A COMPARISON OF DRUG TREATMENT FOR INSOMNIA
AND THE EFFECT OF CAUSAL ATTRAIBUTION

DISSERTATION

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By

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A double-blind comparison was conducted using typical doses of soporific agents from three drug classes and a placebo. Drugs which were used in the study included secobarbital, flurazepam hydrochloride, and thioridazine. Subjects were 40 outpatient volunteers whose primary complaint was difficulty in falling to sleep. Subjects were randomly assigned to one of the three drug groups or the placebo group. One of the drugs or the placebo was administered to each subject for 3 nights. Half of the subjects in each of the four groups were told the drug had caused any observed changes in their sleep behavior and were in this way led to attribute any changed sleep behavior externally to the drug. The other half were told the drugs were not typically used to treat insomnia and changes in their sleep were due to changes made in their own behavior, thus attributing any changes in sleep behavior internally. Latency to sleep in minutes was measured by self-report for a total of 10 days, 3 days during and 7 days subsequent to drug administration. Additionally, subjects were asked to report from memory latency to sleep for 10 days prior to participation in the study. A randomized-block-factorial analysis of variance
was performed on the latencies to sleep during the pre-, post-, and treatment periods. Results indicated that there were reductions in latency with no significant differences among any of the groups ($p > .05$). Additionally, the decreases in latency were maintained regardless of the subjects' attributions about why the change occurred. This is contrary to other research which had shown that behavior changes which were internally attributed were maintained longer than externally attributed behavior changes. Reduction in latency to sleep was significantly greater during drug administration than pre- or post-treatment periods ($p < .01$) and the reduction was maintained during posttreatment as compared to preexperimental levels ($p < .05$).

Previous research reviewed seemed to indicate these discrepant results were due to the additional attributional factors of expectation regarding therapy and level of justification for participation in therapy. Expectancy of improvement of latency was maximized because the research was conducted in a location similar to physicians' offices where in the past symptom relief was likely through medication and because subjects were told initially that the medication would be likely to decrease sleep latency. Level of justification was minimized in the study because subjects participated with promise of no reward except possible reduction in latency to sleep. Previous research had paid subjects to participate.
The implication for clinicians was that a short course of drug therapy using a placebo or one of several soporific drugs might be used equally effectively to treat primary latency insomnia. Additionally, the results demonstrated that clinicians might expect the effectiveness of treatment to be maintained following treatment. Recommendations included a suggestion for future research with soporific drugs in other classes. The recommendation was also made that future research more carefully control the attributional factors of expectation and justification.
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### Dissertation

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A COMPARISON OF DRUG TREATMENT FOR INSOMNIA

AND THE EFFECT OF CAUSAL ATTRIBUTION

Insomnia has been one of the most frequently encountered complaints in medical practice and one of the most difficult disorders to permanently dissipate with current techniques (Smith, Coletta, McBride, & McPeek, 1974; Zimmerman, 1971). The overall prevalence of insomnia was indicated in a large-scale study done by the United States Department of Health, Education, and Welfare (1970) which reported that about 32% of all adults were insomniacs. Other reports using smaller numbers of subjects indicated the rate was between 14 and 25% (Luce & Segal, 1969; Montgomery, Perkin, & Wise, 1975).

An insomniac was commonly defined as anyone who complains of too little sleep (Dement, 1974). There has been a lack of active research on insomnia (Rechtschaffen & Monroe, 1969). Some past research has sought to establish the parameters involved in diagnosis and subsequent treatment of insomnia. Kleitman (1963) devised a tripartite categorization system for insomnia based on time of occurrence: (a) latency to sleep--inability to fall asleep, (b) interrupted sleep--frequent awakenings during the night, and (c) terminal insomnia--early morning awakening. Unfortunately much of the research on insomnia, especially with pharmacological
treatments, did not treat these classification differentially (Zarzolinski, Browne, & Almassy, 1969). Consequently, the treatment effect has not been unequivocably evaluated. In general, those infrequent studies which have differentiated among types of insomnia have defined persons who take 30 minutes or longer to fall asleep as latency insomniacs (Kales, Preston, Tan, & Allen, 1970; Kellogg & Baron, 1975; Weiss, 1973).

Although many insomniacs had no other symptoms, a number of persons suffered insomnia associated with, or resultant from, a variety of additional medical problems. Treatment of persons without medical difficulties might have possibly differed from treatment of those with other physical problems. Karacen, Salis, and Williams (1973) have attempted to deal with this issue by developing a classification system of sleep disorders. They categorized insomnia as either a primary sleep disorder in which sleep abnormality was the primary symptom, a secondary sleep disorder in which there were other clinical problems accompanying the insomnia, or a sleep-modified disorder, in which sleep increased the occurrence of some other symptoms. While this classification system appeared to have potential merit, it has not yet been widely employed by many investigators. Because many subjects of research on insomnia have physical illnesses, generalizing from these results to a population without medical difficulties may be inappropriate.
In medical practice, the treatment of insomnia involved the widespread use of prescription medication, especially hypnotic agents which induce sleep (Kane, 1970; Karacen & Williams, 1971). This term was generally applied to the barbiturates, such as pentobarbital (Nembutal) and secobarbital (Seconal). Nonbarbiturate hypnotics included glutethimide (Doriden) and methaqualone (Quaalude). One benzodiazepine, flurezepam (Dalmame), was also classified as a hypnotic. The primary difference between most barbiturate and nonbarbiturate agents was a chemical one, not a difference in sleep-inducing qualities or side-effects. The benzodiazepine, however, was different in chemistry, physiological action, and side-effects.

The use of hypnotics has risen dramatically in recent years. The retail sales of hypnotics from 1952 to 1963 increased 535% but the average increase in sales for all drugs was only 6.5%. Despite this increase in usage, there were several adverse effects of most hypnotic agents, especially with prolonged use. These effects included psychological dependence and physical addiction. In order to induce sleep, increasingly larger doses were required which may even approach the lethal level (Dement & Villablanca, 1974; Honigfeld & Howard, 1973; Kales, Tan, Swearingen, & Kales, 1971; Kane, 1970; Shapiro, Slone, Lewis, & Jick, 1969). This was true of most barbiturate and nonbarbiturate hypnotics but not of flurezepam. Kales and his co-workers (Kales, Kales,
Scharf, & Tan, 1970; Kales et al., 1970; Kales, Bixler, Tan, Scharf, & Kales, 1974) have demonstrated another side effect of hypnotics. In a series of studies, these researchers have shown a number of hypnotics resulted in sleep pattern alterations that included "drug withdrawal insomnia."

Briefly, an individual with no history of sleep problems was given a hypnotic during a particularly stressful period. Although the drug was helpful for a few days, the individual was likely to experience increased difficulty in getting to sleep after several weeks. Latency was increased when the drug was withdrawn. Another common side effect was Rapid Eye Movement (REM) rebound. Hypnotics typically suppressed REM sleep, and after the drugs were terminated an increased amount of REM sleep was observed. Further, sleep during REM was fitful. Apparently sleep was only "borrowed" when these hypnotics were being used (Montgomery, Perkin, & Wise, 1975).

In view of the difficulties resulting from the long-term prescription of hypnotics, it was appalling that recent epidemiological surveys reported that about one-third of the patients who were treated by practitioners in general medicine were receiving hypnotics for 3 months or longer (Adams, Horder, Horder, Modell, Steen, & Wigg, 1966; Johnson & Clift, 1968; Kales, Heuser, Kales, Rickles, Rubin, Scharf, Underleider, & Winters, 1969). Hazards as described above were inherent in lengthy therapy.
The effectiveness of hypnotics in treating insomnia for short durations has been repeatedly demonstrated in research. Most pharmacological research on insomnia has been conducted to assess the efficacy of various hypnotics compared to other commonly used hypnotics or to placebos (Dement, 1974).

Pattison and Allen (1972) conducted a study typical of research with hypnotics. These authors compared the effectiveness of four hypnotics: secobarbital (100 mg Seconal), pentobarbital (100 mg Nembutal), methyprylon (300 mg Noludar), ethchlorvynol (500 mg experimental drug), and a placebo on 50 hospitalized patients, most of whom were 50 years of age or older. All of these patients were in a chronic disease hospital and all had a history of recent difficulty in sleeping, including difficulty in sleep onset and/or sleep duration. Each patient received one of each of the four drugs and placebo on consecutive nights, one drug per night for a total of five nights in a double-blind randomized-block-design procedure. The effectiveness of medication was measured by objective observers who recorded in half-hour units the time to sleep onset, frequency of interrupted sleep, duration of sleep, as well as subjective reports of quality of sleep and any hangover effects. For sleep onset, methyprylon, secobarbital, and pentobarbital were found to be significantly better than the placebo ($p < .05$). No significant differences between the drugs in effecting sleep onset (latency to sleep) were found. In a similar way, Brown
(1970) compared the differences between methyprylon (300 mg Noludar), glutethimide (500 mg Doriden), and chloral hydrate (1000 mg Noctec), with a population of inpatients suffering interrupted and latency insomnia. He likewise found no significant differences between these drugs in maintaining sleep.

As discussed earlier, these studies did not allow a generalized interpretation of the effectiveness of the drugs for several reasons. Most research was with patients having both insomnia and medical problems. However, in general practice in which many patients had primary latency insomnia, symptoms were limited to insomnia. Most prescriptions for hypnotics were for outpatients, but most research on hypnotics was done with inpatients. Conclusions based on data from inpatients did not generalize to outpatients because of different expectations of treatment. In addition, the types of insomnia were not differentially considered in most research. This was important in that one drug may affect latency insomnia but not terminal awakening.

One study which did differentiate primary from secondary sleep disorders compared the effectiveness of secobarbital (100 and 200 mg Seconal), methaqualone (150 and 300 mg Quaalude), and a placebo (Bloomfield, Tetreault, Lafreniere, & Bordeleau, 1967). The authors used both normal volunteer insomniacs (primary) and psychiatric patients with insomnia (secondary) in a double-blind crossover design and observed
differential drug effects between the two types of patients. Normal subjects had some hangover effects from secobarbital (200 mg) not reported by psychiatric patients. Both normal subjects and psychiatric patients with insomnia reported secobarbital (200 mg) most effective in inducing and prolonging sleep as measured by a subjective sleep questionnaire, whereas methaqualone at both dosages and secobarbital (100 mg) were not found to be significantly different from the placebo. This study suggested that insomniac subject populations with both normal subjects and subjects with emotional or physical complaints masked some differential drug effects. However, the authors contended that because the effect on insomnia was the same in both populations, research on normal insomniac populations was also relevant for emotionally disturbed populations.

As indicated by studies previously cited, most pharmacological research on insomnia compared hypnotics within a given class of drugs. The major exceptions to this were the occasional studies comparing hypnotics with the hypnotic benzodiazepine, flurazepam (Dalmane), also frequently prescribed for insomnia. It was less addictive than the other hypnotics, and had no REM-rebound effect. Kales, Allen, Scharf, and Kales (1970) compared chloral hydrate (1000 mg Noctec) and glutethimide (500 mg Doriden) with flurazepam (30 mg Dalmane) in a 2-week sleep laboratory study. Flurazepam was found to be effective both in inducing and
maintaining sleep over the entire 2-week period of drug administration. Chlora hydrate and glutethimide, as expected of hypnotics, significantly decreased sleep latency on the first 3 nights, but these effects rapidly diminished thereafter. Pines, Rooney, and Arenillas (1976) reported flurazepam (15 mg) likewise to be more effective with insomnia than amylobarbitral (100 mg Amytal).

The use of other soporific drugs in the treatment of insomnia has been suggested, but research on comparisons was sparse (Kane, 1970). One unique study by Lingl (1973) compared the effectiveness of two soporific major tranquilizers, haloperidol (2 mg Haldol), a butyrophenone, and thioridazine (40/80 mg Mellaril), a piperidine phenothiazine compound. Subjects were 37 outpatients diagnosed as psychoneurotic, and who exhibited moderate to severe anxiety, tension, somatic concern, and irritability. They had been resistant to treatment of their anxiety with previous benzodiazepine and/or major tranquilizer and/or antidepressant drug therapy. Results indicated that thioridazine was superior in alleviating insomnia in these subjects.

Kane (1970) offered evidence that physicians frequently prescribed hypnotic agents for prolonged periods of time for patients who said their sleep was disturbed. This was done despite the fact that most hypnotics (except a few such as chlora hydrate) allowed patients only to borrow sleep, and most resulted in dependency or abstinence syndromes.
Unfortunately, however, cross-class comparative studies of drugs which avoided some of the dependence problems of the hypnotics were sparse.

Some classes of drugs with soporific properties (other than hypnotics) included antipsychotics, antianxiety agents, antidepressants, and antihistamines. A review of the literature did not reveal any study which compared the efficacy of hypnotics with other classes of drugs which have hypnotic properties. These drugs did not have the same side and dependence effects as the hypnotics. Lingl (1973) has demonstrated that antipsychotic agents were used to alleviated insomnia without the risk of addiction or sleep pattern disturbance that accompanied hypnotic agents. Antianxiety agents have been demonstrated to be effective in treating insomnia, especially flurazepam (Dalmane) which is chemically a benzodiazepine but effectively a hypnotic (Pines et al., 1976).

In addition to pharmacological treatment of insomnia, there was increasing evidence that insomnia was also decreased by attributional manipulations which effected the causal explanation a person made for any event (Kelley, 1973). The initial study demonstrating the effectiveness of fostering a new attribution of the perceived cause of insomnia was done by Storms and Nisbett (1970) who hypothesized that the arousal reported by insomniac patients at bedtime was blamed by them on their emotions. They further theorized that, to the extent an insomniac went to bed in a state of autonomic
arousal and associated that arousal with cognitions which were emotionally toned, he should have become more emotional, and thus have had greater difficulty in falling asleep than if the arousal had been attributed to a nonemotional source (e.g., a drug). Results which supported these hypotheses had been demonstrated previously by Schachter and Singer (1962). To test these hypotheses, all patients were given a placebo, one-half of the subjects being told the pill would cause arousal (arousal condition) and the remaining half that the pill would reduce arousal (relaxation condition). Thus, patients in the arousal condition were expected to attribute some degree of their arousal to the pill, and to perceive their arousal as nonemotional in nature. Those subjects in the relaxation group were hypothesized to expect a reduction in arousal, not forthcoming from the placebo. This would have resulted in the subjects blaming the arousal on their emotions, and viewing their condition as more serious and intense because it was resistant to treatment. As predicted, the authors found that subjects in the arousal condition apparently were successfully deceived into reattributing their arousal to the nonemotionally perceived pill and subsequently got to sleep significantly faster than during baseline periods. In the relaxation condition, the clients' latency to sleep significantly increased. These findings demonstrated that severity of insomnia had been manipulated by changing a person's attributions about his insomnia.
Several other studies have reported the successful treatment of sleep disturbances by placebo (Lasagna, Mosteller, von Felsinger, & Beecher, 1954; Nicolis & Silvestri, 1967), but these did not deliberately manipulate the client's attributions with regard to his symptomatology or the drug. More recent attributional manipulations have failed to achieve the reverse-placebo effect found by Storms and Nisbett (1970), i.e., insomniacs falling asleep faster when given placebos believed to result in the arousal common in insomnia.

Instead, there was mounting evidence that attributional manipulations involving direct suggestion regarding placebo medication had been more effective in the treatment of insomnia. Bootzin, Herman, and Nicassio (1976) attempted to replicate the reverse-placebo effect found by Storms and Nisbett (1970), as well as to define the mechanism by which this phenomenon occurred. It was hypothesized by Bootzin and associates that the reverse-placebo effect would occur when the drug's effects were described as effecting arousal, but that a direct suggestion effect would result if the pills were described as directly effecting sleep onset latency.

Two of the experimental conditions in this study were exact replications of the Storms and Nisbett arousal and relaxation manipulations. Two other experimental groups were employed to determine the effect of direct, sleep-related suggestions. Subjects in these groups were told that the pills would help them to fall asleep, or that the drug would generally tend
to keep them awake longer than usual. The major finding of
the study was that the direct suggestion effect, in which
subjects were instructed that the pill would help them sleep,
generally feel asleep faster than those subjects told the
pill would arouse them. The reverse-placebo effect was not
found in any of the manipulations. A similar attempt at
replication of the reverse-placebo effect by Kellogg and
Baron (1975) was likewise unsuccessful. Thus, the evidence
seemed to indicate that when the patient was led to believe
that a drug (placebo) would help him fall asleep, and he
could attribute his falling asleep more rapidly to placebo,
direct suggestion would have effectively improved sleep onset
latency. Bootzin et al. (1976) demonstrated that attempts
to achieve the reverse-placebo effect actually resulted in
exacerbation of the insomnia. Because of the demonstrated
effectiveness of direct suggestion in attributional manipula-
tions, the use of direct suggestion with soporific medications
instead of placebos would perhaps be even more effective in
inducing sleep.

The direct suggestion effect has been demonstrated with
severe latency insomniacs, generally taking over 60 minutes
to fall asleep (Bootzin et al., 1976), more moderate latency
insomniacs (Kellogg & Baron, 1975), older subject populations
(Bootzin et al., 1976), and with college undergraduates
(Kellogg & Baron, 1975). Nicolis and Silvestri (1967), how-
ever, found nonsignificant differences between a placebo,
phenobarbital (100 mg Eskabarb), and two experimental drugs in the treatment of mild and moderate insomnia of mixed type (latency and interrupted insomnia), but found phenobarbital more effective with severe insomnia. These different responses due to severity might have been spurious because of the circularity of the operational definition used in this study to determine if someone were a severe insomniac who could rarely have a satisfactory night of sleep without hypnotics.

A variety of other recent attributional literature, though not specifically dealing with an insomniac population, appeared relevant to the treatment of insomnia which, in part, dealt with expectancy and justification (Brehm & Cohen, 1962; Weiner, 1974). These authors have demonstrated in repeated research that the higher the expectancy for change, the more likely change was to occur. They have also shown that the more justification used to induce change, the less likely change became. Other attributional literature, as exemplified by research on token economies, supported the hypothesis that a change in behavior would have been better maintained over time if the behavior was perceived to be internally and not externally controlled (Lepper & Green, 1975; Lepper, Green, & Nisbett, 1973, Luce & Segal, 1969).

One relevant therapeutic study on the treatment of insomnia with regard to durability of treatment due to attributional manipulation was by Davison, Tsujimoto, and
Glaras (1973) who administered 1,000 milligrams of choral hydrate (Noctec) per night to 15 clients with insomnia who were additionally trained in self-induced relaxation. After 1 week of treatment, all clients had significantly reduced latency to sleep. One-half of the group was told that the drug dosage they had been taking was much too low to have produced improvement. The remaining portion were told that they had been taking an optimal dosage of the drug. All clients were taken off the drug, but were instructed to maintain relaxation. It was found that those subjects who were told they had received an inadequate dose of the drug, and thus could not attribute improvements to that drug, showed a significantly greater maintenance of therapeutic gain than did clients in the optimal dosage condition. It was concluded that those subjects unable to attribute improvement to the drug probably attributed it internally. This seemed reasonable, and it was consistent with a growing body of research indicating self-attributed behaviors were better maintained over time following therapy, probably because the subject assumed he could control his own behavior rather than having his behavior controlled by some external force (Deci, 1971, 1972; Levine & Fasnacht, 1974; Winett, 1970).

Although Davison's study with choral hydrate and direct suggestion appeared to be one of the only studies using actual drug treatment (versus placebo treatment) of insomnia, the implications for clinical medicine in the treatment of
insomnia were clear. In view of the effectiveness of direct suggestion coupled with a potent hypnotic with known soporific effects, a therapist might be able to avoid the dangers inherent in prolonged drug treatment of insomnia. If a patient were given a soporific drug for several days to attain a shorter latency to sleep, and then aided in attributing that change to his own control, further drug treatment might be avoided without compromising the patient's ability to obtain adequate sleep. The inherent habituation and tolerance of hypnotics and the side effects from the prolonged drug use could be minimized since only a short course of drug therapy would be necessary.

Accordingly, the present study was designed to test the hypothesis that the durability of drug therapy could be increased by attributional manipulation. It was also designed to determine which of several classes of drugs with soporific properties was the most efficacious in the short-term treatment of primary latency insomnia.

Method

Subjects

Volunteers whose primary complaint was difficulty in falling asleep were referred to sleep clinics established in two cities in Texas. One of these clinics was located in the Health Center of North Texas State University, and referrals were from this source as well as the University's Counseling Center. The other sleep clinic was located in a professional
office building in Waco to accommodate referrals from the faculty at Baylor University, local physicians, psychiatrists, and community health care centers. Both sleep clinics were operated daily and were open free of charge to volunteers who wished to participate in research on drug treatment of insomnia. A total of 49 persons entered the research project as volunteers. Subjects were randomly assigned to cells. Of these, nine subjects across all eight conditions did not complete the study, two complaining that their prescribed drug was too strong, one that his drug was ineffective, two due to illness during the study, and four for unknown reasons. The final sample consisted of 40 subjects, 21 females and 19 males ranging in age from 18 to 73 with a mean age of 29.68 years (SD = 12.77).

All volunteers to participate in the experiment were screened prior to inclusion to assure they met certain criteria, such as absence of drug allergies to the medications used in the study. Persons not meeting these criteria were referred for medical assistance for their insomnia to qualified medical personnel. The latency to sleep (defined as the difference in minutes from that time when a person desired to sleep and the time he actually fell asleep) was 30 minutes or longer for all subjects for 3 or more nights prior to the study. Volunteers were screened to rule out physical illnesses and drug allergies to any of the medications included. Patients were determined to have no known hepatic, respiratory,
or cardiovascular difficulties by their own subjective report, clinical observation, or their health histories on file in the clinic. Because subjects had medical screening prior to referral, most volunteers were allowed to participate.

Materials

The instrument used to measure the latency to sleep was a sleep questionnaire. Since no standardized sleep questionnaires were used in previous research, this questionnaire included critical items found by Monroe (1967) and Wolfe (1974) as well as items used by Bootzin and associates (1976) in attributional manipulations with sleep. The questionnaire was designed for expedient analysis of the effects of both the medication and the attributional manipulations. A questionnaire about the previous night's sleep was filled out immediately upon arising. Because the subjective report has been demonstrated to be accurate in detecting differences in sleep behavior, a subjective report of latency to sleep for 3 days prior to the administration of the drugs was also taken as a baseline measure. A measure by recall rather than a subjective measure taken each morning for 3 baseline days was done to avoid compromising patient care and to circumvent confounding factors, such as ingestion of over-the-counter medications which might otherwise occur during the baseline.

Procedure

All meetings between the subjects and the experimenter took place in the sleep clinics. This location was chosen to
maximize the credibility of the placebo manipulations and to increase convenience to the subjects. All interview sessions were individually conducted by one of two experimenters.

Subjects were greeted during the first contact and asked to sign a consent form indicating that they understood they were participating in an experimental study in which they would be asked to take a drug for a total of 3 nights. Subjects agreed to remain in the study a total of 10 days and to refrain from taking any medications, prescription or non-prescription, or drinking any alcohol during that period. All participants were informed they were free to withdraw at any time since participation was voluntary. Subjects were asked to notify the experimenter prior to withdrawing (five subjects did contact the experimenter; four did not). Data from all nine subjects prematurely withdrawing were dropped from the study. These conditions were rerun with subjects subsequently volunteering for the study so that there were ten subjects total in each of the four groups.

Before any subject was seen, an independent researcher randomly assigned conditions to each of 40 consecutive subject numbers (4 drug conditions X 2 attribution conditions X 5 subjects per cell). These conditions were ordered by the independent experimenter so that subjects were randomly assigned to one of four groups: secobarbital (100 mg Secobarbital), flurazepam (30 mg Dalmane), thioridazine (50 mg Mellaril), or placebo group until these conditions contained
10 subjects on each drug, 5 for each attribution condition. Each drug used had been demonstrated to be a soporific member of one of three classes of drugs (Honigfeld & Howard, 1973; Kales, Allen, Scharf, & Kales, 1970; Lehman, 1975). Since it was the goal of the study to assess the effectiveness of these drugs in general practice, the most commonly used dosage regimens were employed. To control for the effects of the therapeutic manipulations involved, a nonactive drug placebo group, using lactose, was also included (Shapiro, 1971). Neither the experimenter nor the subject knew to which drug condition assignment was made. The experimenter was provided with a set of cards with a number on the front side and an attributional condition on the back side, indicating either self- or drug-attribution. These cards were not reviewed by the experimenter until after the drug administration for 3 consecutive nights. A 3-day course of drug treatment was used since this is the recommended treatment course (Clark & del Guidice, 1970; Goldstein, Graedon, Willard, Goldstein, & Smith, 1970; Kleitman, 1963; Lasagna, 1956).

The experimenter completed a general demographics sheet after interviewing and screening the subject (see Appendix A). Included on this form was latency to sleep during the preexperimental period, the 3 days prior to participation in the study. The subject also signed an informed consent form (see Appendix B). He was then provided with his medication.
All prescriptions were signed by a supervising physician who was blind to the experimental condition of each subject. Subjects were given the instructions to take the drug 1 hour prior to the time at which the subject wished to fall asleep. Each subject was also provided with a telephone number to call in case of any adverse effect or discomfort from the drug.

Each subject was given the following instructions prior to receiving his medication supply:

This drug will generally tend to help you fall asleep. Its effects on your body are fairly diffuse, but you should have less trouble than usual in falling asleep. No side effects are expected, but if any occur, call the number given to you. Follow the directions given. Remember, you are not to take any other medication or drink any alcohol for the time you are on this drug. You are to fill out the sleep questionnaires provided to you immediately upon arising.

The subject was provided with three sleep questionnaires (see Appendix C) to complete upon awakening after having taken the medication the previous night. An appointment was made to return on the day the third questionnaire was completed for another brief interview and for the subject to return the sleep questionnaires.

When the subject returned, the experimenter took the three completed sleep questionnaires and provided the subject
with seven more. As provided for in the random assignment procedure, half of the subjects in each condition were told the following (self-attribution condition):

The drug you took was not a drug typically used to treat insomnia. It is usually used to treat other disorders. Any effects you felt were effects of the drug on your body as is typical with this drug, but the drug should not have affected your ability to sleep. Any change made in your sleeping behavior, that is, if you found it easier to fall asleep, was not due to the drug, but to changes you made yourself in your sleeping behavior.

Subjects in the other condition (drug-attribution) were told the following:

The drug you took is typically effective in causing people to go to sleep more easily. The effects you felt, especially in your sleeping behavior, were probably due to this drug.

Appointments were then made for subjects to return after 7 days for the final debriefing interview in which they were requested to give their subjective feelings about the overall effectiveness of the drug used. Depending on their attributional condition, subjects were asked whether or not they believed that they caused the change in their behavior or that the drug caused the changed behavior. After this, the actual nature of the experiment was explained. Subjects were
thanked for their participation and were referred for further treatment if necessary. Subjects in the self-attribution condition were given the option to complete the sleep questionnaires for an additional 7 days after the 10-day experimental and postexperimental period to determine the effects of the debriefing, i.e., after the subjects were initially told the drugs were not responsible for changed sleep behavior. The experimenter stressed to each of the subjects in this condition who had maintained the change himself that there was no reason to expect he could not continue to do so. Only 6 of the 20 subjects in the self-attribution condition chose to participate in the additional follow-up.

Results

The mean latencies to sleep during the 3-day preexperimental, 3-day experimental, and 7-day postdrug periods were employed as dependent measures. A three-factor fixed effects on all factors analysis of variance with repeated measures on one factor was calculated (Winer, 1971). The results, as indicated in Table 1, demonstrated the only significant main effect was for blocks of trials, $F(2,6) = 14.21, p < .01$.

The latency to sleep varied across the type or block of trials. Thus, preexperimental latencies were highest, latencies to sleep during drug administration were lowest, and postexperimental latencies were lower than preexperimental latencies but higher than latencies during drug administration.
Table 1
Summary of Analysis of Variance of Mean Latencies to Sleep

<table>
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<th>Source</th>
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<tr>
<td>A X B</td>
<td>3</td>
<td>12251.66</td>
<td>2.54</td>
</tr>
<tr>
<td>Subjects X A X B</td>
<td>32</td>
<td>4834.48</td>
<td></td>
</tr>
<tr>
<td><strong>Within subjects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trials (C)</td>
<td>2</td>
<td>27577.44</td>
<td>14.21**</td>
</tr>
<tr>
<td>A X C</td>
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<td>2067.33</td>
<td>1.06</td>
</tr>
<tr>
<td>B X C</td>
<td>2</td>
<td>624.25</td>
<td>.32</td>
</tr>
<tr>
<td>A X B X C</td>
<td>6</td>
<td>2690.93</td>
<td>1.39</td>
</tr>
<tr>
<td>C X Subjects X A X B</td>
<td>64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*\(p < .05\).

**\(p < .01\).

There was no significant difference in drug effects for any of the different types of drugs. Further examination of data in Table 2 of drug effects indicated that regardless of class or type of drug or placebo administered, there was a decrease in latency to sleep.

As demonstrated in Tables 1 and 3, there was no significant effect from any attributional manipulation regarding
effects of drugs. Both drug-attribution and self-attribution groups maintained decreased latency to sleep during the post-experimental period.

Table 2
Summary of Drug Effects on Mean Latencies to Sleep

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Blocks of Trials</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre</td>
<td>Experimental</td>
<td>Post</td>
</tr>
<tr>
<td>Secobarbital</td>
<td>X</td>
<td>89.17</td>
<td>21.33</td>
<td>69.86</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>85.85</td>
<td>10.27</td>
<td>90.60</td>
</tr>
<tr>
<td>Thioridazine</td>
<td>X</td>
<td>94.67</td>
<td>18.33</td>
<td>64.71</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>108.98</td>
<td>14.68</td>
<td>71.93</td>
</tr>
<tr>
<td>Flurazepam</td>
<td>X</td>
<td>64.83</td>
<td>29.50</td>
<td>37.57</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>49.33</td>
<td>22.58</td>
<td>24.54</td>
</tr>
<tr>
<td>Placebo</td>
<td>X</td>
<td>53.17</td>
<td>22.67</td>
<td>20.43</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>18.11</td>
<td>10.84</td>
<td>8.53</td>
</tr>
</tbody>
</table>

As indicated in the tables, all groups were likely to fall asleep faster on experimental nights than on pre- or postexperimental nights. Analysis of simple effects on difference scores, as shown in Table 4, demonstrated that there was a significant decrease in latency to sleep on the nights that drugs were administered from preexperimental nights, as well as from postexperimental nights.
Table 3
Summary of Attribution Effects on Mean Latencies to Sleep

<table>
<thead>
<tr>
<th>Attributions</th>
<th>Blocks of Trials</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Experimental</td>
<td>Post</td>
</tr>
<tr>
<td>Drug</td>
<td></td>
<td>71.75</td>
<td>24.33</td>
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<tr>
<td></td>
<td>SD</td>
<td>69.45</td>
<td>18.20</td>
</tr>
<tr>
<td>Self</td>
<td></td>
<td>79.17</td>
<td>21.58</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>78.65</td>
<td>12.21</td>
</tr>
</tbody>
</table>

Examination of Table 4 also revealed that there was a significant decrease in latency to sleep in postexperimental nights as compared to preexperimental nights. Thus, the effects of the drugs were maintained, regardless of type of drug administered or the attribution regarding the cause of the changed sleep behavior, subsequent to the termination of the drug.

Table 4
Newman Keuls Analysis of Blocks of Trials
Simple Main Effects

<table>
<thead>
<tr>
<th></th>
<th>$X_1$</th>
<th>$X_3$</th>
<th>$X_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$(75.46=)X_1$</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$(48.14=)X_3$</td>
<td></td>
<td>27.32*</td>
<td>-</td>
</tr>
<tr>
<td>$(22.96=)X_2$</td>
<td>52.50**</td>
<td>37.55**</td>
<td>-</td>
</tr>
</tbody>
</table>

*$_{p} < .05$.

**$_{p} < .01$. 
As demonstrated in Table 5, high variability in latency to sleep was found across conditions. An $F_{\text{max}}$ test (Winer, 1971) of data during drug administration indicated homogeneity of subjects. However, an $F_{\text{max}}$ test of postexperimental data revealed heterogeneity. A square-root transformation (Winer, 1971) was done on all postexperimental data, and a two-way analysis of variance fixed on both variables (drugs X attribution) was completed. Results of this analysis likewise revealed no significant difference among types of drugs administered or between attributional conditions in maintenance of drug effectiveness.

Because only six subjects across all conditions opted to collect the 7 additional days of data after the experiment was explained, data analysis was not performed. Likewise, a separate data analysis was not done on the three subjects who did not believe the attribution, and on an additional three for whom data on attributional belief were not collected. These samples would have been too small for adequate interpretation.

Discussion

Results of the present study supported the hypothesis that other drugs were as effective in treatment of insomnia as hypnotic barbiturates. The data indicated that secobarbital (a barbiturate hypnotic), thioridazine (an antischizophrenic piperadine), and flurazepam (a benzodiazepine), all constituted effective short-term therapy for treatment of
Table 5
Summary of High Variability of Cell Means and Standard Deviations

<table>
<thead>
<tr>
<th>Drug</th>
<th>Attribution</th>
<th>Blocks of Trials</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pre</td>
<td>Experimental</td>
<td>Post</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Totals</td>
</tr>
<tr>
<td>Secobarbital</td>
<td>Drug</td>
<td>X 119.57</td>
<td>21.33</td>
<td>97.57</td>
<td>238.57</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>117.94</td>
<td>11.02</td>
<td>127.28</td>
<td>209.34</td>
</tr>
<tr>
<td>Secobarbital</td>
<td>Self</td>
<td>X 58.67</td>
<td>21.33</td>
<td>42.14</td>
<td>122.14</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>18.69</td>
<td>10.76</td>
<td>18.61</td>
<td>16.84</td>
</tr>
<tr>
<td>Thioridazine</td>
<td>Drug</td>
<td>X 43.67</td>
<td>14.33</td>
<td>36.00</td>
<td>94.00</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>13.35</td>
<td>6.93</td>
<td>8.76</td>
<td>13.30</td>
</tr>
<tr>
<td>Thioridazine</td>
<td>Self</td>
<td>X 145.67</td>
<td>22.33</td>
<td>93.43</td>
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</tr>
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<td></td>
<td>SD</td>
<td>141.57</td>
<td>19.92</td>
<td>97.49</td>
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</tr>
<tr>
<td>Flurazepam</td>
<td>Drug</td>
<td>X 69.67</td>
<td>34.33</td>
<td>51.71</td>
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</tr>
<tr>
<td></td>
<td>SD</td>
<td>63.76</td>
<td>31.10</td>
<td>26.85</td>
<td>115.93</td>
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<td>Flurazepam</td>
<td>Self</td>
<td>X 60.00</td>
<td>24.67</td>
<td>23.43</td>
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</tr>
<tr>
<td></td>
<td>SD</td>
<td>36.74</td>
<td>11.02</td>
<td>11.58</td>
<td>63.97</td>
</tr>
<tr>
<td>Placebo</td>
<td>Drug</td>
<td>X 54.00</td>
<td>27.33</td>
<td>23.57</td>
<td>104.91</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>22.47</td>
<td>12.83</td>
<td>9.20</td>
<td>31.71</td>
</tr>
<tr>
<td>Placebo</td>
<td>Self</td>
<td>X 52.33</td>
<td>18.00</td>
<td>17.29</td>
<td>87.62</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>15.21</td>
<td>6.71</td>
<td>7.38</td>
<td>20.67</td>
</tr>
<tr>
<td>Total</td>
<td>X 75.46</td>
<td>22.96</td>
<td>48.14</td>
<td>104.91</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>73.33</td>
<td>15.36</td>
<td>60.51</td>
<td></td>
</tr>
</tbody>
</table>
primary latency insomnia in the population studied. Results from the current study were consistent with previous research, which indicates that between flurazepam and the barbiturates there was relatively little difference in clinical effect in treatment of insomnia (Honigfeld & Howard, 1973; Rech & Moore, 1971). The data also indicated equal effectiveness of a soporific major tranquilizer in reduction of latency to sleep. The only other study which had demonstrated the effectiveness of antipsychotic medication (Lingl, 1973) was completed on a more severely emotionally disturbed population. Evidence from the present study extended these results to a more normal population whose primary complaint was difficulty in sleeping rather than emotional disturbance. The therapeutic effectiveness for all three drugs occurred for a 3-day course of drug treatment, a relatively short course of therapy compared to that typically reported in general practice, but a longer treatment course than used in the 1-night crossover comparative research studies.

The implication of these results for the general practice of medicine in the treatment of insomnia was that drugs other than barbiturate hypnotics may be used to treat primary latency insomnia. Flurazepam or thioridazine (both of which provided soporific properties with little tendency for addiction and little disturbance of REM sleep) could have been prescribed for treatment of insomnia as effectively as the more traditional barbiturate prescription. These drugs were
much safer for prescription due to fewer side effects and less chance of abuse (e.g., by suicidal patients who might have attempted suicide by overdose with the more lethal barbiturates). In addition, a course of treatment much shorter than that generally used was demonstrated effective in treatment of latency insomnia.

For practitioners committed to use of the barbiturates, a 3-day course of treatment might have been used effectively as demonstrated in the present study. Such a therapy would have minimized effects of REM-rebound, which was more likely to have begun subsequent to the 5th day of treatment, and effects of habitation or addiction, resultant from therapy for prolonged periods of time (Kane, 1970).

Drugs from several classes were effective in short-term treatment of primary latency insomnia. Other appropriate drugs would have included amitriptyline (tricyclic antidepressant), diphenhydrazine (antihistamine), and diazepam (benzodiazepine antianxiety agent). It was possible that lower doses of medication than those used in the study may have been equally as effective in the treatment of insomnia. The lowest effective dosage would have been optimal in treatment with any drug to lessen any side effects or toxicity of drugs. The effectiveness of lower dosages has yet to be established.

This study also indicated that a placebo drug may be as effective for treatment of insomnia as a more active drug.
This was consistent with previous research using direct suggestion or placebo treatment, but inconsistent with other research which demonstrated active drugs to be superior. The inconsistency in the literature may have resulted from one of several reasons. First, most experimentation of drug effectiveness was done on an inpatient population and generalized to outpatient usage. Inpatients in most cases have had some physical illness in addition to the sleep difficulty which would not have been affected by placebo administration but might have received secondary relief from sleep due to administration of a soporific drug. Secondly, subjects responsive to direct suggestion probably suffered from mild to moderate transient insomnia and not severe or chronic sleep difficulty. The former patient was the type usually seen in general practice but the latter was frequently used as research subjects. A third factor was that most research used a 1-day crossover design in comparative drug studies. Such a design would have maximized by contrast differences between active drugs and placebos. This design was not typically used in the direct suggestion literature, nor was it typically used in general practice in treatment of insomnia.

The result of the present study was consistent with direct suggestion attributional manipulations in previous studies which have demonstrated that by using placebos and direct suggestion, subjects have fallen asleep more easily (Bootzin et al., 1976; Kellogg & Baron, 1973). The clinical
implication of the demonstrated effectiveness of placebo was that toxicity and side effects of the active drugs might have been avoided by prescription of a placebo for the treatment of insomnia. Such a course of treatment, although as effective as active drugs, might have been undesirable for some physicians who for ethical reasons or because of non-naive patients, may have avoided prescription of a placebo.

The second hypothesis of the study, that attributional manipulations subsequent to drug administration may have enhanced maintenance of drug effectiveness, was not supported in its entirety. Results indicated that decreased latency to sleep following drug therapy was maintained regardless of the drug used or of the subject's attribution regarding his changed sleep behavior. Most drug research with soporific drugs has not studied maintenance of effects following treatment. The results of maintained effectiveness, especially in view of attributional research, was therefore not predicted.

Previous research by Davison and associates (1973) and Kellogg and Baron (1973) seemed to indicate that attributional manipulation regarding changed sleep behavior following drug administration may have affected ability to fall asleep and prolong maintenance of drug effectiveness if attribution was made to an internal causation of change. The present study demonstrated maintenance of drug effectiveness regardless of whether changed behaviors were attributed to the drug or to self-initiated changes. This result may have derived from
affects by several attributional factors additionally operative in the research design.

One factor resulting in different maintenance effects between the present study and previous studies was differences in subject population. Both of the previous attributional studies used only college or university students, while subjects in this study included both college students and volunteers from the general population. However, the major difference in population appeared to be in types of subjects recruited. Kellogg and Baron (1975) and Storms and Nisbett (1973) recruited subjects through the university newspaper who were interested in research on arousal and dreams. The present study was done with volunteers, referred by clinicians and physicians, whose primary complaint was insomnia. Davison et al. (1973) recruited through newspaper advertisements but failed to report what criteria subjects were given in the advertisements. Although one might have assumed that advertisements specified insomniacs, this may not necessarily have been the case.

The differences in recruitment procedures and in primary complaints of subjects between previous attributional research and the present study were likely to have resulted in different expectancy attributions for the subjects involved. Subjects in the present study were referred by professionals and had a primary complaint of difficulty in falling asleep. Although not specifically monitored, it was likely that
volunteers expected relief from symptoms of insomnia during the experimental procedure. These expectations were likely maximized through treatment at a location clearly identified visually and verbally as a sleep clinic and through the referral process. They were not likely to have the same expectation of alleviation of difficulties in sleeping as volunteers of previous studies recruited for dream research. Likewise volunteers in the Davison et al. study (1973), recruited through newspaper advertisements, were taught relaxation techniques in addition to drug therapy, which may have resulted in expectations of different arousal levels and not just in changed sleep behavior. Past research on expectancy (Weiner, 1974) has demonstrated that subjects base expectations on past outcome, and that an expectancy of change enhances actual change. It was likely that volunteers had received medical attention in the past in a professional setting which alleviated identified symptoms. Thus, expectancy for improvement in latency to sleep likely enhanced results of sleep clinic participants, but the attributional factor did not seem operational in the prior attributional research.

A second major attributional factor likely to have contributed to the generalized maintenance was degree of justification for changed behavior. As demonstrated by attributional research, exemplified by the token economy studies (Lepper & Greene, 1975), the higher the justification for
change, the less likely the change was to occur. As a method to increase subject participation, the three prior studies on attributional manipulations in treatment of insomnia paid subjects for their participation or required a monetary deposit. Justification for changed behavior in these studies then became, in part, primarily external or monetary. Further, half of the subjects in the Kellogg and Baron (1975) study were given additional verbal justifications that the experimental results were extremely important to science. However, those subjects given no justification and receiving no placebo had the lowest latency to sleep. This, then, was consistent with other attributional data that the greater the justification and more reasons given, the more external the change appeared to be and the less likely change was to be maintained subsequent to the manipulation.

Justification for participation of volunteers in the present study was apparently internal in nature, a desire to improve sleep behavior. This lower justification condition, regardless of whether change was attributed to the drug or to self-initiated behaviors, was likely to have enhanced maintenance of effectiveness across conditions.

A final direct suggestion factor may have operated in the present study since all subjects were told prior to receiving any drugs that the drugs should help them fall asleep easier. It is possible subjects in the drug-attribution condition, due to expectancy from past relief by
prescription, assumed they were "cured" and maintained their changed behavior. Self-attribute subjects received an additional attribution that their improved latency to sleep was due to changes made in self. Thus it was possible that maintenance of the drugs' effects occurred for these subjects consistent with internal attributional literature. The results of continued decreased latency to sleep across conditions, although not as great as during drug administration, may thus have resulted from a multiple-factor attributional process. This would explain different results in the current study from prior studies. There was, however, no definitive means of showing that the sleep onset changes were produced by different attributional factors. Further research would have been necessary to test these factors.

Perhaps a second alternative was that regardless of treatment or drug, latency to sleep improved over time with any type of therapy. This would have explained effects of the placebo, but extrapolation from the drug administration to other types of manipulations such as relaxation therapy would have required further testing.

A final factor demonstrated by the study was variability of subjects. In view of the sampling procedures used without attention to severity of latency, the sample was likely to be representative of conditions existing for physicians prescribing drugs for the treatment of the general patient population complaining from insomnia.
The applicability of the study was limited to the degree that the subject population, although closer to that seen in general practice than in previous studies, was not representative of the general population due to a high concentration of college students. Another area of limitation in generalizing was that results could be applied only to the drugs used in the study and not the class of the drugs used.

The study, like previous attributional research with sleep, failed to replicate Storms and Nisbett's (1970) reverse-placebo effect; rather, it substantiated mounting evidence for a significant direct suggestion effect. This effect was found regardless of attributional manipulation to self or drug, and this discrepancy in results demanded a consideration in future research. As previously discussed, differences in subject population used in the study from previous studies may have resulted in a population with different cognitive and attributional responses. Further research must be aware of subtle differences in expectation of improvement resulting from subject recruitment, verbal instructions to subjects, and setting of the experiment. The study (unlike previous research) apparently maximized these factors to result in an expectancy of improvement. A difference in justification for participation, in that no money was transferred for participation, apparently maximized an internal justification for participation which may have acted additionally to, or more powerfully than, the drug (external)
or self (internal) attributions which the study was designed to induce. Future research must attempt to more carefully control such attributional factors to gain generalizable results.

Further, recommendations made are for future research to include use of physiological measures to substantiate subjective reports. Such measures could be particularly useful in analysis of the causality of the wide individual variations in latency to sleep. Future research in this area could further refine results found in this study.

The major finding of the present study was that treatment of primary latency insomnia with several drugs was effective in a 3-day course of treatment regardless of whether the drug was soporific or placebo, and regardless of the attribution concerning decreased sleep latency. This effect was maintained after treatment regardless of drug or attribution condition. This finding called into question some previous attributional research. It seemed clear that, at best, attributional effects were elusive and not under strict experimental control. The implications for the treatment of insomnia must, therefore, be cautionary. However, if further research could replicate these findings, insomnia can be treated through direct suggestion regardless of drug used. Results of the study (that several other drugs and placebo were as effective in treatment of insomnia as hypnotic barbiturates and had considerably less risk) may be expanded
in future research to even more drugs and to lower dosage regimens than those in the present study.
Appendix A

General Demographics

Name:__________ Date:__________ Id.No. ___________

Age: 5 6 Sex: 7 (1 = male; 2 = female)

Race: 8 (1 = Caucasian; 2 = Black; 3 = Mexican American; 4 = Other)

Marital Status: 9 (1 = married; 2 = single; 3 = divorced; 4 = separated; 5 = other)

Education: 10 (1 = freshman; 2 = sophomore; 3 = junior; 4 = senior; 5 = graduate)

Latency to sleep over the past three nights:

Last Night Two Nights Ago Three Nights Ago

What time do you usually go to sleep?

(Ex. 2230 = 10:30 pm)

What time do you usually wake up?

What is the usual duration of your sleep?

0 = less than 5 hours 1 = 5-6 hours 2 = 6-7 hours 3 = 7-8 hours 4 = more than 8 hours

How many disturbing awakenings do you usually have?

0 = more than 5 1 = 4-5 2 = 2-3 3 = one

Do you usually dream?

0 = no 1 = yes

Do these dreams usually disturb your sleep?

0 = yes 1 = no 2 = no dreams

How do you usually wake up?

0 = much difficulty 1 = little difficulty 2 = no difficulty 3 = easily 4 = very easily
Appendix A--Continued

Do you usually feel sleepy when you wake up?

0 = yes  1 = no

Do you usually feel rested when you awaken?

0 = no  1 = yes

Do you usually have difficulty concentrating after you waken?

0 = yes  1 = no

Indicate one of the following for each item below:

Coffee (0 = do not drink coffee; 1 = 1-2 cups a day; 2 = 3-4 cups a day; 3 = 5-6 cups a day; 4 = more than 6 cups a day)

Smoking (0 = do not smoke; 1 = less than one pack a day; 2 = 1½ packs a day; 3 = 2 or more packs a day)

Soft Drink (Cokes, Dr. Pepper, carbonated dark soft drinks) (0 = do not drink any; 1 = 1-2 per day; 2 = 3 per day; 3 = more than 3 per day)

Beer and Wine (0 = do not drink any usually; 1 = 1-3 per week; 2 = 1-2 per day; 3 = more than 2 per day)

Mixed Drinks (0 = do not drink any usually; 1 = 1-3 per week; 2 = 1-2 per day; 3 = more than 2 per day)

Have you previously been treated for insomnia?

(0 = yes  1 = no)

If so, did you receive any drugs?  (0 = yes; 1 = no)

If yes, what drug(s)?

When did your present sleep problems begin?

(0 = one week ago; 1 = 2 weeks ago; 2 = 2 weeks-1 month; 3 = 1 month-3 months ago; 4 = 3-12 months ago; 5 = more than 12 months ago)

What is the course of your sleep problem?

(0 = not a problem before this; 1 = rare; 2 = occasional; 3 = constant; 4 = cyclic and intermittent)
Can you specify any event which you link with the onset of your current insomnia? (0 = yes; 1 = no)
If yes, please explain briefly: ________________________________
Appendix B

Informed Consent Form

Name: ____________________________________________________________

Address: __________________________________________________________

Age: _______ Phone Number: ____________________

1. Are you currently taking any medication? ( ) Yes ( ) No
   If yes, please explain: ____________________________________________

2. Are you allergic to any medication? ( ) Yes ( ) No
   If yes, please explain: ____________________________________________

3. Are you currently under a physician's care? ( ) Yes ( ) No
   If yes, please explain for what and how long you have been under
   his care: _________________________________________________________

4. Have you had in the past, or do you currently have, any of the following?
   a. Respiratory difficulties ( ) Yes ( ) No
   b. Low blood pressure ( ) Yes ( ) No
   c. High blood pressure ( ) Yes ( ) No
   d. Cardiovascular problems ( ) Yes ( ) No
   e. Sclerosis, jaundice, or other liver disorder ( ) Yes ( ) No
   f. Diabetes mellitus ( ) Yes ( ) No
   g. Alcoholism ( ) Yes ( ) No

In signing this statement, I agree to the following:

1. I will refrain from taking any other medication, either prescription or
   non-prescription for ten (10) days throughout this treatment unless
   specifically cleared by the interviewer prior to this study (Drug approved):
   ___________________________________ Initials: __________

2. I will refrain from drinking any alcoholic beverage throughout the study for ten days.

3. I will try to maintain my normal daily schedule while on the medication.
4. I will refrain from napping during the day.

5. I agree to immediately call __________________ at the first sign of any adverse reaction to the medication.

6. I agree to participate in the study for ten days and will fill out a Sleep Questionnaire (provided for me) each morning immediately after awakening for these ten days. I realize that I can, at any time, withdraw from the study without penalty but I will first contact the Sleep Clinic if this is my decision.

I have heard a clear explanation and understand the nature and purpose of the treatment, possible alternative procedures that would be advantageous to me, and the attendant side effects or risks involved and the possibility of complications which might arise. I have heard a clear explanation and understand the benefits to be expected. I understand that the procedure is investigational and that I may withdraw my consent for my status. With my understanding of this, having received this information and satisfactory answers to the questions I have asked, I attempt to determine which drug is best for the treatment of insomnia, and I understand I will be asked to take one drug one hour prior to bedtime for three consecutive nights, and to monitor the effects of this drug on each of these nights and on the following seven (7) nights by filling out a Sleep Questionnaire which has been provided for me.

I certify that all the information about my medical condition is true to the best of my knowledge. I fully understand the nature of treatment and request to participate.

Date: __________________________

Signed: _________________________
   Subject

Signed: _________________________
   Witness

Signed: _________________________
   Witness
Appendix C

Sleep Questionnaire

Instructions: Answer this questionnaire as soon as you awaken. Put the number of the item that best answers each question in the blank by that question. Do not skip any questions.

Estimate in minutes how long it took you to go to sleep. (Example: 120 = 120 minutes; 015 = 15 minutes)

What was the effect of the medication before you feel asleep?

0 = very unpleasant 1 = unpleasant 2 = no effect 3 = pleasant 4 = very pleasant

What was the total duration of your sleep?

0 = less than 5 hours 1 = 5-6 hours 2 = 6-7 hours 3 = 7-8 hours 4 = more than 8 hours

How many disturbing interruptions (awakenings) did you have last night?

0 = more than 5 1 = 4-5 2 = 2-3 3 = 1 4 = none

Did you have a good night's sleep last night? (Did you sleep well?)

0 = very poor 1 = poor 2 = fair 3 = good 4 = very good

Do you remember having dreamed last night?

0 = no 1 = yes

Do you consider the dreams to have disturbed your sleep?

0 = yes 1 = no dreams 2 = no

How did you awaken compared to usual?

0 = much more difficulty 1 = more difficulty 2 = normally 3 = more easily 4 = much more easily

Do you feel sleepy this morning?

0 = yes 1 = no
Do you feel rested this morning?
0 = no  
1 = yes

Do you have difficulty concentrating this morning?
0 = yes  
2 = no
References


Kales, J., Tan, T., Swearingen, C., & Kales, A. Are over-the-counter sleep medications effective? All night EEG studies. *Current Therapeutic Research, 1971, 13*, 143-151.


