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PSYCHOLOGICAL AND PHARMACOLOGICAL APPROACHES TO TREATING INSOMNIA: CRITICAL ISSUES IN ASSESSING THEIR SEPARATE AND COMBINED EFFECTS

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ABSTRACT. *This paper reviews the efficacy of psychological and pharmacological therapies, singly and combined, for treating insomnia. Their clinical usefulness are discussed in terms of short- and long-term outcomes, patient's acceptance and adherence to treatment, and cost-benefits/effectiveness. Psychological interventions, mostly cognitive-behavioral in content, produce reliable and durable improvements of sleep patterns in about 60–80% of patients with chronic and primary insomnia. Pharmacotherapy, especially benzodiazepine receptor agents, are effective for short-term usage, but there is little evidence of sustained benefits after drug tapering. Hypnotic medications are not recommended as the sole intervention. Combined approaches have yielded more favorable short-term outcomes relative to drug therapy alone, but not necessarily superior to behavioral treatment alone. Long-term outcomes of combined approaches have been mixed, with no clear advantage to adding sleep medication to behavioral interventions. Additional research is needed to evaluate the effectiveness of integrated biobehavioral approaches, particularly multifaceted, sequential methods that might be more cost-effective than any single approach alone. The high costs associated with insomnia and its long-term consequences point to the benefits of early interventions, both in terms of maintaining quality of life and in reducing health care costs.*

INSOMNIA is among the most frequent health complaint brought to the attention of health care practitioners. Epidemiological surveys indicate that between 9% and 15%

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of the adult population complain of chronic insomnia, with an additional 15–20% reporting occasional trouble sleeping (Ford & Kamerow, 1989; Gallup Organization, 1991; Mellinger, Balter, & Uhlenhuth, 1985). Insomnia is more prevalent among women, older adults, and patients with medical or psychiatric disorders. Chronic insomnia is not a benign problem as it can adversely affect a person's life, causing substantial psychosocial, occupational, health, and economic repercussions. Individuals with chronic sleep disturbances display higher psychological distress, greater impairments of daytime functioning, are involved in more fatigue-related accidents, take more sick leave, and utilize health care resources more often than good sleepers. Persistent insomnia is also associated with increased risks of major depression and prolonged use of hypnotic medications (Becker, Brown, & Jamieson, 1991; Ford & Kamerow, 1989; Gallup Organization, 1991; Mellinger et al., 1985).

Despite its high prevalence and negative impact on quality of life and health care costs, insomnia is undertreated. Fewer than 15% of chronic insomnia patients have received any form of treatment (Mellinger et al., 1985), less than 5% ever consulted a physician specifically for sleep disturbance, and about 26% have talked to a physician about insomnia but only in the context of a visit for another problem (Gallup Organization, 1991). The vast majority of patients resort to a host of self-help remedies, of questionable values, and, when insomnia is brought to professional attention, usually a primary care physician, treatment is often limited to pharmacotherapy. Although health care professionals are receptive to nondrug therapies for insomnia, specific procedures other than general sleep hygiene advises, are not well known and infrequently used in medical settings (Rothenberg, 1992).

A Gallup survey commissioned by the National Sleep Foundation (Gallup Organization, 1991) reported that 20% of those who complained of insomnia had received a prescription for a sleeping pill in the past. In the NIMH survey of psychotherapeutic drug use, 7.1%, or 15% of those reporting serious insomnia, had used either a prescribed or over-the-counter sleeping aid within the previous year (Mellinger et al., 1985). Epidemiological data from European countries indicate much higher rates of hypnotic utilization, with between 24% and 40% of moderate and severe insomniacs using prescribed hypnotics habitually (Hohagen et al., 1993; Weyerer & Dilling, 1991). Older adults, particularly women, and those residing in nursing facilities, are much more likely to use sleeping pills. In addition, more than 50% of insomnia patients seeking treatment at sleep clinics are or have been on hypnotic medications in the past (Morin, Stone, McDonald, & Jones, 1994).

INSOMNIA COMPLAINTS AND DIAGNOSTIC CONSIDERATIONS

Insomnia is a fairly heterogeneous complaint reflecting impaired quality, duration, or efficiency of sleep. It can involve difficulties initiating sleep, trouble staying asleep, such as frequent or prolonged nocturnal awakenings, or early morning awakening with an inability to resume sleep. Difficulties initiating and maintaining sleep are not mutually exclusive, as the same person may suffer from sleep-onset, sleep-maintenance, or mixed onset and maintenance insomnia. Sometimes, the primary complaint involves nonrestorative sleep or, diminished sleep quality, resulting in daytime fatigue and low energy. Insomnia may be situational, a condition lasting a few days and often associated with stressful life-events, episodic, or evolve into more chronic sleep difficulties persisting for months or years.

According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994), 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. In treatment outcome research, insomnia is often operationalized as 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% (Lacks & Morin, 1992). The frequency and duration criteria are also more stringent, usually involving a minimum of 3 nights of disturbed sleep per week of more than 6 months duration.

Insomnia can also be associated with alcohol and drug abuse, medical, psychiatric, and other sleep disorders. There is a high comorbidity particularly between sleep disturbance and psychopathology. Various estimates suggest that between 35% and 44% of all patients presenting with a complaint of insomnia suffer from a psychiatric disorder, most frequently affective and anxiety disorders (Buysse et al., 1994; Morin & Ware, 1996). Treatment outcome research has predominantly focused on primary insomnia, with only a few studies investigating treatment efficacy for sleep difficulties associated with psychiatric disorders.

NONPHARMACOLOGICAL THERAPIES

Increasing recognition that psychological and behavioral factors play an important mediating role in insomnia has prompted the development of more than a dozen nonpharmacological interventions for this sleep disorder in the last 20 years. About half of those treatment modalities, which are described in several other sources (Espie, 1991; Hauri, 1993; Morin, 1993), have received adequate empirical evaluation of their clinical efficacy. They include stimulus control therapy, sleep restriction, relaxation-based interventions, cognitive therapy, and sleep hygiene education. These treatments 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. In this section we provide a brief description of treatment methods and their rationale, and an integrative summary of the main outcome findings, with a special emphasis on magnitude, durability, and clinical significance of changes in sleep patterns.

Description of Treatment Methods

Stimulus control therapy (Bootzin, Epstein, & Wood, 1991) consists of a set of instructional procedures which are: (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. The main objectives of stimulus control therapy is to reassociate temporal (bedtime) and environmental (bed and bedroom) stimuli with rapid sleep onset, by curtailing sleep-incompatible activities (overt and covert), and to enforce a more consistent circadian sleep-wake rhythm, by enforcing a strict adherence to a regular arising time and avoidance of daytime naps.

Sleep restriction therapy (Spielman, Saskin, & Thorpy, 1987) consists of curtailing the amount of time spent in bed to the actual 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 × 100%) exceeds 85–90%, decreased by the same amount of time when sleep efficiency is lower than 80%, and kept stable when sleep efficiency falls between 80% and 85%. Adjustments are made periodically until an optimal sleep duration is achieved. Poor sleepers often increase their time in bed in a misguided effort to provide more opportunity for sleep, a strategy that is more likely to result in fragmented and poor quality sleep. Sleep restriction creates a mild state of sleep deprivation and produces faster onset and more efficient sleep.

Relaxation therapies (Lichstein, 1988) are designed to alleviate excessive arousal, a key factor interfering with the initiation of sleep. Procedures such as progressive muscle relaxation, autogenic training, and EMG biofeedback focus primarily on somatic arousal (e.g., muscle tension), whereas attention-focusing procedures, such as imagery training, meditation, and thought stopping target cognitive arousal (e.g., intrusive thoughts).

Cognitive therapy for insomnia is designed to alter dysfunctional beliefs and attitudes about sleep. Specific treatment targets include unrealistic expectations, faulty causal attributions, amplification of the consequences of insomnia, and misconceptions about healthy sleep practices. These factors play an important mediating role in insomnia, particularly in exacerbating emotional arousal, performance anxiety, and learned helplessness as related to sleeplessness. Cognitive restructuring procedures, which are similar to those used for anxiety and depression, seek to identify dysfunctional cognitions and reframe them into more adaptive substitutes in order to short-circuit the vicious cycle of insomnia, emotional distress, and further sleep disturbances (Morin, 1993).

Sleep hygiene education is concerned with health practices (e.g., diet, exercise, substance use) and environmental factors (e.g., light, noise, temperature) that may be either detrimental or beneficial to sleep. Although these factors are rarely of sufficient severity to be the primary cause of insomnia, they may potentiate sleep difficulties caused by other factors. Sleep hygiene recommendations are usually incorporated with other treatment protocols to prevent or minimize interference from poor sleep hygiene. Several additional nonpharmacological treatment methods including hypnosis, acupuncture, electrosleep, and light therapy have been used for insomnia, but these methods have not received adequate empirical evaluation to draw conclusion about treatment efficacy.

Magnitude of Changes on Sleep Symptoms

More than 60 group outcome studies involving over 2,000 patients have evaluated the efficacy of nonpharmacological interventions for insomnia. Several authors have recently reviewed many of those studies (Lacks & Morin, 1992; Lichstein & Riedel, 1994; Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995) and the discussion here will be limited to providing an integrative summary of the main outcome findings. The large majority of controlled treatment studies have shown that behavioral treatment methods produce reliable changes in the sleep patterns of chronic insomniacs. The magnitude of these changes is greater than the effects produced by nonspecific factors (e.g., expectations, demand characteristics, self-monitoring) as controlled for by wait-list, attention, or placebo control conditions.

Two meta-analyses (Morin, Culbert, et al., 1994; Murtagh & Greenwood, 1995) 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. Analyses of other sleep parameters have also yielded reliable effect sizes falling in what is conventionally defined as moderate to large: 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 in percentile ranks, these data also indicate that the average treated insomnia patient sleep longer, awake less frequently and for shorter durations, and report higher sleep quality after treatment than the large majority (50–70%) of untreated control patients.

In terms of absolute changes over time, sleep-onset latency is reduced from an average of 60–65 minutes at baseline to about 35 minutes at posttreatment. Although fewer studies have targeted sleep-maintenance insomnia, similar results are obtained for the duration of awakenings, which is reduced from an average of 70 minutes at baseline to about 38 minutes following treatment. The number of awakenings, averaging less than two per night at baseline, is only reduced marginally at posttreatment. Total sleep time is increased by a modest 30 minutes, from 6 hours to 6.5 hours after treatment, but ratings of sleep quality are significantly enhanced with treatment. Thus, for the average hypothetical insomnia patient, treatment effects may be expected to reduce sleep onset latency from 60–65 minutes to 30–35 minutes, reduce the number of awakenings to one or less per night, and its duration to a little over half an hour, and increase total sleep time to about six and one half h per night. These results are averaged across all treatment modalities and, as such, represent a conservative estimate of treatment efficacy. However, they are based on subjective measures, from prospective daily sleep diaries, and are subject to the same limitations than self-monitoring in general.

When efficacy is examined across treatment modalities, stimulus control and sleep restriction therapies tend to produce better outcomes for either sleep onset (Espie, Lindsay, Brooks, Hood, & Turvey, 1989; Lacks, Bertelson, Gans, & Kunkel, 1983; Spielman et al., 1987) or sleep-maintenance insomnia (Friedman, Bliwise, Yesavage & Salom, 1991; Lacks, Bertelson, Sugerman, & Kunkel, 1983; Morin & Azrin, 1987, 1988; Schoicket, Bertelson, & Lacks, 1988). Their improvement rates average between 50% and 60%, and the absolute values of sleep onset latency and wake after sleep onset often fall below or near the 30-minute cut-off criterion used to define insomnia. Relaxation-based interventions, the most frequently used treatment for insomnia, also produce significant reductions of sleep onset latency, with methods targeting cognitive arousal (45%) yielding slightly better outcomes than those focusing on somatic arousal (40%). These results are comparable to those obtained with biofeedback (Hauri, 1981; Nicassio, Mendlowitz, Fussell, & Petras, 1985; Sanavio, 1988; Sanavio, Vidotto, Bettinardi, Roletto, & Zorzi, 1990). Sleep hygiene education, when used as the sole intervention, produces limited sleep improvements and lower satisfaction among patients (Schoicket et al., 1988). Formal cognitive therapy has not yet been evaluated as a single treatment modality. However, all studies that have incorporated cognitive restructuring procedures to multifaceted interventions and targeted dysfunctional sleep cognitions have reported positive results (Edinger, Hoelscher, Marsh, Lipper, & Ionescu-Pioggia, 1992; Morin, Kowatch, Barry, Walton, 1993; Morin, Stone, McDonald, & Jones, 1994; Sanavio 1988; Sanavio et al., 1990). Multi-component interventions have produced therapeutic gains comparable but not always superior to the most effective single approaches. The best outcomes from multifaceted interventions have been reported when sleep restriction and/or stimulus control procedures were integrated with other methods such as relaxation or cognitive restructuring methods (Jacobs, Benson, & Friedman, 1993; Sanavio et al., 1990). Thus, it may be that stimulus

control and sleep restriction procedures are the most active therapeutic ingredients (Murtagh & Greenwood, 1995).

An important reservation about psychological treatments for insomnia is that assessment of efficacy is too often limited to self-report measurement. Although subjective indices of sleep improvement are essential to document efficacy, it is also important to validate treatment effects with objective methods. Several recent studies have documented the effects of nonpharmacological treatment with polysomnography (Jacobs et al., 1993; Morin et al., 1993), wrist-actigraphy (Friedman et al., 1991; Guilleminault et al., 1995), and other behavioral devices (Espie et al., 1989; Morin & Azrin, 1988). In general, therapeutic gains documented with objective measurement methods are in the same direction as those reported on daily sleep diaries, although the magnitude of improvements are smaller. In one study for example (Morin et al., 1993), sleep diary data for a 2-week baseline period indicated a nightly average wake after sleep onset of 62 minutes that was reduced by 54% after treatment; polysomnographic data, based on 2 nights of EEG recording, showed a value of 73 minutes for the same variable at baseline that was reduced by 51% at posttreatment. Similar results have been reported by Jacobs et al. (1993) and, together, these findings indicate that psychological interventions produce, not only changes in sleep perception as reported on daily diaries, but also objective alterations of EEG sleep.

Although there is ample evidence to show that insomnia treatments produce therapeutic effects beyond those expected from nonspecific factors, there is little data on the specific mechanisms of change mediating treatment outcome. Current etiological models of insomnia point to the importance of reducing arousal, performance anxiety, excessive amounts of time spent in bed, as well as enhancing self-efficacy and more adaptive sleep cognitions (Lacks & Morin, 1992), but the few studies that have examined process-outcome relationships have failed to demonstrate a link between sleep improvements and the hypothesized mechanism of change. Nevertheless, some evidence suggest that stimulus control and sleep restriction procedures may be most effective for improving sleep efficiency, relaxation-based interventions to improve sleep quality, and cognitive restructuring to decrease emotional distress associated with sleep disturbance.

Durability of Changes

A consistent finding across studies is that behavioral treatment for insomnia produces stable therapeutic changes over time (Morin, Culbert, et al., 1994; Murtagh & Greenwood, 1995). The large majority of studies with follow-ups indicate that treatment-produced changes in sleep patterns are well maintained, and sometimes enhanced, at short- (3-month) and intermediate (6-month) range follow-ups. Because treatment is typically implemented in the context of relatively brief intervention programs (average of 6 treatment sessions), some patients may require more time to fully integrate the procedures. This may be particularly true for relaxation-based treatments (Espie, 1991). Despite robust long-term outcomes, follow-up data must be interpreted cautiously as there are still relatively few studies reporting long-term follow-ups and, among those that do, attrition rates increase substantially over time.

Clinical Significance

The clinical significance of insomnia treatment outcome has been examined by computing percentages of patients achieving a "meaningful" improvement, resuming a "normal" sleep pattern, or reducing/stopping hypnotic medications. A clinically meaningful improvement is usually defined by a 50% or greater improvement rate on the

main target symptom, with the absolute values falling near or below the 30-minute criteria used to define insomnia. In a clinical replication series of 100 patients seeking treatment at a sleep disorders clinic (Morin, Stone, et al., 1994), about half of the patients achieved a 50% or better improvement rate, and between 37% and 40% reached a dual criterion of clinical improvement (i.e., 50% reduction of their target symptom, with the absolute value of that symptom also falling below the 30-minute cut-off criterion). There were 38 patients whose sleep efficiency moved from a dysfunctional to a normative range. Also, the number of habitual users of sleep medications had decreased from 59 at baseline to 27 at posttreatment. In a reanalysis of seven outcome studies ($n = 216$), Lacks and Powlishta (1989), reported that 39% of treated subjects achieved reliable change, whereas 23% became good sleepers. At the 1-year follow-up, 49% showed reliable change, 32% became good sleepers, 76% were medication-free (compared to 35% at baseline), 63% had at least a 50% decrease in complaint, and 31% reported they no longer had insomnia. While these results suggest that behavioral interventions produce clinically significant outcome, the majority of treated insomniacs do not become good sleepers after treatment, and a small proportion of patients do not improve with treatment. Furthermore, little attention has been paid to the impact of treatment on other important variables that often prompt patients to seek treatment (i.e., daytime performance, fatigue, and quality of life).

Treatment Response and Moderating Variables

Several demographic, clinical, and treatment-related variables (format, delivery mode) have been examined as potential moderators of treatment response, but very few have been reliably associated with outcome. Age and gender are unrelated to outcome, although some evidence suggest that relaxation may be less effective with elderly insomniacs (Friedman et al., 1991; Lichstein & Johnson, 1993). Outcome is unrelated to the nature of insomnia (onset vs. maintenance), and the limited data on chronicity are equivocal. Clinically-referred patients achieve comparable and, perhaps, superior outcomes to research subjects solicited from the community (Chambers & Alexander, 1992; Espie et al., 1989; Morin, Stone, et al., 1994; Spielman et al., 1987). Although some reports (Morawetz, 1989; Morin & Azrin, 1988) indicate that medicated insomniacs do not respond to treatment as well as drug-free patients, increasing evidence suggest that cognitive-behavioral interventions may facilitate reduction or discontinuation of hypnotic medications when a systematic hypnotic withdrawal plan is integrated to treatment (Espie, Lindsay, & Brooks, 1988; Kirmil-Gray, Eagleston, Thoresen, & Zarcone, 1985; Lichstein & Johnson, 1993; Morin, Colecchi, Ling, & Sood, 1995). Very few studies have examined treatment response among patients with concurrent psychopathology; the limited data suggest that they may also respond to behavioral treatment, although the end-stage functioning on sleep parameters is still dysfunctional. Treatment provided by a professional therapist either, individually or in a group format, produce equivalent outcomes, which are more favorable relative to self-administered treatment without therapist guidance (Riedel, Lichstein, & Dwyer, 1995). Whether the duration, intensity, and integrity of treatment mediate these relationships is unclear. Studies using larger samples are needed to reach more definite conclusions about reliable predictors of treatment response.

PHARMACOLOGICAL THERAPIES

Since the introduction of bromides as sedatives in the 1850s there have been many classes of hypnotics prescribed. Most have been addictive (barbiturates, benzodiazepine

receptor agents, carbamates, chloral derivatives, ethanol, methaqualone, paraldehyde, piperidinediones) despite the recurrent hope that the agents will be free of habituating properties. Only the anticholinergic drugs, antihistamines and antidepressants are generally considered free from addiction potential, although this notion may not be entirely correct (Dilsaver, Feinberg, & Greden, 1983). Antipsychotics are also used for insomnia in patients with schizophrenia, bipolar disorder, and organic mental disorders, but are not widely used in the general population for the treatment of insomnia. The focus of this section will be on the use of antidepressants, antihistamines, benzodiazepine receptor agents (includes benzodiazepines and chemically unrelated sedatives with similar effects), chloral hydrate, and ethanol, as these appear to be the most clinically current sedatives.

Antidepressants

The most commonly used antidepressants with sedative effects are the tricyclic antidepressants (TCAs) and trazodone. The TCAs have anticholinergic and antihistaminic effects in common and may have serotonin, norepinephrine, or both, neurotransmitter effects (Richelson, 1994). The most sedating TCAs are amitriptyline, trimipramine, clomipramine, and doxepin (Richelson, 1994; Ware & Morewitz, 1991). These agents have potent sedative effects at doses well below those needed for antidepressant action, whereas other agents such as nortriptyline and imipramine have sedative effects at about the therapeutic dose. Also the sleep induction effect tend to occur on the first night of use, whereas the antidepressant effects occur 2–3 weeks later. Nearly all temporarily suppress REM sleep, although trimipramine has little effect on REM sleep (Ware & Morewitz, 1991; Ware & Pittard, 1990). The antihistaminic and anticholinergic effects closely parallel the sedative effects of these drugs. These side effects often lead to impairment in cognition (Hartmann & Cravens, 1973a) and to a variety of somatic effects including blurred vision, orthostatic hypertension, sexual dysfunction, dry mouth, and constipation. These and the cardiovascular side effects often make the TCAs poor choices for the treatment of insomnia, especially in the elderly, or in various medical conditions (e.g., glaucoma, dysautonomias, cardiac conduction defects, prostate hypertrophy; Richelson, 1994). The toxicity of TCAs also can provide depressed patients an opportunity to complete suicide by overdose. Nevertheless these drugs are helpful to some patients, especially when depression or pain is part of the insomnia problem. Clomipramine may also have benefits in treating obsessive insomniacs, especially when other serotonin reuptake inhibitors are not tolerated or aggravate insomnia.

Clinical trials of TCAs in nondepressed insomniac subjects are few, therefore the effectiveness of TCAs when used as hypnotics is uncertain. Amitriptyline reduces sleep onset latency, increases total sleep time, and reduces REM sleep, but with rapid tolerance development (Hartmann & Cravens, 1973a). It is likely that the TCAs cause the same impairments observed with antihistamine agents, including daytime hangover effects and cognitive impairment (see below).

Trazodone, a nontricyclic antidepressant, has gained popularity as a sedative drug, especially to offset insomnia associated with stimulating non-TCAs (Metz & Shader, 1990; Nierenberg, Adler, Peselow, Zornberg, & Rosenthal, 1994). It is relatively nontoxic, even when taken in an overdose (Richelson, 1994). The few studies of trazodone for chronic insomnia alone (Montgomery, Oswald, Morgan, & Adam, 1983; Ware & Pittard, 1990) show increased slow wave sleep, reduced REM sleep and increased REM sleep onset latency. Studies of depressed patients show increased total

sleep time, decreased sleep onset latency, increased slow wave sleep, increased REM sleep onset latency and reduced awakenings (Mouret, Lemoine, Minuit, Benkelfat, & Renardet, 1988; Sharf & Sachais, 1990; van Bemmel, Havermans, & van Diest, 1992). Nefazodone, which has been recently released and is related to trazodone, may be a useful alternative without the risk of priapism. This agent appears to improve sleep continuity and does not alter REM sleep as observed with most of the TCAs.

Antihistamines

Antihistamines have long been used as sedatives, both by prescription and over the counter (e.g., Sominex, Unisom, Sleep-Eze). In comparison to benzodiazepines, antihistamines used as hypnotics are more likely to cause morning drowsiness and residual psychomotor impairment (Balter & Uhlenhuth, 1991, 1992; Rickels et al., 1983). Antihistamines have a rapid onset of action and variable effects on the number of awakenings and total sleep time. They seem to be most effective in patients who have had no previous exposure to sedative hypnotics (Kudo & Kurihara, 1990). Studies have also indicated rapid tolerance to the hypnotic effects following repetitive use (Manning, Scandale, Manning, & Gengo, 1992; Mattila, Mattila, & Konno, 1986; Nicholson, 1979). Studies evaluating the effects of antihistamines on alertness the morning after bedtime administration (Balter & Uhlenhuth, 1992; Rickels et al., 1983), and after daytime administration (Mattila et al., 1986; Nicholson & Stone, 1986; Rice & Snyder, 1993; Roth, Roehrs, Koshorek, Sicklesteel, & Zorick, 1987) show significant sleepiness, cognitive and psychomotor impairments. As with other sedatives, subjects may experience cognitive and psychomotor impairment without any awareness of the decrements (Seidel, Cohen, Bliwise, & Dement, 1987). While antihistamines do not cause physical dependence, serious potential problems such as dyskinesia and lowering of seizure threshold exists (Meltzer, 1990). Patient satisfaction with antihistamines compared to benzodiazepines is poor (Balter & Uhlenhuth, 1991).

Benzodiazepines Receptor Agents

Benzodiazepines receptor agents (BRAs) are now the most commonly used group of prescription sedative hypnotics. They are effective sedatives and present a lower risk of physical dependence and lethal overdose than older sedatives with addictive potential (American Psychiatric Association, 1990). The BRAs include the benzodiazepines (e.g., flurazepam, triazolam, temazepam, quazepam, estazolam), and newer agents that are not chemically benzodiazepines, but that have activity on benzodiazepine receptor subtypes (zolpidem), or on the related $GABA_A$ complex (zopiclone). The therapeutic, abuse potential, and side effect profile is comparable among these agents, although differences in onset of action and duration of action give relative advantages to the rapid onset, short-to-intermediate duration BRAs (e.g., triazolam, zolpidem, zopiclone, estazolam). At the time of this writing, zolpidem and zopiclone also have the minor distinction of producing little documented change in sleep architecture, or rebound insomnia (Hoehns & Perry, 1993; Langtry & Benfield, 1990; Wadworth & McTavish, 1993).

All BRAs improve sleep continuity and efficiency through a reduction of sleep onset latency and time awake after sleep onset. They also increase total sleep time and reduce the number of awakenings and stage shifts through the night. Their effects on sleep stages vary with the specific class of medications. All BRAs increase stage 1 and stage 2 sleep. Benzodiazepines tend to suppress slow-wave (stages 3–4) sleep and, in some cases, REM sleep is also reduced. BRAs are better sedatives than other previously

prescribed compounds (e.g., barbiturates) with longer effectiveness (Kales, Kales, Bixler, & Scharf, 1975). All BRAs have been reported to impair cognition, memory and psychomotor function (Evans, Funderburk, & Griffith, 1990; Jonas, Coleman, Sheridan, & Kalinske, 1992; Wadworth & McTavish, 1993). Shorter acting BRAs such as triazolam, midazolam, brotizolam, zopiclone, and zolpidem typically result in high next-day patient satisfaction, and are largely free from next day effects when used at the appropriate doses (Jonas, Coleman, Sheridan, & Kalinske, 1992). Long acting benzodiazepines such as flurazepam, quazepam and flunitrazepam, although working rapidly, tend to accumulate, leading to more psychomotor and cognitive impairment, especially in the elderly (Carskadon, Seidel, Greenblatt, & Dement, 1982; Moskowitz, Linnoila, & Roehrs, 1990; Roehrs, Kribbs, Zorick & Roth, 1986; Roth & Roehrs, 1991). Recent data suggest that long acting benzodiazepines result in an increased rate of hip fractures in the elderly (Ray, 1992). Benzodiazepines can cause respiratory depression. While some studies show little effect of the BRAs in mild to moderate respiratory disorders, the BRAs can cause worsening of sleep apnea, and are more prone to worsen respiration in severe COPD (Cirignotta et al., 1988; Dolly & Block, 1982; Mendelson, Garnet & Gillin, 1981).

The phenomenon of rebound insomnia has received much attention in comparative clinical trials, but its clinical relevance is equivocal when BRAs are prescribed and taken appropriately (Balter & Uhlenhuth, 1992; Jonas et al., 1992; Schneider-Helmert, 1988). Anterograde amnesia has also been reported to occur more frequently with shorter acting sedatives (Jonas et al., 1992; Wysowski & Barash, 1991), although it appears to occur with long acting agents as well (American Psychiatric Association, 1990). An interesting observation is that untreated insomniacs also have a high rate of anterograde amnesia (Balter & Uhlenhuth, 1991).

Recent public and scientific controversy about abuse potential and abnormal behavior with benzodiazepines, especially triazolam, has led to polarized views about the role of sedative hypnotics in the treatment of insomnia (Balter & Uhlenhuth, 1992; Cowley, Springen, Iarovici, & Hager, 1991; Jonas, Coleman, Sheridan, & Kalinske, 1992; Regestein & Reich, 1985; Roehrs, Vogel, & Roth, 1990; Weedle, Poston, & Parish, 1988; Wysowski & Barash, 1991). Analysis of the various reports suggest that excessive doses, combining hypnotics with alcohol and other drugs, as well as usage in patients of advanced age, are responsible for most of these adverse events.

Chloral Hydrate

Chloral hydrate is also addictive and its therapeutic benefits are more variable when compared to benzodiazepines. It loses its effectiveness within 1–2 weeks (Hartmann & Cravens, 1973b; Kales, Allen, Scharf, & Kales, 1970). It is rapidly absorbed and it has no discernible effects on sleep stages at low doses (Hartmann & Cravens, 1973b), but at higher doses, it may suppress REM sleep. Drug hangover effects can occur the next day after chloral hydrate administration (Goldstein, Birnbom, Lancee, & Darke, 1978). A lethal dose of chloral hydrate is only about 10 times a therapeutic dose.

Ethanol

Ethanol is the most frequently self-prescribed sedative agent. Sometimes, uninformed physicians recommend ethanol as a nightcap to help their patients sleep. Although alcohol may help tensed insomniacs fall asleep faster, it usually causes frequent and prolonged nocturnal awakenings in the second part of the night. Ethanol also sup-

presses REM sleep in the early part of the night and is followed by a rebound effect in the second half of the night (Pokorny, 1979). In severe cases of alcohol dependence, withdrawal results in severe REM sleep rebound with hypnagogic hallucinations. There is some suggestion from alcoholics themselves that 60% of them use alcohol to self treat their insomnia (Mamdani, Hollyfield, Ravi, Dorus, & Borge, 1988). Although sedative hypnotics are among the highest group of alternate substances of abuse in the elderly, alcohol abuse clearly outstrips prescription sedative hypnotic abuse (Finlayson & Davis, 1994; Miller, Bekin, & Gold, 1991). Recidivism and alcoholism are highest in patients previously hospitalized for sedative hypnotic abuse (Allgulander, Borg, & Vikander, 1984).

Prescribing Trends

Over the last three decades there have been shifts in prescribing patterns in the medical community (Wysowski & Baum, 1991). Prescribing patterns are more conservative nowadays than two or three decades ago, and the specific agents used in pharmacotherapy have also changed over time. Most physicians have switched from older drugs such as the barbiturates and nonbenzodiazepine hypnotics to short- and intermediate-acting benzodiazepines. BRAs offer safer and more effective alternatives. Within the group of BRAs, longer acting benzodiazepines are more prone to cause cumulative side effects impairing cognition and increasing accidents. However, regardless of the apparent overall shift in prescribing toward more efficacious, safe and effective medications, nonpsychiatrist physicians may inappropriately prescribe sedatives (Schorr & Bauwens, 1992).

Media dramatization of adverse reactions and addiction to Halcion (e.g., Cowley et al., 1991), coupled with scientific literature expressing concern about Halcion s side effects (Wysowski & Barash, 1991), led to changes in prescribing patterns, and regulatory interventions by federal and state governments (Shader, Greenblatt, & Balter, 1991; Wysowski & Baum, 1991). In New York State, for example, stricter and more expensive prescribing requirements for benzodiazepines led to a marked increase in prescribing of older, less effective, and more dangerous sedative hypnotics (Shader, Greenblatt, & Balter, 1991; Weintraub, Singh, Byrne, Maharaj, & Guttmacher, 1991). Thus, despite the development of new and better sedatives, the influence of the media and government may be interfering with the much needed education of nonpsychiatrist physicians toward more appropriate prescribing patterns. Until the controversies are moderated, care quality and cost of treatment will continue to be adversely impacted. It is apparent, however, that the use of hypnotic agents is highly prevalent when all classes of substances are combined. When judging from surveys from the public and physician prescribing patterns, there is a substantial amount of inappropriate, and expensive usage (Gallup Organization, 1991; Schorr & Bauwens, 1993; Walsh, Englehardt, & Hartman, 1995; Willcox, Himmelstein, & Woodhandler, 1994; Wysowski & Baum, 1991; Zorc, Larson, Lyons, & Beardsley, 1991).

In summary, when looking back historically to the first hypnotics, great strides have been made in the pharmacological management of insomnia. The newest agents are tailored to work more rapidly, are comparatively safe, affect sleep parameters little, and when used appropriately, have few daytime sequelae. Agents such as antihistamines and chloral hydrate are largely ineffective for chronic insomnia, and ethanol, despite its prevalent use as a sleep aid, is a poor and hazardous choice. And while BRAs may provide comparable therapeutic effects, most patients will eventually develop tolerance to the sedative and anxiolytic effects providing the benefits to sleep. In

addition, all sedatives developed thus far have adverse effects on cognition, psychomotor skills, and mood, especially in those predisposed due to age or illness, and all effective sedatives carry some risk of dependence. The complaint of insomnia may shroud either a single cause, or a number of underlying physical and/or psychological causes which must be addressed if insomnia is to be treated effectively. It would be simplistic to think that any sedative could achieve complete control of insomnia; it appears that the best role for sedatives is as an adjunct to behavioral interventions.

COMPARATIVE STUDIES OF SINGLE AND COMBINED TREATMENT APPROACHES

Despite the extensive literature on the efficacy of behavioral and pharmacological therapies, only 5 studies have directly compared the separate and combined effects of those treatment modalities within the same study design. The first published study (McClusky et al., 1991) compared a 3-week regimen of triazolam, 0.5 mg used nightly, to a behavioral approach combining stimulus control and relaxation training. The sample consisted of 30 young adults (mean age = 32 years) with primary and chronic sleep-onset insomnia (mean baseline sleep onset latency = 81 minutes). Both treatments resulted in equivalent reductions of sleep onset latency at the end of treatment (mean of 36 minutes per night), but the patterns of change over time were different for the two conditions. Subjects receiving triazolam were significantly more improved after the first week of treatment, whereas behaviorally-treated subjects sustained greater benefits at the 1-month follow-up after drug tapering.

In a subsequent study (Milby et al., 1993), the same group of investigators examined the differential effectiveness of triazolam (0.25 mg nightly), alone and combined with stimulus control and relaxation training. The sample consisted of 15 sleep-onset insomniacs and the same design and treatment duration as in the previous study were used. 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. Despite the relatively small sample, these findings suggest that a combined intervention is superior to drug therapy alone in sustaining therapeutic gains.

Lewin et al (1994) investigated the comparative efficacy of drug therapy (estazolam, 0.5-1.0 mg) plus information about sleep physiology, to its combined effects with either muscle relaxation or imagery training. Thirty two middle-aged subjects (mean age = 47.2 years) with primary insomnia were administered one of those conditions in seven group therapy sessions conducted over a 4-week period. During the initial treatment period, the two conditions combining drug with somatic or mental relaxation improved on measures of sleep efficiency and sleep duration but the drug therapy alone condition did not. Although follow-up data were provided, these results were probably confounded by the provision of additional treatment (i.e., sleep hygiene and a self-help book on insomnia treatment) to all patients after the initial intervention phase.

Hauri and Wisbey (1993) isolated the psychological rather than pharmacological treatment component. They compared sleep hygiene education and relaxation training, alone or in combination with an occasional hypnotic drug (triazolam, no more than once per week), to a wait-list control condition. Polysomnographic data showed that sleep efficiency and total sleep time were more improved in the two active treatment conditions than in the control group, but there was no differential treatment effects. At the 10-month follow-up assessment, subjects treated with the behavioral approach alone were sleeping better than those who had received the combined

intervention. These findings might suggest that subjects treated with a combined intervention may attribute their initial therapeutic gains to the drug alone and become more vulnerable to relapse when medication is discontinued.

Morin and colleagues (Morin, Colecchi, Stone, Sood, & Brink, 1995) conducted a placebo-controlled study of cognitive-behavior therapy and pharmacotherapy (temazepam, 7.5-30 mg), singly or combined, for late-life insomnia. The sample consisted of 78 older adults (mean age = 65 years) with primary and chronic insomnia, suffering predominantly from sleep-maintenance or mixed onset and maintenance difficulties. The findings showed that all three active treatments were more effective than drug-placebo at mid (4 weeks) and late treatment (8 weeks) on the two main outcome measures of time awake after sleep onset and sleep efficiency. Although there was a slight trend for the combined approach to yield better outcomes, no statistically significant differences emerged among the active treatment conditions at posttreatment. These results were documented with both self-report data from daily sleep diaries and EEG-defined sleep measures from nocturnal polysomnography. Follow-up data obtained at 3, 12, and 24 months after treatment, showed that subjects treated with cognitive-behavior therapy sustained their clinical gains, whereas those treated with drug therapy alone did not. Long-term effects of the combined intervention showed a significant loss of therapeutic benefits from posttreatment to follow-ups, although there was much variability across subjects in that condition and over the three follow-up periods.

Collectively, the data from comparative studies of behavioral and pharmacological treatments suggest that both treatments are effective in the short-term. Drug therapy may yield quicker results and a combined intervention a slightly better outcome. Long-term effects are not as clear as the short-term ones. Behavioral treatments produce more durable sleep improvements than drug therapy alone, whereas combined interventions yielded more variable results across patients and over time.

CRITICAL ISSUES IN ASSESSING EFFICACY AND USEFULNESS OF TREATMENT

Several factors must be considered in judging the relative efficacy and clinical usefulness of psychological and pharmacological therapies for insomnia. In this section, we discuss some of those issues, including the short-term benefits and quickness of action, long-term effects and relapse rates, treatment acceptability and patient adherence, and cost-effectiveness factors.

Trajectory of Changes

Short-term efficacy and quickness of action. Most hypnotic medications are effective in the short-term management of insomnia. They have a quick onset of action, often producing significant therapeutic benefits upon the very first night of usage. These benefits usually last for several nights and, in some cases, up to a few weeks. Their main drawbacks include some side effects and the tolerance that develops with most agents when used nightly. Behavioral interventions produce substantial benefits during the initial 4–6 week treatment period, with between 60% and 80% of treated patients improving more than untreated control subjects, and about half reaching a clinically meaningful stage of improvement. Drug therapy produces more rapid improvement in sleep patterns relative to behavioral interventions, especially those that require learning new skills such as relaxation training. One exception may be sleep restriction, a particularly potent intervention, producing important changes in sleep efficiency as early as the first week of treatment in older insomniacs (Morin et al., 1995).

Despite valuable therapeutic benefits produced by both behavioral and pharmacological approaches, it is clear that neither approach is effective for all subtypes of insomnia patients.

Long-term effects. Although behavioral interventions require more time to improve sleep patterns, these changes are fairly durable over follow-up periods averaging six to eight months in duration (Morin et al., 1994; Murtagh & Greenwood, 1995). Also, initial improvements may be further enhanced after treatment completion. Such delayed therapeutic effect is more common when the treatment is implemented over a short period of time (e.g., 2–4 weeks) and when it involves relaxation procedures. It may be that patients do not have sufficient time to fully integrate newly learned skills. Despite the fairly robust long-term outcomes with nonpharmacological interventions, several studies still limit their follow-up assessments to very brief intervals, often not exceeding one month, and some still fail to provide any data on maintenance of therapeutic gains.

There is a paucity of follow-up data in controlled trials of hypnotic medications, but the limited empirical evidence suggests that most hypnotics lose their effectiveness when used nightly over prolonged periods of time. As drug therapy is recommended only for acute and situational insomnia (NIH, 1984; 1991), a more appropriate question may be: When used on a short-term basis, are there lasting benefits from drug treatments after their discontinuation? Unfortunately, little research has examined this issue, perhaps, because there is a widespread clinical assumption that medication will not be needed for a prolonged duration if insomnia is indeed situational. Nevertheless, when insomnia is recurrent or persistent, it is unlikely that a short-term trial of hypnotic drug alone will produce lasting benefits. Three of the four comparative studies that have isolated the drug therapy component and reported follow-up data have shown that insomnia symptoms often return to baseline level after drug discontinuation (McClusky et al., 1991; Milby et al., 1993; Morin et al., 1995).

Mechanisms of changes. The hypothesized mechanisms of changes with drug and nondrug therapies vary according to the specific treatment. In general, pharmacotherapy works presumably by altering the neurochemistry of sleep, whereas psychological interventions seek to reduce arousal and modify maladaptive sleep habits and dysfunctional cognitions. On a clinical basis, it might be argued that an integrated biobehavioral intervention should optimize treatment outcome by capitalizing on the more immediate and potent effects of drug therapy and the more sustained effects of psychological interventions. Empirically, however, it remains unclear whether a combined intervention has an additive or a substractive effect on outcome. Studies with short-term follow-ups (< 1 month) indicate that a combined intervention produces more sustained benefits than with drug therapy alone (McClusky et al., 1991; Milby et al., 1993), whereas investigations with follow-ups of six months or longer durations find much more variable long-term outcomes among patients receiving a combined intervention relative to those treated with a behavioral treatment modality alone (Hauri & Wisbey, 1993; Lewin et al., 1994; Morin et al., 1995). In light of the mediating role of psychological factors in chronic primary insomnia, behavioral and attitudinal changes appear essential to sustain improvements in sleep patterns, even when integrating behavioral and drug therapies. Subjects' attribution of the initial clinical benefits may be critical in determining the subsequent likelihood of relapse. For instance, attribution of the initial therapeutic benefits to the drug alone, without integration of self-management skills, may place a patient at significantly greater risk for relapse once the

drug is discontinued. Additional research is needed to examine more systematically potential mechanisms of changes mediating short- and long-term outcomes.

Patient's Acceptance of and Adherence With Treatment

Patient's acceptance of and their adherence to alternative treatments are important factors in judging their clinical usefulness. Treatment acceptance by prospective patients is an important variable in seeking, initiating and adhering to treatment recommendations. Even though a particular intervention is efficacious, if it produces adverse side effects, is too time consuming, or too costly, adherence is likely to be poor. Thus, regardless of how efficacious a given treatment is, if it is not acceptable to patients, it may be of little clinical use. The limited data on this issue suggest that when insomnia patients are given the opportunity to chose between an active or a placebo pill they prefer the active medication (Pedrosi, Roehrs, Stepanski, Zorick, & Roth, 1993). However, if given the option to chose between a behavioral and a pharmacological treatment, the first option is perceived as more acceptable and more suitable than pharmacotherapy (Morin, Gaulier, Barry, & Kowatch, 1992).

A related issue is concerned with treatment adherence. Although patients may find a particular treatment more attractive and express the intent to comply with it, there is no guarantee that they will remain in therapy and that treatment will be carried out or implemented as recommended. In general, drug therapies are more easily and more quickly implemented than behavioral interventions which, despite their relative brief durations, require considerable investment of time and effort from both the patient and therapist. As such, compliance may be the most critical factor mediating success of behavioral interventions (Chambers, 1992). Nonetheless, drop out rates tend to be higher among subjects treated with drug therapy alone, relative to behavioral treatment or a combined approach (Morin et al., 1995). Clearly, a great deal more research is needed to examine the relationship of treatment acceptance and adherence to outcome, especially with clinical patients seeking treatment in various settings (family practice, outpatient clinics, sleep clinics). Meanwhile, it may be important to match patients, not only to the most efficacious treatment, but also to the most acceptable one that is likely to be implemented as intended.

Cost-Effectiveness Analysis

The cost associated with alternative treatment methods has become a key factor in guiding the decision-making process of policy makers in government, health care insurance industry, and research funding agencies. It is no longer sufficient to demonstrate that a treatment works, it must also be accessible and affordable. Some eloquent models to assess the relationship of cost-effectiveness and cost-benefits to outcomes have been proposed (Yates, 1994), but these equations have not yet been applied to sleep disorders in a prospective empirical design. The direct costs associated with insomnia refers to the treatment resources consumed, such as professional time for consultation and therapy, costs of medications, office spaces, and patient's time off from work, transportation, etc. The indirect costs, which are more difficult to quantify, include those associated with the consequences of insomnia, such as diminished productivity, sick leave from work, utilization of health care resources for insomnia and related problems, and diminished quality of life.

The National Commission of Sleep Disorders Research (NCSDR, 1993; Stoller, 1994; Walsh et al., 1995) estimated that American people pay over one billion dollars annually for the pharmacolological treatment of insomnia. This amount included

about $392 million dollars for prescription drugs to induce sleep, $84 million for over-the-counter sleep aids, and another $566 million for alcohol used as a sleep aid. The annual expenses associated with outpatient visits to physicians and mental-health professionals for insomnia was estimated at close to $600 million, including $474 million for physicians, $66 million for doctoral level psychologists, $41 million for social workers, and $9 million for sleep specialists. Individuals with insomnia are generally more preoccupied with their health and tend to use health-related services more frequently than good sleepers, taxing further the health care system. Indirect costs from sick days, diminished productivity, and even industrial and motor vehicle accidents resulting from untreated sleep disturbances are tremendous, and these expenditures do not even consider the more difficult to quantify costs associated with the impact of chronic insomnia on quality of life.

These figures illustrate the enormous socioeconomic impact associated with insomnia, both with its treatment or lack thereof. Although there has been no systematic analysis of cost-benefit or cost-effectiveness for either behavioral or pharmacological therapies, a hypothetical case might illustrate the economic savings of early interventions. About two thirds of all those who complain of insomnia suffer from situational sleep difficulties. Assuming that these difficulties are situational and not recurring over time, a short-term hypnotic trial would seem fairly inexpensive and, perhaps, the most cost-effective intervention. However, for those with recurring episodes of insomnia, the cost of pharmacotherapy could escalate rapidly, with a corresponding reduction of efficacy. For chronic insomnia, drug therapy alone is not recommended (NIH, 1984), as it does not address the underlying etiological and perpetuating factors. Behavioral and educational interventions that directly target these factors seem essential to resolve chronic sleep difficulties. With individual therapy time averaging about 6 hours per patient (Morin, Culbert, & Schwartz, 1994; Morin, Stone, et al., 1994), direct professional treatment costs might be estimated at about $500. These costs could be further reduced by providing treatment in a group rather than individual format, two modalities that have produced equivalent outcomes. If insomnia is effectively treated, as might be expected in 60% to 80% of the cases, treatment could reduce the direct costs associated with hypnotics drugs and the indirect costs associated with decreased productivity, leave time from work, and utilization of other health services. Thus, behavioral interventions, which may cost more in the short-term, may also prove in the long run to be the most cost-effective approach currently available for the management of chronic insomnia. While this hypothesis obviously await empirical testing through prospective and longitudinal studies, it is clear that the overall costs and consequences of not treating insomnia are much greater than the costs of treatment options currently available (Chilcott & Shapiro, 1996).

SUMMARY AND RECOMMENDATIONS

Controlled trials of psychological and pharmacological therapies have proceeded along two separate lines, often using different research design, clinical samples, and methods for assessing outcomes. There has been little effort at integrating these two approaches, with less than half a dozen studies having directly compared their separate and combined effects. Nevertheless, the available empirical evidence indicates that pharmacotherapy is effective for the short-term treatment of situational and acute insomnia, but is generally not recommended as the sole intervention for chronic insomnia. Psychological interventions, mostly cognitive-behavioral, are effective for persistent insomnia, but there has been no controlled evaluation of their efficacy for

acute insomnia. In terms of trajectory of changes, drug therapy produces acute changes in sleep patterns, but initial improvements may be offset by side effects, daytime residual impairments, and risk of dependence. Also, there is little evidence that these benefits are maintained over time. Behavioral interventions are more time consuming and take longer to produce therapeutic benefits. However, these gains are fairly durable over time. Combined approaches have yielded either equivalent or slightly better short-term outcomes to either form of therapy alone. Long-term effects have been mixed. Although integrated approaches are preferable to drug therapy alone, it is yet unclear whether the addition of sleep medications to behavioral treatment enhances outcome.

Both drug and nondrug treatment modalities have benefits and shortcomings, and neither approach is effective for all types of insomnia patients. Some patients either fail to respond to treatment, regardless of its nature, and the majority of responders do not reach a normative sleep patterns after treatment. Additional research is needed to design and evaluate more efficient models integrating biobehavioral approaches, using multifaced, sequential, or maintenance therapies. For example, to take full advantages of the quicker results from drug therapy and the more sustained effects of behavioral interventions, a sequential approach might be preferable to a combined approach. Unlike the combined method, where both treatments are initiated and discontinued at the same time, in a sequential approach, drug treatment is initiated first and gradually discontinued while the behavioral intervention is implemented concurrently. This method would ensure that patients are still in treatment after drug tapering, giving them the opportunity to fully integrate newly learned self-management skills, especially at a time when rebound insomnia is likely to reinforce the belief that medication is needed indefinitely. Additional research is also warranted to evaluate the long-term efficacy of maintenance therapies involving nightly low-dose, or occasional hypnotic use. These approaches are, perhaps, best suited for patients unresponsive to behavioral treatment, for those more vulnerable to relapse following drug discontinuation, or for individuals with situational but recurring insomnia.

Despite the widespread prevalence of insomnia, there is still little clinical attention devoted to its treatment. Less than 15% of chronic insomniacs are treated (Mellinger et al., 1985) and, when treatment is initiated, pharmacotherapy is often the only treatment recommended. Nonpharmacological interventions are clearly under-utilized by health care practitioners, although patients are usually more receptive to such interventions than to pharmacotherapy. A recent NIH panel recommended wider acceptance and broader use of behavioral therapies for treating insomnia, as well as greater integration of these procedures with more conventional medical treatment (NIH, 1995). The panel identified a number of barriers that have limited wider acceptance and use of behavioral interventions. Two of those were the general emphasis in treating these conditions strictly as medical conditions without considering the psychosocial components and the reluctance of insurance companies and other third party payers to reimburse for psychosocial interventions for insomnia at rates comparable to standard medical care. The lack of formal training in recognizing and treating sleep disorders, as well as the more time-consuming nature of psychosocial interventions, might represent two additional deterrents to using behavioral interventions in the clinical management of insomnia.

To promote wider use of behavioral treatments for insomnia, it will be essential to educate policy makers and health care professionals in recognizing insomnia as a real and costly health problem. Greater dissemination of empirically-validated treatment protocols through formal training courses in medical and graduate schools curriculum

is also warranted. Further research is also needed to develop more cost-effective approaches for both situational and chronic insomnia. Greater use of group therapy (Davies, 1989) is particularly indicated as treatment outcome is often equivalent to individualized therapies. Minimal interventions provided in the forms of brief consultations (Hauri, 1993), bibliotherapy, or educational audio (Morawetz, 1989) and video cassettes (Riedel et al., 1995) may also prove useful in some cases, although additional outcome evaluations are needed to evaluate their effectiveness. From a cost-effectiveness perspective, clinicians might use a stepwise approach from the most easily administered and less costly therapy, with the more timely and costly interventions introduced later if patient is unresponsive to the initial approach. In light of recent findings that insomnia is often a recurrent problem (Vollrath, Wicki, & Angst, 1989), and is associated with increased utilization of health services (Mellinger et al., 1985) and with increased risk of depression (Ford & Kamerow, 1989), early interventions may prove particularly cost-beneficial, both in maintaining adequate quality of life and in reducing health care costs.

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