Nonpharmacologic Treatment of Chronic Insomnia

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Abstract: This paper reviews the evidence regarding the efficacy of nonpharmacological treatments for primary chronic insomnia. It is based on a review of 48 clinical trials and two meta-analyses conducted by a task force appointed by the American Academy of Sleep Medicine to develop practice parameters on non-drug therapies for the clinical management of insomnia. The findings indicate that nonpharmacological therapies produce reliable and durable changes in several sleep parameters of chronic insomnia sufferers. The data indicate that between 70% and 80% of patients treated with nonpharmacological interventions benefit from treatment. For the typical patient with persistent primary insomnia, treatment is likely to reduce the main target symptoms of sleep onset latency and/or wake time after sleep onset below or near the 30-min criterion initially used to define insomnia severity. Sleep duration is also increased by a modest 30 minutes and sleep quality and patient's satisfaction with sleep patterns are significantly enhanced. Sleep improvements achieved with these behavioral interventions are sustained for at least 6 months after treatment completion. However, there is no clear evidence that improved sleep leads to meaningful changes in daytime well-being or performance. Three treatments meet the American Psychological Association (APA) criteria for empirically-supported psychological treatments for insomnia: Stimulus control, progressive muscle relaxation, and paradoxical intention; and three additional treatments meet APA criteria for probably efficacious treatments: Sleep restriction, biofeedback, and multifaceted cognitive-behavior therapy. Additional outcome research is needed to examine the effectiveness of treatment when it is implemented in clinical settings (primary care, family practice), by non-sleep specialists, and with insomnia patients presenting medical or psychiatric comorbidity.
Key Words: Insomnia; Treatment; Non-pharmacological treatment; Behavioral treatment

1. INTRODUCTION

INSOMNIA IS A PREVALENT CONDITION AFFECTING ONE THIRD OF THE ADULT POPULATION OCCASIONALLY AND BETWEEN 9% AND 12% ON A CHRONIC BASIS.[1-3] Insomnia is more frequent among women, older adults, shift workers, and patients with medical and psychiatric disorders. Chronic difficulties initiating and maintaining sleep are often associated with psychosocial and occupational impairments such as daytime fatigue, mood disturbances, performance impairments, and reduced quality of life.[1,3] Significant healthcare costs may occur from prescription and over-the-counter sleep medications, as well as with visits to health-care providers.[4,5,101]

Although insomnia often remains untreated, the first line of treatment is usually self-initiated with over-the-counter sleep aids and alcohol. When professional treatment is sought, usually from a primary care physician, pharmacotherapy is the most widely used and often the only recommended treatment. Two consensus conferences, sponsored by the National Institutes of Health,[6,7] have concluded that short-term usage of hypnotic medications may be useful for acute and situational insomnia, although long-term use remains controversial because of the potential risk of tolerance and dependency.

Increasing recognition of the mediating role of psychological and behavioral factors in insomnia has led to the development and evaluation of several nonpharmacological interventions for its clinical management.[8-10] Despite repeated calls for their integration with more traditional biomedical interventions,[6,7,11] these treatment methods are not well known by health-care practitioners[12] and remain under-utilized in clinical practice. Before reviewing the efficacy of nonpharmacological treatment methods for insomnia, a brief overview of definition and diagnostic issues is presented.

1.1 Definition

Insomnia is a heterogeneous complaint reflecting reduced quality, duration, or efficiency of sleep. The subjective complaint may or may not be corroborated by objective evidence from polysomnography or observations by others. Insomnia may involve trouble falling asleep,

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problems staying asleep, such as frequent or prolonged nocturnal awakenings, or early morning awakening with an inability to resume sleep. The complaint may also be of nonrestorative sleep or diminished sleep quality, resulting in a feeling of being unrefreshed in the morning and low energy during the daytime. Difficulties initiating and maintaining sleep are not mutually exclusive, and the pattern of insomnia may also shift over time.[13]

The severity of insomnia is judged along several dimensions including frequency, intensity, and duration of sleep difficulties, as well as their impact on daytime functioning, mood, and quality of life.[14,15] In treatment outcome studies, insomnia is often defined by a sleep-onset latency and/or wake after sleep onset that is greater than 30 minutes, with a corresponding sleep efficiency (ratio of time asleep to time spent in bed) lower than 85%; the sleep disturbance must be present three or more nights per week.[16] The 30-minute criterion is rather arbitrary and has been chosen primarily for historical reasons. The duration the insomnia has been present is another important index to determine potential causes and the most appropriate treatment. Transient insomnia is defined in terms of days (i.e., < 1 week), and is usually associated with acute psychological or medical stress, jet lag, or environmental factors. Short-term or sub-acute insomnia lasts between one and four weeks, and chronic insomnia refers to a complaint lasting more than one month.[14,17]

1.2 Diagnostic considerations

The diagnostic criteria of primary insomnia are: (a) difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month; (b) the sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning; and (c) the sleep disturbance does not occur exclusively during the course of another mental or sleep disorder, and is not due to the direct physiological effects of a substance or a general medical condition.[14] Several subtypes of primary insomnia (psychophysiological, idiopathic, sleep state misperception) have been proposed in the ICSD nosology, although this level of subtyping may be premature in the absence of adequate empirical supporting evidence.[18] Because insomnia can be a symptom of several other conditions, the diagnosis of primary insomnia (i.e., the syndrome) is often made by exclusion.

In addition to its primary subtypes, insomnia can also be associated with psychiatric, alcohol or drug abuse, medical, or other sleep disorders. Insomnia comorbidity is particularly high with psychiatric disorders. Between 35% and 44% of all patients presenting to sleep specialists with a complaint of insomnia suffer from a concomitant psychiatric disorder, most frequently affective and anxiety disorders.[19] Numerous medical conditions can also cause insomnia, either because of the underlying pathophysiology (e.g., fibromyalgia, congestive-heart failures) or secondarily to the treatment for the medical condition (e.g., bronchodilators, steroids). Insomnia may also be the presenting complaint when it is caused by an underlying sleep disorder such as restless legs syndrome/periodic limb movements, or sleep apnea. In such instances, excessive daytime sleepiness is a more typical subjective complaint than insomnia. Prolonged usage of hypnotic medications can also exacerbate insomnia (hypnotic-dependent insomnia).

Whatever the initial causes of insomnia, behavioral and conditioning factors often act as mediating variables.[20] The temporal course of insomnia may be conceptualized as follows: (a) individuals with chronic insomnia have specific predisposing factors; (b) the onset of insomnia can be related to a number of precipitating factors; (c) chronic insomnia is maintained by a set of perpetuating factors. Predisposing factors remain constant throughout the course of insomnia. By contrast, precipitating factors appear at the onset of the insomnia problem, and in most cases diminish over time. Perpetuating factors (such as conditioned arousal or habituation to hypnotics) tend to become more prominent and play a much more important role over time.[20] Accordingly, most behavioral interventions will focus on altering those conditions that perpetuate chronic insomnia, whereas problem-solving and supportive therapies may also be needed to address some of the triggering factors (e.g. occupational stress).

2. PURPOSE

The objective of this paper is to review the empirical evidence regarding the short- and long-term efficacy, and practical advantages and limitations, of nonpharmacological interventions for the clinical management of insomnia.

3. METHODS

3.1 Identification and Selection of Treatment Studies

Treatment studies selected for review in this paper were identified through PsycLIT and MEDLINE searches (1970-1997) using the following key words: Insomnia, nonpharmacological-nondrug, behavior-cognitive-psychological, treatment-therapy-intervention-management. In addition, bibliographies of meta-analyses[21,22] or other literature reviews[8,16,23,24] and references cited in empirical studies themselves were also systematically reviewed. The criteria for inclusion of a study were as follows: (a) the main sleep diagnosis was insomnia, (b) one of the treatment conditions was nonpharmacological, (c) the dependent measures included one or more of the following variables: Sleep onset latency, number and/or duration of awakenings, total sleep time, or sleep quality, and (d) the study design was a group design with a control/comparison con-

dition or a clinical case series evaluating a well-defined treatment modality with a minimum of 10 clinical patients. Case reports and single-subject design studies were excluded, as were studies whose sample was composed predominantly of college students. These latter studies may be referenced in the text but they are not listed in the evidence table. The initial search yielded approximately 100 treatment studies, but more than half were excluded because they did not meet inclusion criteria. The main reasons for exclusion were that treatment was exclusively pharmacological, the study included less than 10 patients, or the sample was composed predominantly of college students recruited on a university campus. The present paper is based on the evidence from 48 individual studies (n > 2,000 patients) that met inclusion criteria; those studies are listed in Table 1. In addition, findings from two meta-analyses (see Tables 2 and 3), which were themselves quantitative reviews of individual treatment studies, are used in estimating treatment efficacy and improvement rates for the different treatment modalities.

The large majority of clinical studies selected for this review have relied on prospective daily sleep diaries to document treatment outcome. Participants are typically required to complete a daily sleep log for a minimum of a two-week baseline period, for the duration of treatment, and for an additional one or two-week period at posttreatment and follow-up. A few studies have also included polysomnography and behavioral assessment devices (e.g., actigraphy) to validate subjective reports from daily sleep diaries. Those studies are identified in Table 1 and in the appropriate subsections of the results.

3.2 Treatment Procedures: Description, rationale, and targets

Nonpharmacological interventions for insomnia consist primarily of short-term cognitive-behavioral therapies. These methods focus primarily on factors that are presumed to perpetuate insomnia; as such, they seek to modify maladaptive sleep habits, reduce autonomic and cognitive arousal, alter dysfunctional beliefs and attitudes about sleep, and educate patients about healthier sleep practices. Although more than a dozen treatment methods have been used in the management of insomnia, only those that have received adequate empirical evaluation (at least 2 controlled studies) are described in this section. The reader is referred to other sources for a more detailed description of these and other behavioral treatments for insomnia [8-10].

3.2.1 Stimulus control therapy

Stimulus control therapy[25] is based on the premise that insomnia is a conditioned response to temporal (bedtime) and environmental (bed/bedroom) cues that are usually associated with sleep. Accordingly, the main objective of stimulus control therapy is to train the insomnia patient to reassociate the bed and bedroom with rapid sleep onset by curtailing sleep-incompatible activities (overt and covert) that serve as cues for staying awake and by enforcing a consistent sleep-wake schedule. Stimulus control therapy consists of the following instructional procedures: (a) go to bed only when sleepy; (b) use the bed and bedroom only for sleep and sex; (c) get out of bed and go into another room whenever unable to fall asleep or return to sleep within 15-20 minutes, and return to bed only when sleepy again; (d) maintain a regular arising time in the morning regardless of sleep duration the previous nights, and (e) avoid daytime napping.

3.2.2 Sleep restriction

Sleep restriction therapy[26] consists of curtailing the amount of time spent in bed to more nearly match the subjective amount of time asleep. For example, if a person reports sleeping an average of 5 hours per night out of 8 hours spent in bed, the initial prescribed sleep window (i.e., from initial bedtime to final arising time) would be 5 hours. Subsequently, the allowable time in bed is increased by 15-20 minutes for a given week when sleep efficiency (ratio of total sleep/time in bed X 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 (usually on a weekly basis) until an optimal sleep duration is achieved. Sleep restriction creates a mild state of sleep deprivation and promotes a more rapid sleep onset, more efficient sleep, and less internight variability. To prevent excessive daytime sleepiness, time in bed should not be less than 5 hours per night.

3.2.3 Relaxation therapies

Relaxation-based interventions are predicated on the observation that insomnia patients often display high levels of arousal (physiological and cognitive), both at night and during daytime. Relaxation methods are used to deactivate the arousal system and selection of a specific technique varies depending on whether physiological or cognitive arousal is targeted for treatment. Progressive muscle relaxation (a method of tensing and relaxing different muscle groups throughout the body) and biofeedback (a visual or auditory feedback is provided to the patient to control some pre-determined physiological parameters) seek to reduce somatic arousal (e.g., muscle tension), whereas attention-focusing procedures such as imagery training (visualization technique to focus on some pleasant or neutral images) and thought stopping are intended to lower presleep cognitive arousal (e.g., intrusive thoughts, racing mind). Additional relaxation therapies (e.g., abdominal breathing, meditation, hypnosis) have been advocated, but there is currently no

evidence to support their use in the clinical management of insomnia. As for most self-management skills, all these relaxation techniques require regular practice over a period of several weeks, and professional guidance is often necessary in the initial stage of training (see Lichstein [27] for a detailed review of relaxation procedures).

3.2.4 Cognitive therapy

Cognitive therapy seeks to alter faulty beliefs and attitudes about sleep. For example, insomniacs often display a great deal of apprehension about bedtime and performance anxiety in their attempt to control the process of sleep onset; some even entertain catastrophic thinking about the potential consequences of insomnia, all of which may heighten their affective response to poor sleep. The objective of cognitive therapy is to short-circuit the vicious cycle of insomnia, emotional distress, dysfunctional cognitions, and further sleep disturbances. Examples of treatment targets for cognitive therapy include: (a) unrealistic sleep expectations (e.g., "I must get 8 hours of sleep every night"); (b) misconceptions about the causes of insomnia (e.g., "my insomnia is entirely due to a chemical imbalance"); (c) amplifications of its consequences (e.g., "I can accomplish nothing after a poor night's sleep"); and (d) performance anxiety resulting from excessive attempts at controlling the sleep process.[10] Cognitive therapy consists of identifying patient-specific dysfunctional sleep cognitions, challenging their validity, and replacing them with more adaptive substitutes through the use of restructuring techniques such as reattribution training, decatastrophizing, hypothesis testing, reappraisal, and attention shifting.[10]

3.2.5 Paradoxical intention

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. This procedure may be conceptualized as a form of cognitive restructuring technique to alleviate performance anxiety.

3.2.6 Sleep hygiene education

Sleep hygiene education[9] targets health practices (e.g., diet, exercise, substance use) and environmental factors (e.g., light, noise, temperature, and mattress) that may be either detrimental or beneficial to sleep. Although these factors are rarely severe enough to be the primary cause of chronic insomnia,[18] they may complicate an existing sleep problem and hinder treatment progress. Sleep hygiene recommendations may include discontinuation of caffeine and nicotine 4-6 hrs before bedtime, avoidance of alcohol as a sleep aid, and exercising 5-6 hours before bedtime but not closer than 3 hours; and minimizing noise, light, and excessive temperature during the sleep period with ear plugs, window blinds, or an electric blanket/air conditioner. Additional recommendations, which tend to overlap with stimulus control and sleep restriction, may also include curtailing daytime napping and time spent in bed. While poor sleepers are generally better informed about sleep hygiene, they also engaged in more unhealthy practices than good sleepers.[28] Thus, the objectives of sleep hygiene education are not only to heighten the patient's awareness of those factors, but to promote better sleep.

4 SUMMARY OF RESULTS

Table 1 summarizes the 48 individual studies selected for the present review based on the criteria outlined in the method section. Those studies have focused predominantly on patients with insomnia that is both chronic and primary, treatment outcome was typically measured with prospective daily sleep diaries, and the main dependent measures were sleep onset latency, number and duration of awakenings, total sleep time, and sleep quality. Tables 2 and 3 summarize the main findings from the two meta-analyses of the efficacy of nonpharmacological interventions for insomnia. Because of their slightly different selection criteria and periods covered, those two meta-analyses are based on most (but not necessarily all) of the same individual studies selected for the present paper and listed in Table 1. The following sections will summarize the magnitude of changes obtained on five sleep parameters, the clinical significance of those changes, the durability of sleep improvements over time, and the comparative efficacy of single and combined treatment procedures.

4.1 Magnitude of therapeutic changes

Table 2 summarizes the findings of two meta-analyses[21,22] of non-drug treatments for insomnia. These data (z and d scores) represent a composite index of treatment effects for all non-drug treatments combined together and for all studies grouped together. These meta-analyses have yielded virtually identical effect sizes (0.87 and 0.88) for sleep-onset latency, the main target symptom in studies of sleep-onset insomnia. An effect size of this magnitude indicates that, on average, insomnia patients are better off (fall asleep faster) after treatment than about 80% of untreated control subjects (*see footnote). Reliable effect

*Effect sizes are calculated by subtracting the mean of the control group from the mean of the treated group at post-treatment and dividing by the pooled standard deviations of the two groups. The result is expressed as a standardized z or d score, which can be interpreted as the distance, in standard deviation units, between the average treated patient 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. Assuming a normal distribution, effect sizes can also be transformed in percentile ranks. An effect size of 0.50 would indicate that approximately 60% of treated patients perform better (or sleep better) after treatment than untreated controls. 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.[29]

sizes, falling in what is conventionally defined as moderate to large, have also been reported for other sleep parameters: total sleep time (0.42-0.49), number of awakenings (0.53-0.63), duration of awakenings (0.65), and sleep quality ratings (0.94). When transformed into percentile ranks, these data indicate that treated insomnia patients sleep longer, awake less frequently and for shorter durations, and report higher sleep quality after treatment than 50%-70% of untreated control patients. While these data (effect sizes and percentile ranks) provide estimates of the proportion of treated patients improving to a greater extent than untreated control patients, they do not inform us about the magnitude of improvements.

In terms of absolute changes over time, the results from these two meta-analyses show that sleep-onset latency is reduced from an average of 60-65 min at baseline to about 35 min at posttreatment for all treatments combined, relative to an average reduction of only 8 minutes for control subjects. Although fewer studies have specifically targeted sleep-maintenance insomnia, similar results are obtained for the duration of awakenings, which is reduced from an average of 70 min at baseline to about 38 min following treatment compared to a reduction from 67 to 57 minutes for control subjects. The number of reported awakenings, averaging less than two per night at baseline, is reduced to about one awakening per night at posttreatment. Total sleep time is increased by a modest 30 min, from 6 hours to 6.5 hours after treatment (relative to only four minutes for controls), but subjective sleep quality ratings are significantly enhanced with treatment. Thus, for the average insomnia patient, treatment effects may be expected to reduce the latency to sleep onset and the amount of time awake after sleep onset by about 50% each (i.e., 30 min.) and to increase total sleep time to about six and one half hours per night. Because those results are averaged across all treatment modalities, they represent a very conservative estimate of treatment efficacy.[21,22] The relative efficacy of different therapies is summarized below.

4.2 Source of outcome assessment

The majority (> 90%) of treatment studies have relied on daily sleep diaries to document outcome. Despite some limitations inherent to self-report assessment, patients' reports represent an essential source of data in assessing treatment efficacy because it is the subjective perception of poor sleep that prompts them in the first place to seek treatment. Also, daily self-monitoring of specific sleep parameters (e.g., sleep latency, number and duration of awakenings) over several weeks represents a more reliable index of insomnia than a single global and retrospective assessment, and may reflect more accurately, than one or two nights of polysomnography, on the typical night-to-night variability that characterize the sleep patterns of chronic insomniacs.

Nonetheless, about a dozen studies have added more objective assessment methods, including standard polysomnographic evaluations,[30-37] mechanical devices (e.g., actigraphy[38-42]), and collateral reports from spouses.[34,42] Results based on these assessment modalities have provided converging evidence supporting the efficacy of treatment. For example, in a study of sleep-onset insomnia,[33] baseline data for sleep onset latency were 77 min and 84 min for sleep diary and polysomnography respectively. At posttreatment, sleep latency had decreased to 19 min and 21 min, respectively, on both measurement methods. In another study of late-life insomnia,[34] baseline values for wake after sleep onset were 62 minutes and 73 minutes for diary and polysomnographic measures, respectively. At posttreatment, the values for that dependent measure had decreased to 29 minutes (diary) and 35 min (polysomnography), yielding improvement rates of 54% and 51%, respectively for the two measurement methods. These data indicate that non-drug interventions are effective not only in altering sleep perception but also in improving electrophysiological sleep. The magnitude of improvements recorded on polysomnographic measures may be smaller but in the same direction as that obtained on daily sleep diaries.

4.3 Clinical significance of changes

Aside from showing that treatment produces reliable and statistically significant sleep improvements, it is equally important to demonstrate that these changes are clinically meaningful - that is, do they bring patients within a "normal" sleep pattern and do they make a real difference in the patient's life. Although there are currently no agreed-upon standards upon which to base such evaluations, several indicators (a, b, or c as listed) have been used to estimate clinical significance: (a) the proportion of patients who reached a dual improvement, i.e., (1) a 50% reduction on the main target symptom (sleep onset latency or time awake after sleep onset) plus (2) an absolute value of that symptom falling near or below the 30-min criteria typically used to define insomnia, (b) the proportion of patients whose sleep efficiency moved from a dysfunctional to a normative level (i.e., > 80%-85%), and (c) a reduction of hypnotic usage.

Murtagh and Greenwood[22] estimated from their meta-analysis that about 50% of individuals treated for sleep onset insomnia with nonpharmacological interventions met criterion of meaningful clinical improvements. Lacks and Powlishta[43] reported that 39% of 216 participants in seven outcome studies showed reliable changes (i.e., exceeded chance expectation[44]) in their sleep, whereas 23% became "good sleepers" after treatment. At the one-year follow-up, 49% showed reliable change, 32% became good sleepers, 63% had at least a 50% decrease in complaints, and 31% reported they no longer had insomnia. In a clinical replication series (a series of patients treated with the same

intervention) of 100 patients treated at a sleep disorders clinic,[45] about one half of all patients achieved a 50% or better improvement rate, and between 37% and 40% reached a dual criterion of clinical improvement (i.e., a 50% reduction of their target symptom, with the absolute value of that symptom falling below 30 min). Of the total 100 patients, 63 had a sleep efficiency of 80% or more after treatment, compared to 25 patients at baseline for an absolute gain of 38 patients whose sleep efficiency approached or moved within a normative level. Another study using normal sleepers as control groups, showed that a multifactor intervention was effective in bringing the majority of sleep-onset insomniacs within normative range (i.e., sleep latency of 30 min. or less[33]).

Another useful index to judge the clinical significance of behavioral treatment for insomnia is whether a patient who initially used hypnotic medications has achieved any meaningful reductions on this measure. Lacks and Powlishta[43] reported that 76% of 216 treated insomnia subjects were medication-free at the 1-year follow-up, compared with 35% at baseline. In a case series of 100 patients,[45] 84% had used sleep aids at least once in the month preceding their initial interview, with 59 patients who were classified as habitual users (i.e., 3 times or more per week). At posttreatment, the number of hypnotic users dropped to 48, with 27 patients considered habitual users. Comparative data for three subgroups of patients showed that 22 psychophysiological (n = 31), 9 psychiatric (n = 22), and 9 drug-dependent insomniacs (n = 21) were drug-free at posttreatment relative to 9, 2, and 0, respectively, at the initial interview.

Little attention has been paid to the clinical impact of treatment on daytime variables such as performance, fatigue, mood, and quality of life. Two studies have reported reductions of depressive and anxious symptoms that parallelled sleep improvements.[33,38] Jacobs et al.[33] reported mean reductions of 11 on the Center for Epidemiological Study of Depression scale, and 7.3 (State) and 13.1 (Trait) on the Spielberger State-Trait Anxiety Inventory. Espie et al.[38] reported similar reductions of psychological symptoms with insomnia treatment. Additional research attention is needed to document changes in fatigue and in cognitive functioning (e.g., concentration, memory) since it is often daytime impairments in these areas that worry patients most and prompt them to seek treatment. At this time, however, there is no clear evidence that sleep improvements produced with behavioral interventions have any significant impact on daytime performance and psychological well-being.

4.4 Durability of sleep improvements

A very robust finding across behavioral treatment studies is that treatment-produced changes in sleep parameters are very well maintained at short- (3-month) and intermediate (6-month) range follow-ups.[21,22] For example, the average duration of follow-ups for the studies reviewed in the Morin et al. meta-analysis was six months.[21] Follow-up values for sleep onset latency and wake after sleep onset were 33 min and 38 min respectively, compared to 37 and 38 min at posttreatment. Thus, although behavioral treatment may require a few weeks to produce clinical benefits, these improvements are durable over time. Additional gains are often noted from posttreatment to follow-ups on measures of sleep latency and total sleep time. For example, in the Morin et al. meta-analysis, total sleep time was increased from 349 min at baseline to 378 min at posttreatment and to 396 min at follow-up.[21] Because behavioral treatments are typically implemented in the context of relatively brief periods of time (average of 6 treatment sessions), some patients may require more time to fully integrate the newly learned self-management clinical procedures. This may be particularly true for relaxation-based treatments.[8] Despite fairly robust long-term outcomes, follow-up data must be interpreted cautiously as there are relatively few studies reporting long-term (> 1 year) follow-ups (see Table 1) and, among those that do, attrition rates increase substantially over time.

4.5 Comparative efficacy of treatment modalities

Since its introduction by Bootzin in 1972, a total of 29 studies meeting inclusion criteria have evaluated the efficacy of **stimulus control therapy** for insomnia, either as a single treatment modality (12 studies) or in combination with other interventions (17 studies). A complete listing of those studies meeting inclusion criteria is presented in Table 1. All 12 studies evaluating stimulus control therapy as a single treatment modality demonstrated improved sleep by established criteria when compared to controls or to other single interventions.[38,42,46-55] None of the studies reviewed demonstrated negative results with stimulus control or treatment benefits that were inferior to control conditions. Stimulus control was shown to be superior to other treatment modalities such as progressive relaxation, imagery training, and paradoxical intention in five studies;[38,42,47,50,53] four other studies did not find that stimulus control was more effective than other treatments, although it was superior to controls.[46,49,54,55]

Treatment benefits produced by stimulus control therapy have been documented for both sleep onset and sleep maintenance insomnia. In meta-analyses combining data from multiple studies,[21,22] stimulus control was found to reduce the average self-reported sleep onset latency from 64 min at baseline to 33 min at posttreatment, and wake time after sleep onset from 84 min to 44 min at posttreatment. Four studies have also documented the effects of stimulus control with polysomnography,[30] mechanical devices,[40,42,56] and collateral reports from significant others.

Nine studies meeting inclusion criteria have used **sleep**

restriction in the treatment of insomnia,[26,33,34,45,57-61] but only two of those studies have evaluated the efficacy of this procedure as a single treatment modality.[26,57] In the original clinical case series by Spielman and colleagues,[26] sleep latency was reduced from an average of 48 min at baseline to 19 min at posttreatment, and time awake after sleep onset was reduced from 111 min to 31 min, with a corresponding increased in sleep efficiency from 67% at baseline to 87% at posttreatment. In another study with older insomniacs,[57] sleep efficiency was improved by 24% with sleep restriction compared to only 4% for progressive relaxation. A modified sleep restriction procedure has also been used in combination with a sleep education program with elderly insomniacs;[58] when self-administered via a video tape, this treatment combination was less effective (reduction of WASO from 92 min to 63 min) than when it was implemented with therapist guidance (68 to 37 min). In the remaining six studies,[33,34,45,59-61] sleep restriction was integrated into multifaceted interventions including stimulus control, relaxation training, and cognitive therapy. All six studies yielded significant improvements on various sleep parameters but, because of the multicomponent nature of those interventions, the specific contribution of sleep restriction was unknown.

Of the 48 studies meeting inclusion criteria, 37 evaluated at least one condition that involved some forms of relaxation-based interventions (i.e., progressive muscle relaxation, imagery training, meditation, biofeedback) (see Table 3). **Standard progressive muscle relaxation** (PMR) has been the most widely investigated treatment method for insomnia. It has been used as a single treatment method in 17 studies[37,38,46,47,54,55,57,62-71] and several more have combined PMR with other clinical treatments (see Table 3). With only one exception,[47] PMR has been shown superior to credible placebo, wait-list, and no-treatment controls. According to meta-analyses, PMR reduced self-reported sleep latency and wake after sleep onset by an average of 20-30 min from baseline to posttreatment with equivalent increases in total sleep time.[21,22] The subjective perception of sleep quality is also enhanced with PMR.[22,38] Some studies have shown that relaxation procedures are less effective than sleep restriction[57] or stimulus control[38,47] and others suggest that this treatment modality is less effective with older adults, particularly when it is not implemented under therapist supervision.[72]

Three additional relaxation-based treatments evaluated in controlled clinical trials include imagery training, autogenic training, and meditation. Meditation is a technique that involves focusing one's attention on a repetitive stimulation and repeating silently a mantra. Imagery training seeks to reduce cognitive arousal rather than somatic arousal. Of the three studies that have evaluated imagery training, two studies indicated that it was no more effective than a wait-list control during the inital treatment period, although sleep improvements became more noticeable at follow-up.[42,50] Conversely, one study focusing on sleep onset insomnia yielded significantly better outcome with imagery training than with standard progressive muscle relaxation.[71] For example, imagery training reduced sleep onset latency from 108 min at baseline to 50 min at posttreatment, whereas standard progressive muscle relaxation training (with somatic focusing) produced a reduction of 30 min (from 98 to 68 min) for the same period.[71] Three studies have evaluated the effects of meditation for insomnia.[53,70,73] All three studies reported significant improvements on the main outcome measure of sleep latency or wake after sleep onset. Meditation, alone or combined with PMR, was significantly more effective than control conditions and, in one study,[53] the reduction of time awake after sleep onset obtained with meditation was comparable to that obtained for stimulus control therapy. Finally, two studies of autogenic training[36,67] showed that this treatment modality produces equivalent outcome to standard PMR or biofeedback. Although additional relaxation treatments are sometimes advocated for the management of insomnia (e.g., abdominal breathing, hypnosis, thought stopping), there are currently no data to support those clinical recommendations.

Nine studies meeting inclusion criteria have evaluated the effects of **biofeedback** (e.g., EMG and EEG activity) for treating insomnia.[31,32,36,37,46,68,74-76] All nine studies have shown that biofeedback is an effective treatment modality for insomnia; however, two of those studies[46,74] showed that a pseudobiofeedback (i.e., placebo) was as effective as the active feedback modality. Improvements rates for patients treated with biofeedback procedures are comparable to those obtained with standard relaxation procedures. For example, average reductions of sleep onset latency for the studies reviewed by Morin et al.[21] were from 53 min at baseline to 33 min at posttreatment, to 27 min at follow-up. Four studies have also documented the effects of biofeedback with polysomnographic measures.[31,32,36,37] There has been only one study of biofeedback treatment for insomnia in the last ten years.[76] This decrease in interest may be due to the fact that biofeedback training takes longer than other forms of relaxation therapy, with little appreciable advantage.

Paradoxical intention has been evaluated in six studies meeting inclusion criteria.[38,47,49,54,55,77] All six studies focused on the problem of sleep-onset insomnia. In four studies,[38,49,54,77] paradoxical intention was more effective than control conditions in reducing sleep onset latency, whereas two studies[47,55] failed to report significant differences between this treatment and a placebo or wait-list control condition at posttreatment. Paradoxical intention produces smaller treatment gains than stimulus control or relaxation training. Reductions of sleep latency across studies averaged about 20 min from baseline (60 min) to post-

treatment (40 min). In addition, there is significant variability in the treatment response of insomnia patients to this treatment modality.

There has been no controlled evaluation of formal **cognitive therapy** for insomnia. However, at least six studies meeting inclusion criteria[34,45,59,75,76,78] and two others[35,39] have incorporated cognitive restructuring therapy as part of a multifaceted intervention. All of these studies have reported positive results and none have shown a negative outcome. While the specific contribution of cognitive therapy remains unclear, clinical evidence suggests that this treatment modality is particularly useful to alter dysfunctional beliefs and attitudes about sleep which often contribute to perpetuating emotional distress and sleep disturbances.[10] Because patients often perceive themselves as victims of insomnia, an important goal of treatment is to strengthen their sense of control in coping with the sleep problem. There is a definite need for additional studies to document the unique contribution of cognitive therapy in the management of insomnia.

Sleep hygiene education is often incorporated into insomnia treatments in order to safeguard against interference from poor sleep hygiene. At least ten studies[34,39,45,58-61,63,79,80] have combined sleep hygiene education with stimulus control, relaxation training, and several other nonpharmacological interventions. However, only three studies have evaluated the benefits of this educational component alone.[30,40,53] The available evidence indicates that sleep hygiene alone may have limited benefits for persistent insomnia. For example, one study[53] found that sleep hygiene produced a modest 27% reduction of time spent awake after sleep onset, and patient satisfaction was significantly lower with this intervention than with other treatment modalities such as stimulus control and meditation. Other studies[30,40] have shown that, unless additional interventions (e.g., stimulus control, bright light) are added, sleep education is of limited therapeutic value. Although inadequate sleep hygiene is rarely the primary cause of insomnia,[18] it may complicate an existing problem and hinder treatment progress. As such, sleep hygiene education is a necessary, if not a sufficient, treatment component and should be incorporated into the overall intervention. Although the exact contribution of this educational component is not entirely clear, there is no evidence that education has a detrimental effect on outcome.

4.6 Empirically-Supported Treatment

Criteria developed by the American Psychological Association[81] for empirically-validated psychological treatments, and subsequently revised,[82] were used to determine whether sufficient evidence was available to support a given treatment modality. Two sets of criteria have been proposed, those indicating well-established treatments and those for probably efficacious treatments. Criteria for well-established treatments require at least two between-group design studies demonstrating efficacy in one or more of the following ways: I. (a) superior to pill or psychological placebo or to another treatment, (b) equivalent to an already established treatment in a study with adequate statistical power; or II. a large series of single case design experiments (n > 9) demonstrating efficacy; such experiments must have used an experimental design and compared the intervention to another treatment as in I. a (above); III. the studies must be conducted with treatment manuals; IV. the characteristics of the sample must be well-described; V. the effects must have been demonstrated by at least two different investigators or investigatory teams. Criteria for probably efficacious treatments are: I. two studies showing the treatment is more effective than a waiting-list control group, or II. one or more studies meeting the well-established treatment criteria I, III, and IV, but not V., or III. a small series of single case design studies (n > 3) otherwise meeting well-established treatment criteria II, III, and IV. According to these criteria (see evidence Table 4), stimulus control therapy, progressive muscle relaxation, and paradoxical intention would meet criteria for well-established psychological treatment for insomnia, whereas sleep restriction, biofeedback, and multicomponent cognitive-behavior therapy would meet criteria for probably efficacious treatments.

5. TREATMENT INTEGRATION

5.1 Single, tailored, and multi-faceted interventions

A great deal of research effort has been devoted to comparing the relative efficacy of single and combined treatment methods. For instance, at least 15 studies have compared two or more of the following interventions: Stimulus control, relaxation, sleep restriction, sleep hygiene education, and paradoxical intention. Only five of these studies[38,42,47,50,57] have found a statistically significant advantage of one treatment over the others, usually stimulus control and sleep restriction being more effective than relaxation, paradoxical intention, or sleep hygiene education. The lack of more reliable differences is not surprising given that the sample sizes are often too small to provide enough statistical power to detect significant differences between two treatment conditions.

As most behavioral interventions are not incompatible with one another, some of those treatments can be combined to optimize outcome. Nine studies have examined the efficacy of multicomponent approaches combining two or more treatment modalities.[33,34,40,45,59,60,63,78,83] All of those studies indicate that a combined approach is significantly more effective than no treatment; however, combined approaches are not always more successful than the simpler and often shorter stimulus control or sleep restriction therapies. As shown in Table 3, average effect sizes ranging

from 0.92 to 1.05 on the main outcome measures of sleep onset latency and wake after sleep onset have been reported for combined approaches, whereas mean effect sizes for stimulus control have ranged from 0.70 to 1.16.[21,22] The best outcomes from multicomponent interventions have been reported when sleep restriction and/or stimulus control procedures were integrated with other methods such as cognitive restructuring and relaxation methods.[33,34] Jacobs et al.[33] obtained average sleep latency reductions of 75%, which is significantly higher than the average reduction of 43% obtained for all single treatments combined.[21] Likewise, in a study of late-life insomnia with cognitive-behavior therapy,[34] time awake after sleep onset was reduced by 54% (from an average baseline of 62 min), compared to the average reduction of 46% obtained for all treatment modalities combined. Thus, it may be that stimulus control and sleep restriction procedures are the most active therapeutic ingredients. A combined approach must be planned carefully in order to avoid the possible effect of substituting time in therapy with less effective advice and intervention that may dilute the overall treatment effect.[56]

An appealing alternative to combining treatment procedures is to tailor treatment to patients' characteristics (i.e., progressive muscle relaxation with tensed patients, or stimulus control for those with sleep-incompatible activities). The only two studies of tailored treatment have yielded negative results on the efficacy of this strategy.[56,75] For example, Espie et al.[56] found that randomized treatment produced a larger reduction of sleep latency from baseline (82 min.) to posttreatment (42 min.) compared to treatment tailored to patients' characteristics (85 min to 56 min). In another study,[75] Sanavio found no differential improvements when patients with high tension level at baseline were assigned to EMG biofeedback treatment and patients with a high rate of intrusive thoughts were assigned to cognitive therapy compared to mismatched conditions. This lack of differential improvement may be in part due to lack of statistical power or to the lack of reliable and valid measures to investigate causal factors, combined with incorrect assumptions about causation.

Despite the equivocal evidence regarding combined and tailored treatment approaches, it is unlikely that any single treatment will be effective with all patients. Effective clinical management of insomnia will often involve a combination of treatment procedures. Additional research on multifactor and tailored treatments is warranted, particularly towards the planning of symptom-specific interventions and investigations of mechanisms of change.

5.2 Combining behavioral and pharmacological therapies

It is common clinical practice to provide general sleep hygiene guidelines along with a hypnotic drug when treating insomnia. This biobehavioral approach should theoretically maximize outcome by capitalizing on the more rapid effects from drug therapies and longer lasting effects from behavioral methods. Only four studies, including three that were conducted during the search period covered by this paper, have directly compared the separate and combined effects of behavioral and pharmacological therapies for insomnia.

The first published study[84] compared a 3-week regimen of triazolam, 0.5 mg used nightly, to a behavioral approach combining stimulus control and relaxation training. Using a parallel group design, both treatments produced equivalent improvements at posttreatment (mean sleep latency of 36 min), but the trajectory of change over time was different for the two conditions. Subjects receiving triazolam were more improved after the first week of treatment, whereas behaviorally-treated subjects sustained greater benefits at the 1-month follow-up after drug tapering. A subsequent study[85] examined the differential effectiveness of triazolam (0.25 mg nightly), alone and combined with stimulus control and relaxation training. The two conditions produced equivalent changes at posttreatment, but the combined intervention yielded a slightly better outcome at the short-term follow up, especially for total sleep time and ratings of restedness in the morning.

Hauri[80] compared sleep hygiene education and relaxation training, alone or in combination with occasional hypnotic use (triazolam, no more than once per week), to a wait-list control condition. Sleep efficiency (increased from 80% to 85%) and total sleep time (increased by 38 min) were more improved in the two active treatment conditions than in the control group (sleep efficiency stayed at 81% and total sleep time decreased by 5 min); there was no difference between the two active treatment conditions. At the 10-month follow-up assessment, subjects treated with the behavioral approach alone had a higher sleep efficiency (83%) than those who had received the combined intervention (79%).

Morin and colleagues[35] conducted a placebo-controlled study of cognitive-behavior therapy and pharmacotherapy (temazepam, 7.5-30 mg), singly or combined, for late-life insomnia. The findings showed that all three active treatments were more effective than drug-placebo. Posttreatment sleep efficiency averaged 84% for the active treatment conditions and 73% for the placebo condition. These results were corroborated with PSG measures, with sleep efficiency moving from 76% at baseline to 84%-87% at posttreatment for the active conditions, whereas the placebo condition remained stable (79% to 80%) for the same period. Follow-up data obtained at 12 and 24 months after treatment, showed that subjects treated with cognitive-behavior therapy sustained their clinical gains (sleep efficiency of 85%), whereas those treated with drug therapy alone did not (77%). The combined intervention showed a significant loss of therapeutic benefits, although there was much variability across subjects in that condition

and over the follow-up periods.

Collectively, the findings suggest that hypnotic drugs may produce faster sleep improvements, particularly in the first few days of treatment,[84] compared to behavioral methods such as relaxation and sleep hygiene education. Therapeutic gains in the intermediate term (i.e., 4-8 weeks), however, are comparable for behavioral, pharmacologic, and combined biobehavioral therapies.[35,80,85] The long-term effects (i.e., 6-24 month follow-ups) of behavioral and pharmacological treatment modalities are fairly clear in that the former retains its clinical benefits very well over time, whereas the latter tends to return toward baseline values as the medication is discontinued after completion of treatment.[35,84,85] What is more uncertain is the long-term outcome of patients receiving a combined approach. Although it might be expected that a combined intervention would be superior to either of its single components, the evidence available indicates that subjects receiving both hypnotic drugs and behavior therapy do not retain their clinical gains at follow-up as well as those treated with behavior therapy alone.[35,80] One could speculate the explanation is that of a negative attributional effect. Patients treated with hypnotic drugs, even when combined with a behavioral intervention, may attribute sleep improvements to the drug alone. In turn, such attributions may undermine the development of appropriate coping skills and, when sleep medication is discontinued, the patients may become helpless and is more likely to return to the vicious cycle of insomnia, emotional distress, and further sleep disturbances.[24]

6. TREATMENT RESPONSE AND MODERATING VARIABLES

Several patient (demographic and clinical) and treatment-related variables (format, delivery mode) have been examined as potential moderators of treatment response. Few have been reliably associated with outcome.

6.1 Patients' characteristics

The relationship of the patient's age to treatment outcome is equivocal. For example, one study[72] reported that older adults were less responsive to self-help treatments for insomnia than younger persons. In a re-analysis of their data from seven treatment studies (n = 216), Lacks and Powlishta[43] reported that younger age was predictive of better treatment response. Contrary to those findings, more recent studies of late-life insomnia[34,51,57,86] indicate that when older adults are well screened for medical and sleep disorders such as sleep apnea and periodic limb movements, the magnitude of their treatment response is comparable to that obtained with younger patients. One exception may be for relaxation training, which appear less effective with older insomniacs.[57,64] Although insomnia is more prevalent among women, and women represent about 60% of the participants in clinical trials, there is currently no evidence that gender is related to treatment response.

Although most treatment studies report multiple dependent measures (i.e., sleep latency, number and duration of awakenings, total sleep time), less than 10 studies from the 50 reviewed have focused on sleep maintenance problems. Older adults have been the primary target of those few studies of sleep maintenance insomnia. The evidence from meta-analyses (see Table 2) indicates that behavioral interventions produce comparable to slightly smaller benefits for sleep maintenance than for sleep onset difficulties. The average effect size for time awake after sleep onset is 0.65, which corresponds to a reduction of time awake after sleep onset from 70 min at baseline to 38 min at posttreatment. This improvement rate is comparable to that for sleep latency (from 64 min to 37 min).[21] The average effect size for the number of awakenings is 0.58, which represent a mean reduction of about one awakening from baseline to posttreatment.[21,22] Sleep maintenance problems have been treated effectively with stimulus control therapy and sleep restriction[42,48,57] and with cognitive-based relaxation treatments.[87,88] One study reported minimal changes on sleep-maintenance measures following countercontrol therapy[86] and another study reported only modest changes when using progressive muscle relaxation with older adults.[64] No study has yet addressed the problem of early morning awakening, other than with bright-light treatment.[89]

Only one study[47] has directly examined whether treatment response was mediated by initial insomnia severity. In a study of primary chronic insomnia, stimulus control treatment was found to be more effective than relaxation or paradoxical intention regardless of initial severity levels. In a clinical replication series,[45] patients with co-existing depressive or anxiety disorders reported more severe sleep difficulties than those with primary insomnia, both at baseline and posttreatment. Nonetheless, patients with secondary insomnia showed sleep improvements that were similar in magnitude to those obtained by primary insomniacs. Four additional studies have shown that clinically-referred patients achieve comparable and, perhaps, superior outcomes to research participants solicited from the community,[26,38,45,59] even though they may also display higher emotional distress than this latter group.[90]

The results from one meta-analysis[22] and from three individual studies[42,52,91] indicate that insomniacs using hypnotic medications do not respond to behavioral treatment as well as unmedicated patients, at least during the initial intervention. However, preliminary evidence from four studies[64,92-94] suggest that psychological interventions may facilitate reduction or discontinuation of hypnotic medications when a supervised withdrawal taper is integrated to treatment. Because of rebound insomnia, the sleep of hypnotic users may need more time to normalize.

6.2 Contextual variables

A number of contextual factors on "how treatment is implemented" may influence outcome. These factors include the format (e.g., individual vs. group treatment) and dosage of therapy (e.g., number of sessions, duration of treatment), as well as some therapist variables (e.g., competency, training).

6.2.1 Treatment format

Insomnia patients seen in clinical practice are typically treated on an individual basis. However, group therapy has been used in an approximately equal number of studies (e.g.,[34,42,47-51,71,95]) as individual therapy (e.g.,[26,30,38,54,57,76]). Although there has been no direct comparison of these two treatment formats, the evidence from a meta-analysis suggest a modest superiority of individual over group therapy for insomnia.[21] Because few studies have incorporated measures of clinical significance of change, it is yet unclear whether individual treatment reflects more effective clinical practice. From a cost-effectiveness perspective, there are certainly advantages to implementing treatment in a group format.

Self-help material on insomnia[96,97] has attracted considerable interest in the general population. Five studies have evaluated self-help insomnia programs offered under different formats (e.g., books, audio/video tapes).[58,61,62,72,91] The evidence from those studies indicates that self-help treatment is effective but not for everyone. For instance, people with more severe or chronic insomnia, and those using hypnotic medications are likely to benefit less from self-help treatment than from therapist-led treatment. For instance, Morawetz[91] reported that self-help treatment (audiotape and written material) was equally effective with or without therapist guidance for unmedicated insomniacs (48% vs. 50% SOL reductions); however, medicated participants benefited less from the self-help material alone. Another study[58] showed that therapist-assisted treatment produced a more favorable outcome (posttreatment SOL and WASO of 27 min and 37 min.) relative to self-administered treatment without therapist guidance (post SOL and WASO of 38 and 63 min). It is likely that self-management programs will remain a useful adjunct to more formal insomnia treatment. Prospective investigations are needed to identify patients' characteristics that predict satisfactory outcome from self-help treatment and those who may benefit from other forms of minimal interventions such as brief consultations.[98]

6.2.2 Treatment duration/dosage

In clinical practice treatment is usually maintained until satisfactory symptom reduction has been achieved; in contrast, most research studies have relied on a predetermined number of treatment sessions. While there are no clear guidelines for optimal treatment duration, in the 59 studies (i.e., 183 treatment conditions) reviewed by Morin et al.,[21] treatment averaged a total of 5 hours (sessions) over a mean interval of 5 weeks. Although such a regime may be effective for the average patient with primary insomnia, successful outcome may require more time for patients with comorbid medical or psychiatric disorders and for those with prolonged hypnotic usage. For example, heterogeneous patients treated at a sleep clinic attended an average of 8 therapy sessions conducted over a mean interval of 14 weeks.[45] Some treatment methods (e.g., relaxation training) may require more training sessions and longer intervals than others (e.g., sleep restriction). Treatment progress may occur at differential rates depending on the treatment modality applied. For example, an early treatment response to stimulus control has been reported with significant effects emerging after only one week of active treatment.[47,56] It is likely that several factors may influence the optimal treatment dosage for a given patient (e.g., initial insomnia severity and comorbidity, the patient's motivation and compliance with treatment), but there is not enough evidence currently available to make specific recommandations on this issue. Additional studies are needed to determine optimal treatment dosage in the clinical management of insomnia.

6.3 Therapist variables

The majority of insomnia patients are managed by the family physician without recourse to specialized therapists or sleep disorders centers. In contrast, behavioral treatment studies of insomnia have relied on manual-driven treatment protocols implemented by well-trained therapists. Although there is no clear evidence that treatment response is dependent on level of training, the evidence suggests that outcome is slightly superior when treatment is administered by a professional (e.g., psychologist) rather than by a trainee or by an automated approach such as a manual or a tape.[21] Although there is now greater availability of resource material on insomnia treatment designed for clinical practitioners,[8,9,10,99] behavioral interventions are not very well known and remain underutilized by health-care professionnals. With adequate training, most clinicians should be capable of educating patients about basic sleep information and sleep hygiene principles. These elements form the basis for most cognitive-behavioral interventions. Basic principles of stimulus control and sleep restriction can be taught by most clinicians as well, but relaxation-based interventions and cognitive therapy will often require more specialized training. One recent study showed that stimulus control procedures implemented by family physicians produced treatment gains that were very similar to those obtained by well-trained mental-health professionals.[100]

7. CONCLUSIONS

Significant advances have been made in the behavioral management of insomnia in the last two decades. Our review of 48 treatment studies indicate that several well-defined nonpharmacological interventions produce reliable and durable changes in the sleep of patients with chronic and primary insomnia. Between 70% and 80% of insomnia patients benefit from treatment, 50% achieve clinically meaningful outcomes, and about one third become good sleepers. Although the majority of patients benefit from treatment, there is significant variability in the magnitude of treatment response, and most treated insomniacs do not become good sleepers. Posttreatment values of sleep onset latency and wake after sleep onset, the two main target symptoms of treatment, often fall below or near the 30-min cut-off criterion used to define insomnia, whereas sleep duration is increased by a modest 30 minutes. Sleep quality is also significantly enhanced with treatment. Most studies have relied only on daily sleep diaries to assess outcome, but a few have also validated treatment effects with polysomnography. Sleep improvements achieved with nonpharmacological interventions are sustained over time, for at least up to six months after treatment completion in these reports. Little attention has been paid to the important issue of whether improved sleep produces meaningful changes in daytime functioning and quality of life. Six treatment modalities meet the American Psychological Association criteria for empirically-validated (e.g., stimulus control, progressive muscle relaxation, paradoxical intention) or probably efficacious (i.e., biofeedback, sleep restriction, multicomponent cognitive-behavior therapy) treatments for insomnia. Although there may be advantages to combining behavioral and pharmacological interventions, there is not enough empirical evidence at this time to guide clinicians about the most appropriate indications for implementing such integrated biobehavioral interventions.

Our current knowledge of the effects of nonpharmacological interventions comes predominantly from efficacy studies conducted with primary insomnia patients recruited specifically for clinical trials. Aside from pilot studies, there is little evidence that non-drug therapies are effective to treat acute or chronic insomnia in primary care, or insomnia associated with medical, substance abuse, or psychiatric disorders. These types of insomnia patients may represent a significant proportion of those seen in clinical practice.[101] Prospective research is needed to evaluate the effectiveness of nonpharmacological interventions and to validate available treatment procedures with patients seeking treatment in various clinical settings (primary care) and with various co-existing illness (secondary insomnia). Additional research is also needed to define more precisely several parameters mediating treatment outcome (i.e., patients, treatment, and contextual). For instance, what is the optimal treatment dosage (number of therapy sessions, treatment duration) and most cost-effective treatment delivery model (individual, group, self-help, therapist-assisted)? Who should be administering these therapies and what qualifications and training are required? Finally, more research is needed to examine the indications, risks and benefits, and limitations of integrating behavioral and pharmacological interventions.

Table 1. Studies of Nonpharmacological Treatments of Insomnia.

Author(s) (year)	Design (control)	N; % of female; Age (mean)	Treatment conditions	Format of treatment	Treatment (Weeks/hours) Follow-Up (months)	Bias	Outcomes
Alperson & Biglan (1979)[1]	RCT (waiting-list)	29; 48.0; 17-80 (range)	Relaxation/stimulus control (young); Relaxation/stimulus control (old); Relaxation/in-bed-activities; Self-monitoring	Self-adminis-tered	4/3 2	Small sample, only short-term (2-month) FU.	All self-administered treatments reduced reported SOL (66 to 41 minutes), but older adults improved significantly less than younger persons.
Asher & Turner (1979)	RCT (placebo, no treatment)	25; 60.0; 39	Paradoxical intention; Placebo; No treatment	Individual	4/3 no FU	Small sample, no FU.	Paradoxical intention decreased SOL significantly (62 to 29 min) more than placebo (63 to 51 min) or no-treatment (71 to 62 min) control conditions.
Carr-Kaffashan & Woolfolk (1979)	RCT (placebo)	30; 60.0; 40.1	Progressive relaxation plus meditation; Placebo	Individual	4/4 6	High attrition rate at FU.	Active treatment (relaxation and meditation) reduced SOL significantly more (46%) than the placebo (12%). Treatment gains were equivalent for both moderate and severe insomniacs. Significant reductions of anxiety symptoms.
Chambers & Alexander (1992)[1]	CRS	103; 67.0; 39.9	Multicomponent CBT (stimulus control, sleep restriction, sleep hygiene, cognitive restructuring).	Individual	1/2.5 6	No control/ comparison; Global and retrospective measures	Treatment reduced reported SOL and WASO by 30 and 50 minutes respectively. 58% of patients rated their sleep as significantly improved.
Coursey et al. (1980)	RCT (electro-sleep)	22; 45.5; 38.6	EMG biofeedback; Autogenic training; Electrosleep	Individual	6/8 1	Small sample	More patients in the biofeedback and autogenic training conditions improved significantly on the main outcome measures of SOL and SE. Corroborated by PSG data.
Davies (1989)[1]	CRS (no)	15; 86.7; 46 (median)	Multicomponent CBT (stimulus control, cognitive restructuring, problem solving, anxiety management, relaxation, drug withdrawal.)	Group	12/NA 12	No control group; global and retrospective measures of sleep	Subjective improvements of sleep satisfaction and total sleep time; significant reductions of health symptoms and hypnotic medications. Gains well maintained at FU.

Davies et al. (1986)	RCT (waiting list)	34 47.1 58.6	Countercontrol therapy; Waiting list	Group	4/4 12	Short-term (1-month) FU	Significant but small reductions of WASO (80 to 58 min) from baseline to posttreatment. Sustained improvements at 4-week follow-up.
Edinger & Stout (1985)[1]	CRS (no)	20 50.0 40.0	Multicomponent (education, relaxation, stimulus control, sleep hygiene).	Group	5/5 1	No control/ comparison	Treatment reduced SOL from 59 to 38 minutes; 15% of participants decreased their use of sleep medication.
Engle-Friedman et al. (1992)	RCT (waiting list)	53 66.0 59.6	Sleep hygiene; Sleep hygiene/relaxation; Sleep hygiene/stimulus control; Waiting list	Individual	4/4 24		Significant improvements on measures of SOL and number of awakenings in all active treatments but not in the control condition. Stimulus control more effective at post and 2-year FU. No significant changes on PSG measures.
Espie et al. (1989)[1]	RCT (placebo, no treatment)	70 60.7 44.9	Stimulus control; Progressive relaxation; Paradoxical intention; Placebo; No treatment	Individual	8/8 17		SOL was reduced by 62% (stimulus control), 51% (paradoxical intention), 37% (relaxation), 26% (placebo) and 14% (no treatment). Stimulus control more effective to reduce SOL, but relaxation yielded better results on perceived sleep quality.
Freedman & Papsdorf (1976)	RCT (placebo)	18; 55.6; 17-39 (range)	EMG biofeedback; Progressive relaxation; Placebo	Group	2/3 2	Small sample, No long-term FU.	Biofeedback and relaxation produced greater reductions of SOL (30 and 23 min) than placebo, but were not significantly different from each other. PSG data corroborated those findings.
Friedman et al. (1991)[1]	RCT (no)	22 63.6 69.2	Sleep restriction; Relaxation	Individual	4/4 3		Improvement rates for sleep restriction was twice that of relaxation (33% vs 16% for WASO). SE was increased from 67% to 83% with sleep restriction; total sleep time was increased by 51 minutes at FU relative to baseline.
Guillemi-nault et al. (1995)	RCT (no)	30 56.3 44.0	Stimulus control/sleep hygiene; Stimulus control/sleep hygiene/exercise; Stimulus control/sleep hygiene/bright light	Individual	4/NA 9-12		Improvements noted for all three conditions on measures of SOL and TST but only the bright light condition showed statistically significant changes over time. Treatment outcome corroborated with actigraphy.
Gustafson (1992)[1]	CRS (no)	22; 54.5; 42.0	Relaxation	Self-adminis-tered	NA 12	Global and retrospective measures No control/ comparison	86% rated the treatment as successful; 27% felt they still needed additional treatment. 32% reported a reduction in the use of sleep medications.

Hauri (1981)[1]	RCT (no treatment)	48; 62.5; 41.3	EMG Biofeedback; Combined EMG and theta feedback; Sensorimotor rhythm feedback; No treatment	Individual	8/25 9		EMG biofeedback produced best results for tensed insomniacs, whereas sensori-motor feedback was more effective for those already relaxed at baseline. PSG data available.
Hauri et al. (1982)[1]	RCT (no)	16; 37.5; 48.8	Theta feedback; Sensorimotor rhythm biofeedback	Group	13/13 9	Small sample	Tensed insomniacs benefited only from theta biofeedback, whereas relaxed patients benefited only from sensorimotor feedback. PSG data available.
Hauri (1997)	RCT (waiting list)	26; 73.1; 47.7	Sleep hygiene/relaxation; Sleep hygiene/relaxation /medication Waiting list	Individual	6/6 10		The two active treatment conditions were more improved on SE and TST than the control group at post; however, at FU, subjects treated with the behavioral approach alone had a higher SE (83%) than the combined intervention (79%). Actigraphy data available.
Hughes & Hughes (1978)[1]	RCT (placebo)	36; 66.7; 34.2	EMG biofeedback; Pseudobiofeedback; Relaxation; Stimulus control	Individual	2-8/1-5 12	Small sample	Significant pre to post reductions of SOL across all four conditions (50 to 28 minutes), but no between group difference.
Jacobs et al. (1996)[1]	CRS	102; 61.0; 39.3	Multicomponent (sleep restriction, modified stimulus control, relaxation, education, cognitive restructuring, medication withdrawal).	Group	10/14 6	Global and retrospective measures; No control/ comparison	58% of patients reported significant sleep improvement, 33% moderate and 9% slight improvement. 91% of sleep medication users eliminated or reduced medication use. 90% of patients maintained or enhance their sleep at FU.
Jacobs et al. (1993)	NRCT (good sleepers)	26; 58.3; 37.8	Multicomponent (sleep restriction, modified stimulus control, relaxation); Good sleepers	Individual	10/2.5 6		Significant improvements on measures of SOL and TST based on daily sleep diary and PSG. No significant difference between treated patients and good sleepers at posttreatment. Significant reductions of anxiety and depressive symptoms.
Jacobs, Rosenberg et al. (1993)	RCT (no)	20; 80.0; 36.7	Sleep education/stimulus control; Sleep education/stimulus control/relaxation	Individual	10/3 1	No control/no long term FU	Combined stimulus control and relaxation reduced SOL by 77% compared to the 63% for stimulus control alone.

Lacks, Bertelson, Gans et al. (1983)	RCT (placebo)	64; 75.0; 40.6	Stimulus control; Progressive relaxation; Paradoxical intention; Placebo	Group	4/4 3		Treatment reduced SOL by 42 (stimulus control), 11 (relaxation), 11 (paradoxical intention) and 10 (placebo) minutes. Stimulus control was the most effective intervention across insomnia severity levels.
Lacks, Bertelson, Sugerman et al. (1983)	RCT (placebo)	15; 60.0; 43.0	Stimulus control; Placebo	Group	4/5 3	Small sample	Stimulus control reduced WASO to 26 minutes at post, compared to 51 minutes for the placebo condition. Gains well maintained at FU (29 and 45 minutes).
Ladouceur & Gros-Louis (1986)[1]	RCT (waiting list)	27; 66.6; 41.8	Stimulus control; Paradoxical intention; Sleep education Waiting list	Group	4/10 2	No long term FU Small sample	Stimulus control and paradoxical intention were equally effective and significantly better than education and waiting list in reducing SOL.
Lichstein & Johnson (1993)[1]	NRCT (good sleepers)	57; 100.0; 66.2	Relaxation	Individual	2/3 1.5	No long term FU	Relaxation produced sleep improvement for unmedicated insomniacs (WASO = 71 to 47 minutes). SE improved for unmedicated (64% to 72%) and medicated (62% to 70%) insomniacs. Medication reduced by 47% in medicated patients.
Lick & Heffler (1977)[1]	RCT (placebo, no treatment)	40; 65.0; 47.5	Progressive relaxation with/without tape; Placebo; No treatment	Individual	4/6 1	No long term FU.	Relaxation, with or without tape, more effective than controls to reduce SOL (63 to 34 minutes vs 65 to 64 minutes). Treatment also reduced medication usage and anxiety symptoms.
McClusky et al. (1991)	RCT (no)	30; 56.7; 32.0	Stimulus control/relaxation training; Triazolam	Group	3/NA 1	No long term FU	Triazolam produced a faster reduction of SOL, whereas the behavioral condition yielded better outcome at FU.
Mitchell (1979)	RCT (no treatment)	24; 62.5; 37.4	Progressive relaxation; Relaxation/cognitive control; Information/environmental change No treatment	NA	8/4-8 1.5	Small sample, no long term FU.	Significant reductions of SOL in all three conditions; better results for the relaxation plus cognitive control condition.
Morawetz (1989)[1]	RCT (waiting list)	141; NA; 44.0	Stimulus control/relaxation (audio tape and manual); Stimulus control/relaxation (with therapist); Waiting list	Individual, self-administe-red	5/10 4		For unmedicated subjects, the self-help treatment reduced SOL by 48% compared to 50% for the therapist condition. For medicated subjects, self-help treatment was less effective than therapist condition.

Morin & Azrin (1987)	RCT (waiting list)	21; 66.0; 57.0	Stimulus control; Imagery training; Waiting list	Group	4/4 12	Small sample	Stimulus control effective in treating sleep-maintenance insomnia (65% reductions of WASO) but imagery training was not (16% reductions).
Morin & Azrin (1988)[1]	RCT (waiting list)	27; 63.0; 67.4	Stimulus control; Imagery training; Waiting list	Group	6/7 12	Small sample	Stimlus control yielded higher improvements than imagery training and control on measures of WASO and TST. Changes well-maintained at FU. Outcome validated with electromechanical timer.
Morin et al. (1993)	RCT (waiting list)	24; 70.8; 67.1	Multicomponent CBT (stimulus control, sleep restriction, cognitive therapy, education); Waiting list	Group	8/12 12		CBT was effective in reducing WASO and in increasing SE (69% à 83%). Results were corroborated by PSG data. Therapeutic gains were maintained at 3 and 12-month FU.
Morin et al. (1994)[1]	CRS (none)	100; 64.0; 45.1	Multicomponent CBT (stimulus control, sleep restriction, cognitive therapy, education, medication withdrawal)	Individual	14/NA 24	No control/ comparison	Reported SE improved from 68% to 80% for the total sample. Significant reductions in usage of sleep aids (46% to 28% medicated nights). Sleep clinic patients.
Nicassio & Bootzin (1974)	RCT (no treatment)	30; 70.0; 45.1	Progressive relaxation; Autogenic training; Self-relaxation; No treatment	Individual	4/4 6		Both treatments reduced SOL (120 to 60 min) significantly more than controls (120 to 108 min). Gains well maintained at FU.
Nicassio et al. (1982)	RCT (placebo, no treatment)	40; 77.5; 43.5	Progressive relaxation; EMG Biofeedback; Biofeedback placebo; No treatment	Individual	6/5 6		Relaxation and biofeedback reduced SOL 57% and 63% respectively, compared to 39% reductions in the biofeedback placebo condition.
Puder et al. (1983)	RCT (waiting list)	16; 81.3; 67.1	Stimulus control; Waiting list	Group	4/5 1.5	No long term FU Small sample	Reported SOL was reduced from 68 to 27 minutes with stimulus control treatment in a group of older adults, (64 to 62 minutes for the control subjects).
Riedel et al. (1995)	RCT (waiting list)	75; 65.6; 67.4	Education/sleep restriction (Video); Education/sleep restriction (Video/therapist guidance); Waiting list	Self-help or Group	Video :2/30min Therapist : 2/4 2	No long term FU	Sleep restriction and education administered by video with or without therapist guidance. The video only yielded reductions in WASO (92 to 63 minutes), but the addition of therapist guidance enhance outcome on measures of SOL and WASO (68 to 37 minutes).

Sanavio (1988)	RCT (no)	24; 58.3; 39.0	EMG Biofeedback; Cognitive therapy (cognitive restructuring, paradoxical instructions, thought stopping)	Individual	2/6 12	No control	Both treatments reduced SOL by 54%. Cognitive therapy reduced pre-sleep intrusions and biofeedback reduced pre-sleep tension. No differential results found at 3 and 12 month FU.
Sanavio et al. (1990)	RCT (waiting list)	40; 65.0; 39.6	EMG biofeedback; Cognitive therapy; Stimulus control/progressive relaxation; Waiting list	Individual	2/6 36	High attrition rate at FU	All three treatments more effective than control in reducing SOL (37% vs 0.6%) and WASO (50% vs 1%). Benefits were maintained at 1 and 3 year FU.
Schoicket et al. (1988)	RCT (no)	65; 56.9; 52.1	Stimulus control; Meditation; Sleep hygiene	Group	4/4 1.5	No long term FU	Significant reductions of WASO for stimulus control (36 min), meditation (30 min) and sleep hygiene (22 min). Sleep hygiene subjects were less satisfied with treatment and more likely to consider themselves insomniacs at FU.
Spielman et al. (1987)[1]	CRS (no)	35; 48.6; 46.0	Sleep restriction	Individual	8/NA 9	No control/ comparison condition	Treatment reduced SOL and WASO by 29 and 109 minutes respectively; SE increased from 67% to 87%. Gains were maintained at FU.
Stanton (1989)	RCT (placebo)	45; 57.8; 23-67 (range)	Sleep hygiene/stimulus control; Sleep hygiene/relaxation; Placebo	Group	4/2 6		Hypnotic relaxation was more effective to reduce SOL (51 to 22 minutes) than stimulus control (SOL = 53 to 39 minutes) and placebo (SOL = 50 to 45 minutes).
Toler (1978)	RCT (no treatment)	27; 100.0; 26.8	Relaxation training; Relaxation training/stimulus control; No treatment	NA	2/10 2	Small sample, global and retrospective measures	Combined condition reduced the number of awakenings compared to controls. Both treated groups decreased their anxiety. Gains not maintained at FU.
Turner & Asher (1979)[1]	RCT (placebo, waiting list)	50; 50.0; 39.0	Stimulus control; Progressive relaxation; Paradoxical intention; Placebo; Waiting list	Individual	4/3 0	No FU	All treatments improved sleep significantly more than placebo and waiting list controls.
Turner & Asher (1982)[1]	RCT (waiting list)	60; 48.3; 37.0	Stimulus control; Progressive relaxation; Paradoxical intention; Waiting list	Individual	4/2.5 NA	No FU	Stimulus control, progressive relaxation and paradoxical intention reduced SOL to 22, 28 and 29 minutes respectively at posttreatment.

VanderPlate & Eno (1983)	RCT (waiting list)	36; NA; 20.0	EMG biofeedback; Pseudobiofeedback; Self-monitoring; Waiting list	Individual	NA/3.3 2	Small sample; No long term FU	Biofeedback reduced SOL from 30 to 15 minutes, whereas pseudobiofeedback reduced from 40 to 17 minutes. There was no difference between the two groups.
Woolfolk, Carr-Kaffashan & McNulty (1976)	RCT (waiting list)	24; 75.0; 44.3	Progressive relaxation; Meditation; Waiting list	Group	4/4 6	Small sample, global and retrospective measures	Both treatments were superior to control in reducing SOL (70 to 32 minutes). Both treated groups remained improved at FU.
Woolfolk & McNulty (1983)	RCT (waiting list)	44; 68.2; 43.3	Imagery training; Imagery training/muscle tension-release; Somatic focusing; Progressive relaxation; Waiting list	Group	4/4 6		All the treatment conditions improved reported SOL more than controls (mean of 103 to 62 minutes). Imagery training more effective than standard relaxation at 6-month FU.

[1] Use of sleep medication upon entering treatment was permissible.

Abbreviations: RCT = Randomized Clinical Trial; NRCT = Non Randomized Clinical Trial; CRS = Clinical Replication Series, SOL=sleep onset latency; WASO=wake after sleep onset; SE=sleep efficiency; TST=total sleep time; TWT=total wake time; PSG=polysomnography; CBT=cognitive behavior therapy; FU=longest available follow-up; NA=information not available.

Table 2—Mean effect size obtained in two meta-analysis of non-pharmacological treatments of insomnia.

	Meta-Analysis	
Sleep Variables	Morin, Culbert, & Schwartz (1994) z	Murtagh, & Greenwood (1995)[a] d
Sleep-Onset Latency	0.88 (91)	0.87 (116)
Wake After Sleep Onset	0.65 (15)	—
Number of Awakenings	0.53 (38)	0.63 (55)
Total Sleep Time	0.42 (36)	0.49 (60)
Sleep Quality	—	0.94 (53)

[a] sample-size-weighted mean effect size
d = effect size (number of comparisons on which effect size is based)

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Table 3—Mean effect size of non pharmacological treatments for insomnia.

Sleep Variables	Sleep-Onset Latency		Wake After Sleep Onset		Number of Awakenings		Total Sleep Time		Sleep Quality	
Treatments	Morin z	Murtagh d	Morin z	Murtagh d	Morin z	Murtagh d	Morin z	Murtagh d	Morin z	Murtagh d
Stimulus control	0.81 (15)	1.16 (20)	0.70 (5)	—	0.59 (11)	0.61 (12)	0.41 (7)	0.38 (6)	—	1.30 (6)
Sleep restriction	0.98 (1)	0.85 (4)	0.76 (1)	—	—	—	-1.06 (1)	0.37 (4)	—	—
Relaxation, somatic	0.83 (32)	—	0.06 (1)	—	0.56 (13)	—	0.25 (10)	—	—	—
Relaxation, cognitive	1.20 (7)	—	0.28 (2)	—	0.56 (3)	—	0.28 (1)	—	—	—
Progressive muscle relaxation	—	0.81 (36)	—	—	—	0.57 (15)	—	0.52 (16)	—	0.97 (15)
Other relaxation	—	0.93 (13)	—	—	—	0.52 (20)	—	0.53 (24)	—	0.98 (16)
Relaxation	—	0.84 (49)	—	—	—	0.37 (5)	—	0.57 (5)	—	1.08 (1)
Non relaxation	—	0.97 (36)	—	—	—	0.73 (18)	—	0.28 (16)	—	1.00 (14)
Biofeedback	1.00 (7)	—	0.70 (2)	—	0.97 (2)	—	0.38 (4)	—	—	—
Paradoxical intention	0.63 (9)	0.73 (12)	0.81 (1)	—	0.73 (4)	1.00 (6)	0.46 (5)	0.10 (6)	—	0.77 (8)
Sleep hygiene	0.71 (2)	—	—	—	-0.12 (1)	—	1.16 (2)	—	—	—
Multicomponent	1.05 (15)	1.00 (18)	0.92 (3)	—	-0.05 (4)	0.84 (10)	0.75 (6)	0.78 (18)	—	1.12 (17)
Placebo	—	0.46 (13)	—	—	—	0.41 (7)	—	0.10 (5)	—	0.21 (6)
All treatments combined	0.88 (91)	0.81 (116)	0.65 (15)	—	0.53 (38)	0.63 (55)	0.42 (36)	0.49 (60)	—	0.94 (53)

a sample-size-weighted mean effect size; d = effect size (number of comparisons on which effect size is based)

Table 4—Key studies supporting efficacy of nonpharmacological treatments of insomnia.

Treatment	Study reference	Evidence
Stimulus control[1]	Espie et al. (1989)	SC > PLA & PMR
	Lacks, Bertelson, Sugerman et al. (1983)	SC > PLA
	Morin & Azrin (1987;1988)	SC > IT
	Turner & Asher (1979)	SC > PLA
Relaxation (PMR)[1]	Licks & Heffler (1977)	PMR > PLA
	Nicassio et al. (1982)	PMR > no treatment
	Turner & Asher (1979)	PMR > PLA
	Woolfolk & McNulty (1983)	PMR > WL
Paradoxical Intention[1]	Espie et al. (1989)	PI > PLA
	Turner & Ascher (1979)	PI > PLA
Sleep restriction[2]	Friedman et al. (1991)	SR > PLA
EMG biofeedback[2]	Nicassio et al. (1982)	BF > no treatment
	Sanavio et al. (1990)	BF > WL
	VanderPlate & Eno (1983)	BF > WL
	Freedman & Papsdorf (1976)	BF > PLA
Muticomponent CBT[2]	Morin et al. (1993)	CBT > WL
	Morin et al. (1999)	CBT > PLA

[1] Well established treatments according to APA criteria for empirically supported treatments.
[2] Probably efficacious treatments according to APA criteria for empirically supported treatments.
Abbreviations: SC = Stimulus Control; SR = Sleep Restriction; PMR = Progressive Muscle Relaxation; IT = Imagery Training; BF = EMG Biofeedback; CBT = Cognitive Behavior Therapy; PLA = Placebo; PI = Paradoxical Intention; NA = Non Applicable; WL=Wait List

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