. Mechanical devices, such as the Sleep Assessment Device (Kelley & Lichstein, 1980), may offer a useful compromise to the problem of obtaining accurate but unobtrusive objective measurement in the normal sleep setting.

Conclusion

The meta-analysis indicated that psychological interventions produce reliable and durable clinical benefits in the treatment of insomnia. It appears that active treatments are superior to placebo therapies but that they do not differ greatly among each other in efficacy. Stimulus control may have a slight advantage over other approaches. Treatment gains for SOL were greater for clinically referred patients and for insomniacs who were not regular users of sleep medications. Empirical research is needed to evaluate treatment efficacy on alternative indices of insomnia and to identify the conditions under which outcomes are optimal. Given the iatrogenic consequences of drug therapies and the gains that psychological approaches may offer in terms of quality of life and long-term physical and mental health, it is trusted that patients and clinicians alike will be able to look beyond quick and easy pharmacological options for the treatment of insomnia.

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Appendix

Seventeen Potential Moderating Variables for Treatment Effect Size

- Age
- Sex (% male)
- Duration of insomnia
- Severity of insomnia (mean sleep onset latency [SOL])
- Occupation
  - Students
  - General community
- Source
  - Solicited volunteers
  - Clinically referred
- Hypnotic drug use
  - Nonusers
  - Previous withdrawal
  - Drug use permitted
- Mode of treatment
  - Individual
  - Group
- Therapist experience
  - Student
  - Practicing therapist
  - Self-help tape
- Home practice used
- Amount of treatment
  - No. of weeks
  - Total no. of minutes
- Form
  - Published
  - Unpublished
- Year of publication or presentation
- Study Design
  - One group
  - Multiple treatment groups, random assignment
  - Multiple treatment groups, matching on SOL
  - No treatment control group, random assignment
  - No treatment control group, matching on SOL
- Use of objective validation
- Assessment of expectancy
- Comprehensive reporting of results

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