Models of Sleep Disturbance in Primary Insomnia and Major Depressive Disorder

and Research Portfolio

PART ONE

(Part two bound separately)

Linda Harvey (BSc. (Hons.))

August 2001

Submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology within the Faculty of Medicine, University of Glasgow

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Contents

Part 1 (this bound copy)

Acknowledgements

I would firstly like to thank Professor Colin Espie for all his good supervision over the past three years. I would also like to thank him for all the perils of wisdom that saw me through the "darker" moments e.g. "there is never time to panic!"

Thank-you also to Dr Elizabeth Campbell for supervising my small-scale research project.

Thank-you to Dr Paul Fleming for all the advice regarding single subject research designs and for carrying out my inter-rater reliability checks. Thank-you also for all the support.

I am indebted to all the participants who took part in my research study and to all the colleagues/professionals who helped me get in touch with potential participants. Thanks go to the Psychologists in the East and West sectors of Glasgow, staff at Auchinlea House, Easterhouse, staff at Glasgow Association for Mental Health (South West project), staff at Parkhead Health Centre and Townhead Health Centre.

I would also like to say a big thank-you to the many wonderful Clinical Psychologists I have met who have taught me so much clinically and inspired me to "keep going" towards my goal of being a Clinical Psychologist.

And finally, thank-you to the greatest of friends, John, for all the support and never letting me forget what is important in life. To Ralph, Alex, Irene and Christine - words cannot express my gratitude. Thank-you for your unconditional love and support and always believing in me.

CHAPTER 1:SMALL SCALE RESEARCH PROJECT

FACTORS AFFECTING ATTENDANCE AND OUTCOME IN ANXIETY MANAGEMENT GROUPS

Prepared in accordance with the instructions for contributors for the Journal of Mental Health (see appendix 1.1).

Factors Affecting Attendance and Outcome In Anxiety Management Groups

Linda Harvey, Dr Elizabeth Campbell
Department of Psychological Medicine, University of Glasgow

Running Head: anxiety management groups

ABSTRACT

White et al (1990, 1992, 1995 and 1998) have shown the effectiveness of large-scale didactic Anxiety Management (AM) groups for the treatment of Generalised Anxiety Disorder. This study investigated factors that may affect attendance and outcome at such groups. The sample consisted of 95 patients (taken from three AM groups), referred to the Department of Clinical Psychology with a primary anxiety disorder, who had agreed to attend an AM group. Information was collected on duration of problem, motivation to attend the group and expectations about outcome. Patients were also given an ICD-10 diagnosis of a primary anxiety disorder. The results indicated that, in general, few individuals completed treatment and only one individual showed any clinically significant change. The diagnosis of Social Phobia was shown to affect whether a patient completed treatment. Duration of problem was also shown to affect attendance and completion. The effectiveness and efficiency of large-scale didactic AM groups is discussed.

INTRODUCTION

Generalised Anxiety Disorder (GAD) has been described as the most prevalent anxiety disorder e.g. data from the NIMH Epidemiological Catchment Area (ECA) study gives an estimate of the prevalence of GAD as 4% in a community sample (cited in Maser and Cloniger, 1990).

Before the 1990s there were few well controlled outcome studies of GAD. However, in recent years there has been an increase in the number of such studies (e.g. Barlow et al, 1992, Borkovec & Costello, 1993, Butler et al, 1991 and Durham et al, 1994). Most studies suggest that gains made during treatment are maintained at six months post-therapy. However, compared to other anxiety disorders, the more longer-term results are not favourable.

In 1990, White and Keenan carried out pilot work investigating a method of improving therapeutic gains and increasing the number of GAD patients seen. They designed "Stress Control", a six session Anxiety Management (AM) evening class for GAD patients. The course was didactic and had a cognitive behavioural emphasis.

Subsequently, a controlled comparative trial was carried out (White et al, 1992, 1995, 1998). "Stress Control" was shown to produce improvements which were maintained at up to two-year follow-up and which were greater than both a placebo condition and a waiting list control.

In light of this and the high number of anxiety referrals received, the Department of Clinical Psychology in the East of Glasgow runs AM classes based on White et al's model.

Although White et al's model was based on patients with GAD, in the East of Glasgow patients with various anxiety disorders are invited to the group. The rationale is that the educative elements of the class, and the AM strategies taught, can help patients cope with anxiety, independent of the form the anxiety takes. The first research question was:

1. Does anxiety disorder diagnosis affect attendance and/ or treatment outcome ?

The other research questions were:

2. Does the duration of the anxiety problem affect attendance and/ or outcome ?

3. Does higher motivation to attend lead to better attendance and/ or outcome ?

4. Do higher expectations about the usefulness of the group lead to better attendance and/ or outcome ?

An analysis of the factors which are linked to better outcome and lower attrition is important as it can provide indications of the best treatment for individual patients

and helps Clinical Psychologists provide a cost effective service. No studies to date have examined the effects of diagnosis, duration of problem, motivation or expectations on attendance or outcome in anxiety management groups.

METHODOLOGY

Participants

Participants consisted of a total of 95 patients. These comprised: 36 patients, 27 patients and 32 patients who were assessed and invited to attend a five week AM group in September 1998, March 1999 and June 1999 respectively. In addition, five patients attended the assessment interview but felt that group treatment was not appropriate for them and were offered individual appointments. Also, ten patients failed to attend the assessment interview. Information is not available on these 15 patients.

The 95 patients on whom data were available were referred by a variety of professionals including GPs, Psychiatrists, Psychologists and Community Psychiatric Nurses (CPNs). The demographic characteristics of each of the three groups are shown in Table 1.

INSERT TABLE 1

Measures

Diagnosis of primary anxiety disorder, according to the International Classification of Diseases -10th edition criteria (ICD-10, World Health Organisation, 1994) was made

by the Clinical Psychologist at assessment using a non-structured clinical interview. The categories used were Generalised Anxiety Disorder, Agoraphobia, Agoraphobia with Panic Disorder, Panic Disorder and Social Phobia.

Information on duration of anxiety problem (measured in years and months) was gathered at assessment and found in the casenotes.

At week 1 of the class, participants were asked to rate their motivation to attend on a 0-10 scale (where 0= very reluctant to attend, 5= just OK about attending and 10= very keen to attend).

Participants were also asked to rate their expectations about their anxiety symptoms at the end of the class on a 0-10 scale (where 0= I expect my anxiety symptoms to be about the same, 5= I expect my anxiety symptoms to be less, and 10= I expect my anxiety symptoms to be considerably less).

At weeks 1 and 5, participants completed the Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983). Treatment outcome was defined by whether or not there was evidence of clinically significant change on the HADS (decrease of two or more standard deviations from the group mean) for each individual participant.

Regarding attendance, participants were divided into completers (attended three or more sessions), defaulters (attended one or two sessions) and non-attenders (attended zero sessions).

Method

The method (described below) was identical for all three groups.

Patients who had been referred with what appeared to be a primary diagnosis of an anxiety disorder were invited for an assessment interview (patients with what appeared to be OCD or PTSD were not assessed for the group, as treatment in the form of an AM group was not thought to be sufficient for these patients and the group was marketed as a complete treatment in keeping with White et al's model). The same Clinical Psychologist carried out the assessments, in the form of a clinical interview, or was present at the assessments carried out by a Trainee Clinical Psychologist. However, a patient was not invited for assessment if they had been referred by another Clinical Psychologist who had previously assessed them (ICD-10 diagnoses were provided for these patients). Participants were informed about the AM group and invited to attend the next group.

The group ran for a five week period. This was in contrast to White et al's six week class. It was decided by the therapists running the group that the material for two of the weeks could be combined to make the group more efficient. As all the material from White et al's model was covered, shortening the class was not thought to be a significant departure from the model. Two therapists ran the class, one presenting each week. Presenting involved verbally providing further information around relevant points seen on slides. The slides that were used in the class had been produced by White et al. The accompanying handouts for each week had been

derived from White et al's more lengthy booklets to reduce costs. Again, this was not considered to be a significant departure from White et al's model as the salient points included in the handout had been chosen by a Clinical Psychologist who was obviously familiar with anxiety management techniques.

RESULTS

It was decided to combine the data from the three anxiety groups for analysis purposes. The assessment and treatment procedures for each group were virtually identical. In addition, as can be seen from Table 1, the three groups contained similar numbers of males and females and the mean age of patients in each group was similar. The three groups differed only in time of year and in the combinations of two therapists running the group. However, the presentation of the class was very didactic and there was little room for individual style. Therefore, there were no confounding factors across the three groups and so the data from the groups were combined.

The Sample

Data were available on 95 patients.

26 patients attended 3 or more sessions of the 5-week class ("completers").

21 patients attended 1 or 2 sessions ("defaulters").

48 patients attended 0 sessions ("non-attenders").

There were some missing data and, as a result, outcome data were available on 21 completers.

The results will be presented for each research question in turn.

Research Question 1

Attendance

Table 2 shows the numbers of completers, non-attenders and defaulters in terms of diagnosis.

INSERT TABLE 2

Chi-square tests and Fisher's Exact Probability tests were carried out to investigate whether diagnosis affected whether a patient **completed** treatment or not. Thus, for example, the number of patients with GAD who attended three or more sessions as opposed to less than three sessions or none at all was compared to the number of patients who did not have a diagnosis of GAD (i.e. all other patients) who attended three or more sessions as opposed to less than three sessions or none at all. Analyses for the other four diagnoses were carried out in a similar way. The tests revealed that only the diagnosis of Social Phobia affected whether a patient completed or not. These patients were more likely to complete treatment than patients with any other anxiety disorder (Fisher's P exact =0.039; two-tailed; d.f.=1).

Chi-square tests and Fisher's Exact Probability tests were also carried out to determine whether diagnosis affected whether a patient **attended one or more sessions**. For example, the analysis compared patients with GAD who were defaulters or completers as opposed to non-attenders with patients who did not have a diagnosis of GAD but were a defaulter or completer as opposed to a non-attender. They revealed that there was no significant effect between diagnosis and attendance.

Outcome

Table 3 shows the range, mean and standard deviation of pre-treatment HADS scores (anxiety and depression subscales) for the 21 participants who completed treatment. Only one participant showed clinically significant change (decrease of greater or equal to two standard deviations below the mean) on the anxiety subscale of the HADS. This individual had a diagnosis of GAD. These results can only be presented descriptively as the numbers in each group are too small for any further analysis. None of the participants showed clinically significant change on the depression subscale.

INSERT TABLE 3

Research Question 2

Attendance

Table 4 shows the numbers of patients who completed, dropped out or defaulted from treatment in terms of duration of problem. Duration of problem was split into

two categories, less than or equal to five years and greater than five years. These cut-off points were chosen for simplicity (i.e. two categories made analysis manageable taking into account the scale of the project) and because the proportion of individuals falling within each of the two categories was approximately equal.

INSERT TABLE 4

A Chi-square test was carried out to investigate whether duration of problem affected whether a patient **completed** treatment or did not. For example, the number of patients who had had their problem for five years or less who attended three or more sessions as opposed to less than three sessions or none at all was compared to the number of patients who had had their problem for greater than five years and who completed as opposed to attending fewer than three sessions or none at all. The results revealed that there was a significant effect between those who completed treatment and those who did not, in terms of duration of anxiety problem. Significantly more patients completed the group if they had had their anxiety problem for greater than five years ($X = 4.51$; $d.f. = 1$; $p < 0.05$).

A Chi-square test was carried out to investigate whether duration of problem affected whether a patient **attended** the group. Thus the number of patients who had had their problem for five years or less who attended one or more sessions as opposed to not attending at all was compared to the number of patients who had had their problem for greater than five years who attended one or more sessions as opposed to none at all. The results showed that there was a significant effect between those who

attended the group and those who did not, with significantly more patients attending the group if they had had their problem for greater than five years ($X = 4.228$; d.f.=1; $p<0.05$).

Outcome

As stated above only one individual showed clinically significant change on the anxiety subscale of the HADS. This individual had had their problem for less than four years. None of the completers showed clinically significant change on the depression subscale. Again the numbers of completers who showed clinically significant change was too small to allow a between groups comparison between the two categories of "duration of problem".

Research Question 3

Motivational scores should have been available on 47 patients but there were data missing on 6 patients. Therefore, data was available on 41 patients. Of the 47 patients, 26 were completers and 21 were defaulters, however, there was data missing on one completer and 5 defaulters.

Attendance

A Pearson product-moment correlation coefficient was carried out to investigate the relationship between number of sessions attended and motivational ratings given. The results indicated that there was no significant relationship between the two variables ($r=0.07$; $N=41$; $p=0.66$).

Outcome

The number of participants who showed clinically significant change was too small to investigate whether motivational rating given at Week 1 affected outcome.

Research Question 4

There should have been expectations ratings available on 47 patients; 26 completers and 21 defaulters. However, there were data missing on 2 completers and 5 defaulters.

Attendance

A Pearson product-moment correlation coefficient was calculated to determine the relationship between number of sessions attended and expectation ratings. The results indicated that there was no significant relationship between the variables ($r=0.123$, $N=40$, $p=0.451$).

Outcome

As above, the effect of expectations on outcome was not investigated as the numbers who showed clinically significant change were too small for further analysis.

DISCUSSION

It is clear from the results of this study that the attendance rate at the AM groups run in the East of Glasgow was poor. Approximately half of the sample did not attend any sessions and an additional 22% defaulted from treatment after they had attended one or more of the sessions. Although monthly figures from the general service in

the Department in the East of Glasgow are not available on the *number of patients* who do not attend appointments or default from treatment, figures show that approximately 78% of *appointments* are kept, 11% are cancelled and 11% are not attended by out-patients who are "returns" (the AM patients can be considered to be "returns" as they have all attended an assessment interview). It is clear then that the attendance rate at the AM groups is lower than the attendance rate at the general service. An area of future work could be to investigate the reasons that patients give for defaulting or not attending the classes.

In terms of the research questions that were posed, the results indicated that only the anxiety disorder diagnosis of Social Phobia affected whether a patient completed treatment. This was an interesting finding given the nature of the group setting. The findings, though, are consistent with the results of a study carried out by Cox et al (1998) that demonstrated that Social-Phobia patients made significant gains on most of the outcome measures in a Cognitive-Behavioural group therapy programme. Implicit in such results is that Social Phobics were able to attend and complete group therapy. It may be that the didactic style of the group puts such patients at ease as they are not the centre of attention. It would be interesting to directly compare the attendance rates of Social Phobic patients in group settings with rates of attending individual appointments.

The results showed that only one patient out of 21 showed clinically significant change on the anxiety subscale of the HADS. It can be concluded from these results that the anxiety management groups run in the East in Glasgow were not, in general,

clinically effective. As the number of individuals showing clinically significant change on the anxiety subscale was so small the hypotheses regarding factors affecting outcome could not be investigated. It is, perhaps, not surprising that none of the individuals showed clinically significant change on the depression subscale as the classes were predominately concerned with management of anxiety. Depression was mentioned only briefly in the last session.

Of interest were the findings examining whether duration of anxiety problem affected attendance at AM groups which revealed that patients were more likely to attend the group and complete three or more sessions if they had had their anxiety problem for more than five years. It is quite difficult to hypothesise why this might be. Perhaps patients who had had their problem longer were more determined and motivated to change. However, the data did not support this hypothesis. The average motivational score of completers/defaulters who had had their problem less than or equal to five years was 6.04. The average motivational score for patients who had had their problem more than five years and who completed/defaulted was 6.94 i.e. the motivational ratings given by individuals in the two different categories of "duration of problem" were very similar.

Rather surprisingly, the results showed that motivational rating (in terms of motivation to attend the group) given at Week 1 did not affect whether a patient attended the group or completed. (Note that it cannot be assumed that attendance at Week 1 shows motivation as there are various other factors which could account for attendance at Week 1, for example, demand characteristics). The motivational

ratings given at Week 1 reflected this i.e. people were present at Week 1 but gave ratings of "very reluctant to attend"). Perhaps a more valid measure of motivation could have been obtained if motivation had been assessed at the initial assessment interview.

Similarly, there was no link between expectations and attendance/completion. Perhaps a rating scale of 0-10 was too simplistic for measuring expectations. Perhaps expectations would have been better measured by also asking patients what factors they thought would decrease their symptoms. For example, it may be that people expect their symptoms to decrease but one meeting with others with a similar problem is all they think it will take to decrease their symptoms. In such a case a correlation between high expectations and number of sessions attended would not be expected.

The findings from this study have clear implications for future anxiety management groups. It would appear that individuals with Social Phobia or individuals who have had their problem for greater than five years are more likely to complete the group. It is worth considering these factors when deciding on who to invite to the classes. However, this study did not show that these factors were linked to better outcome as the numbers who showed clinically significant change were too small for any further analysis. It could be argued, though, that individuals who attend more sessions i.e. are completers have a better chance of learning different ways of dealing with anxiety and, therefore, have a greater chance of a good outcome.

Before running any more classes it would seem essential that we obtain feed-back from people who were invited to attend the anxiety management classes to investigate the factors which affected their attendance. Questionnaires would be a good way of looking at such issues. The questionnaire could contain open questions and more specific questions looking at factors such as location/surroundings, length of group, number of other people present, degree to which identified with other group members, degree to which related to presenters, demand characteristics e.g. referrer's expectations. Factors which affect attendance have obvious implications for outcome too e.g. if someone only comes to one week of the group they will not learn the skills presented in later weeks which they may have found helpful. Information should also be obtained from "completers" and "defaulters" on the information/techniques that they found useful and those things they did not find helpful. This could lead to more tailoring of the current content of the classes with perhaps greater/less emphasis on certain areas.

Future research is necessary to identify other factors that may affect attendance and outcome e.g. an original aim of this study had been to look at whether previous psychological treatment affected attendance and outcome. However, this is a complex question as details such as number of sessions of previous treatment would have to be taken into account. This was thought to be out with the scope of this study. A more detailed examination of factors which affect attendance/outcome at AM groups could allow these groups to be offered to individuals who are more likely to attend and benefit from them and allow alternative treatments e.g. individual therapy to be offered to other patients.

It is worth considering the difference between the findings of this study and the work of White et al (1990, 1992, 1995 and 1998). White et al have shown that anxiety management groups are well attended and associated with positive outcome in GAD patients. Even considering only the GAD patients in this study, only one patient showed change that was clinically significant. In the East of Glasgow referrals were accepted from a variety of referrers including psychiatrists and CPNs, whereas White et al's patients had all been referred by GPs. It may be that there was a greater severity of problems in the current study. On a related note, one obvious difference between White et al's anxiety management classes and those run in the East of Glasgow was the location. White et al's classes were run in the only GP surgery in Clydebank. Thus when attending the group patients were geographically very close to their referrers. In the East of Glasgow the classes were held in a Health Promotion centre. It may well be that the demand characteristics for patients attending and "getting better" were higher in White et al's population. These factors all deserve future investigation.

CRITICAL ANALYSIS

One potential weakness was that not all patients were assessed by the same Clinical Psychologist. Patients who were referred by a Clinical Psychologist were not re-assessed. However, given the large number of patients and demands on Clinical Psychologists' time, this was unavoidable.

Co-morbidity was not investigated in this study. Patients were given a primary anxiety disorder diagnosis. In light of the well established high co-morbidity among anxiety disorders (e.g. Regier et al, 1990), perhaps the study was over-simplified. Future research could take number and nature of additional diagnoses into account.

The Hospital Anxiety and Depression Scale (HADS, Zigmond and Snaith, 1983) was used as a measure of severity of anxiety. It was utilised because it was quick and easy to administer and it was considered useful to have a measure of depressive symptomatology as the content of the class in Week 5 included dealing with depression (due to the high co-morbidity of anxiety and depression, Regier et al, 1990). It may have been simpler in terms of outcome data to use a scale which provided only one score. The Beck Anxiety Inventory (BAI, Beck, 1988) or the State-Trait Anxiety Inventory (STAI, Spielberger et al, 1983) could have been used.

REFERENCES

Barlow, D.H., Rapee, R.M. and Brown, T.A. (1992). Behavioural treatment of Generalised Anxiety Disorder. *Behaviour Therapy*, **23**, 551-570.

Beck, A. (1990). *Beck Anxiety Inventory*, New York: Psychological Corporation.

Borkovec, T.D. and Costello, E. (1993). Efficacy of applied relaxation and cognitive behavioural therapy in the treatment of Generalised Anxiety Disorder. *Journal of Consulting and Clinical Psychology*, **61**, 611-619.

Butler, G., Fennell, M., Robson, P. and Gelder, M. (1991). A comparison of behaviour therapy and cognitive-behaviour therapy in the treatment of Generalised Anxiety Disorder. *Journal of Consulting and Clinical Psychology*, **59**, 167-175.

Cox, B.J., Ross, L., Swinson, R.P. and Direnfeld, D.M. (1998). A comparison of Social Phobia Outcome Measures in cognitive-behavioural group therapy. *Behaviour Modification*, **22**(3), 285-297.

Durham , R.C., Murphy, T.J.C., Allan, T., Richard, K., Treviling, L.R. and Fenton, G. (1994). A comparison of cognitive therapy, analytical psychotherapy and anxiety management training in the treatment of Generalised Anxiety Disorder. *British Journal of Psychiatry*, **165**, 315-323.

Regier, D.A., Burke, J.D. and Burke, K.C. (1990). Comorbidity of affective and anxiety disorders in the NIMH Epidemiological Catchment Area Program. In J.D. Maser and C. R. Cloniger (Eds.) *Comorbidity of mood and anxiety disorders.* Washington, DC: American Psychiatric Press.

Speilberger, C.D., Gorsuch, R.L., Lushene, R.E., Vagg, P.R. and Jacobs, G.A. (1983). *Manual for the State-Trait Anxiety Inventory.* Palo Alto, Calif.: Consulting Psychologists Press.

White, J. (1998). "Stress control" large group therapy for Generalised Anxiety Disorder: two year follow-up. *Behavioural and Cognitive Psychotherapy*, **26**, 237-245.

White, J. and Keenan, M. (1990). "Stress Control": A pilot study of large group therapy for Generalised Anxiety Disorder. *Behavioural Psychotherapy*, **18**, 143-146.

White, J. Brooks, N., and Keenan, M. (1995). Stress control: a controlled comparative investigation of large group therapy for Generalised Anxiety Disorder: Process of change. *Clinical Psychology and Psychotherapy*, **2**, 86-97.

White, J. Keenan, M., and Brooks, N. (1992). Stress control: a controlled comparative investigation of large group therapy for Generalised Anxiety Disorder. *Behavioural Psychotherapy*, **20**, 97-114.

	Males	Females	Mean Age	Age Range
Group 1	18	18	33.97	18-59
Group 2	15	12	34.81	17-60
Group 3	17	15	34.74	19-52
Overall	50	45	34.48	17-60

Table 1 Demographic characteristics of three AM groups

	Completed	Defaulted	Did not attend
GAD (N=45)	12 (26.67%)	15 (33.33%)	18 (40%)
Panic Disorder (N=34)	8 (23.5%)	4 (11.76%)	22 (64.71%)
Agoraphobia (N=3)	0 (0%)	0 (0%)	3 (100%)
Agoraphobia with Panic (N=7)	3 (42.86%)	1 (14.29%)	3 (42.86%)
Social Phobia (N=6)	3 (50%)	1 (16.67%)	2 (33.33%)

Table 2 Numbers of completers, non-attenders and defaulters in terms of diagnosis

	Mean	Standard Deviation	Range
Anxiety Subscale of HADS	15.29	3.10	9-20
Depression Subscale of HADS	8.57	4.32	1-16

Table 3 Distribution of pre-treatment HADS scores for "completers"

	Completed	Defaulted	Did not attend
5 years or less (N=50)	7 (14%)	10 (20%)	33 (66%)
Greater than 5 years (N=45)	19 (42.22%)	11 (24.45%)	15 (33.33%)

Table 4 Numbers of completers, non-attenders and defaulters in terms of duration of problem

CHAPTER 2: MAJOR RESEARCH PROJECT LITERATURE REVIEW

MODELS OF SLEEP DISTURBANCE IN PRIMARY INSOMNIA AND MAJOR DEPRESSIVE DISORDER- A REVIEW OF THE LITERATURE

This literature review has been prepared in accordance with the instructions to authors for the Journal of Consulting and Clinical Psychology (see appendix 2.1)

Models of Sleep Disturbance in Primary Insomnia and Major Depressive Disorder - A Review of the Literature.

Linda Harvey

Department of Psychological Medicine, University of Glasgow

Running Head: models of insomnia

Abstract

The validity of the concept of sleep disturbance *secondary* to depression is challenged by presenting research that suggests that insomnia may be a risk factor for the development/recurrence of depression. In light of this, it is proposed that putative models of primary insomnia may help increase our understanding of sleep disturbance associated with depression. Models of primary insomnia are, thus, reviewed. The lack of evidence for the conceptual basis of some of the models is discussed and the need for research which attempts to address this is highlighted. The dearth of models, particularly psychological (behavioural, cognitive) and environmental, accounting for sleep disturbance in depression is outlined and the usefulness of an investigation of the extent to which putative models of primary insomnia might apply is highlighted.

Sleep Disturbance in Major Depressive Disorder

Sleep disturbance in major depressive disorder is well documented e.g. one of the DSM-IV criteria (American Psychiatric Association, 1994) is "insomnia or hypersomnia". Numerous studies, mainly using electroencephalography (EEG), have investigated the nature of the sleep disturbance in depression. These studies (e.g. Reynolds and Kupfer, 1987-review, Kupfer, Frank, MacEachran & Grochocinski, 1990, Thase, Kupfer, Fasiezka, Buysse, Simons et al., 1996, Emilien and Maloteaux, 1999) show that there is a sleep continuity disturbance i.e. difficulty in falling asleep, increased time spent awake and early morning awakening as compared to normal controls. In addition, individuals with depression have less slow wave sleep than controls and the preponderance of slow wave activity is shifted from the first to the second non-Rapid Eye Movement (non-REM) period. Finally, a shift in the temporal distribution of REM sleep is seen with a decrease in REM sleep in the second half of the sleep period. This is reflected in shorter REM latencies and longer first REM sleep episodes.

More recently actigraphic recording (Hauri and Wisbey, 1992) has been used to investigate the sleep pattern of depressed patients. An actigraph is a small monitor which is attached to a strap and is worn on the non-dominant wrist to measure motor activity. It contains a microprocessor to record and store wrist activity along with actual clock time. The actigraph can be programmed to detect wrist movement at various epoch intervals. Intervals of one minute or less are usually recommended. Participants in studies are generally asked to wear the actigraph for a minimum of three nights. Estimates of sleep onset latency (SOL), wake time after sleep onset

(WASO), total sleep time (TST), sleep efficiency (SE) and total recording time can be obtained by recovering data from the actigraph through microcomputer software. Agreements between actigraphy and polysomnography in the range 89-98% are generally reported (e.g. Jean-Louis, von Gizycki, Zizi, Fookson, Spielman et al., 1996). The American Sleep Disorders Association (1995) published practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. The authors conclude that actigraphy is most accurate when used to differentiate major periods of sleep and waking. The sleep parameters most closely estimated by actigraphy are TST, SE and WASO. The authors acknowledge the major limitation of actigraphy; namely that body movements only indirectly reflect measures of sleep and cardiorespiratory function.

Several studies have been carried out examining the sleep patterns of depressed patients using actigraphic recording e.g. Lemke, Broderick, Zeitelberger & Wolfgang (1997) examined the relationship between self reports of sleep disturbance and actigraphy over three nights in 52 depressed individuals. They found a statistically significant correlation between the two measures of sleep disturbance. No actigraphic studies to date involving depressed insomniacs have utilised a comparison group of non-depressed individuals.

Insomnia as a Risk Factor for Depression

There is a small body of research literature which suggests that individuals who are at risk for developing a mood disorder (e.g. family history of a mood disorder) show sleep disturbances similar to those seen by individuals actually experiencing a

depressive episode e.g. Krieg, Lauer, Hermle, von Bardeleben, Pollmacher et al. (1990) demonstrated increased awakenings leading to decreased sleep efficiency in individuals at high risk for developing a mood disorder. Giles and Kupfer (1994) found that short REM sleep latency predicted subsequent onset of depression in non-affected first degree relatives of individuals with major depressive disorder.

Ford and Kamerow (1989) and Breslau, Roth, Rosenthal & Andreski (1996) assessed the presence/absence of insomnia and psychiatric disorders using DSM-III/III-R respectively to determine whether sleep disturbance in the general population was a risk factor for the onset of a formal psychiatric diagnosis at a later time. Ford and Kamerow (N=8000, all>18 years of age) reported that 17% of respondents with insomnia but no psychiatric disorder at the first interview met the diagnostic criteria at the second interview, one year later (approximately two times greater than respondents without either sleep disorders or psychiatric disorders at the first interview). Breslau et al (N=1,000, all>18 years of age) found that a diagnosis of insomnia at the first interview with no previous history of psychiatric disorder predicted the onset of new cases of major depressive disorder even after controlling for other subsyndromal depressive symptoms. Eaton, Badawi and Melton (1995) carried out a similar study using an overlapping but larger sample than that used in the Ford and Kamerow study. They calculated the "precursor relative risk" for each diagnostic criterion for depression (i.e. the degree to which each criterion could be said to predict later onset of depression). They found that the risk of later onset depression for the criterion "sleep problems" was second only to "feelings of worthlessness and guilt".

Perlis, Giles, Buysse, Tu and Kupfer (1997) found that depressed patients who suffered a recurrence post-treatment exhibited greater levels of sleep disturbance several weeks prior to the recurrence. However, sleep disturbance was measured only using the relevant item on the BDI. Such a measure has questionable validity. Giles, Jarrett, Roffwarg and Rush (1987) found that reduced REM latency during an episode of depression is a predictor of recurrence. They studied 15 individuals with reduced REM latency pretreatment and 10 with non-reduced REM latency for a period of up to two years post-treatment. After a follow-up period of two years 73.9% of the reduced REM latency group had developed depression whereas 46.7% of the non-reduced REM latency group had developed another episode.

These findings challenge the concept of secondary insomnia i.e. insomnia arising as a result of depression. It is proposed here that the term "insomnia associated with depression" is a better term which deals with this conceptual confusion. In light of the above studies it may be useful to investigate the extent to which models of sleep disturbance in primary insomnia can contribute to our understanding of sleep disturbance associated with depression. Such models will now be reviewed.

Models of Sleep Disturbance in Primary Insomnia

Behavioural Models

Bootzin (1972) was the first to suggest that difficulty falling asleep may be due to a failure to establish discriminative stimuli for sleep or the presence of stimuli

which are discriminative of sleep-incompatible behaviour (Bootzin, Epstein & Wood, 1991). Few studies have attempted to provide evidence for this model.

Kazarian, Howe and Csapo (1979) developed the "Sleep Behaviour Self-Rating Scale" to assess sleep-incompatible behaviours associated with a person's bedroom/bed. They found that the scale differentiated between good and poor sleepers only when the criterion measure was SOL. Haynes, Adams, West, Kamens & Safranek (1982) compared a sample of insomniacs and non-insomniacs (defined by sleep-onset latency) on self-reported sleep-incompatible behaviours. Only one out of the twelve behaviours significantly differentiated the two groups. Methodological differences, though, exist between the two studies e.g. Haynes et al's sample was young (mean age = 23) and consisted of students, whereas Kazarian et al's sample included psychiatric patients. Espie et al (1989), in an evaluation of psychological treatment for insomnia, found that greater than half their sample of insomniacs reported reading or watching TV in bed. The absence of a good sleeper control group, however, prevents any firm conclusions being made. Harvey (2000a) found that insomniacs did not differ from good sleepers on daytime napping, whether they stayed in bed when unable to sleep or the extent to which they engaged in sleep-incompatible activities in the bedroom environment.

Zwart and Lisman (1979) attempted to investigate the components responsible for the efficacy of stimulus control treatments. Participants were students. One condition consisted of Bootzin's stimulus control instructions. Other conditions involved a countercontrol condition (participants were instructed to carry out

behaviours incompatible with the principles of stimulus control) and a waiting list condition. Analyses revealed that stimulus control instructions were a efficacious treatment option. That the bed/bedroom environment had become a discriminative stimulus could not be the mechanism responsible for change, though, as the countercontrol condition improved equally well. Davies et al (1986) also found countercontrol instructions to be effective in a group of older adults. More research is needed to examine this controversial area.

On a related note, it is generally held that temporal factors (i.e. the establishment of a regular sleep schedule) can maintain insomnia (e.g. Spielman, 1987). Although temporal factors do not strictly fall conceptually within an operant model, they are linked as an irregular sleep schedule e.g. compensating for sleep loss by going to bed for longer results in more time spent awake in bed which leads to poor stimulus control.

Environmental/Lifestyle Factors

Lifestyle and environmental factors have been shown to affect sleep. These various factors have been termed "sleep hygiene" (Hauri, 1982). A few studies have shown that insomniacs differ from good sleepers in terms of sleep hygiene e.g. Lacks and Rotert (1986) developed a scale to assess the degree of awareness of sleep hygiene principles and the extent to which they are practiced (Sleep Hygiene Awareness and Practice Scale, SHAPS). They then administered this scale to insomniacs and a group of good sleepers. The results showed that insomniacs were better informed but engaged in more unhealthy habits than good sleepers. The

authors state that "sleep hygiene knowledge and practice appeared to be generally high for both good and poor sleepers suggesting that, in most cases, poor sleep hygiene is unlikely to be a primary cause of disturbed sleep" (p367).

Biological Models

Central Nervous System Arousal

Results from neurochemical and neuropathological studies (e.g. Kales and Kales, 1984, Puca, Bricola &Turella, 1973 and Moruzzi and Magoun, 1949) suggest that sleep is controlled by two processes; the action of a sleep-induction process and the inhibition of an arousal process. Thus, insomnia could be the result of a failure to activate a sleep system or a failure to inhibit an alert process.

Circadian Rhythms - the circadian system is a genetic program that provides a temporal ordering of physiological and behavioural events that appears to be necessary for optimal functioning. Entrainment is the process whereby the natural circadian rhythm becomes aligned with the phase of oscillations in the environment. It has been suggested that insomniacs may have some desynchrony between their circadian rhythms and actual sleep pattern e.g. insomniacs often report going to bed early as a response to having slept poorly on previous nights (Morin, 1993) thus creating a desynchrony. The effective use of bright light exposure to entrain circadian rhythms resulting in improvements in sleep variables (e.g. Campbell, Dawson & Anderson, 1993, Lacks, Wright & Paynter, 1995) supports the notion that circadian rhythms may be altered in insomnia. Further evidence comes from temporary sleep disturbance e.g. insomnia associated with transmeridian flights (e.g.

Boulous, 1998). Such flights result in disturbances in circadian rhythms and sleep disturbance is a common complaint.

Physiological Arousal

It has been hypothesised that heightened physiological arousal causes insomnia. Several studies have demonstrated that insomniacs have heightened autonomic arousal (e.g. higher rectal temperature, vasoconstrictions per minute, skin conductance) than good sleepers during the pre-sleep period/ actual sleep (e.g. Monroe, 1967, Haynes, Fitzgerald, Shute & O'Meary, 1985, Stepanski, Glinn, Fortier, Sicklesteel & Zorick, 1989). However, other studies have failed to replicate these findings (e.g. Gross and Borkovec, 1982). Some studies have shown that insomniacs exhibit increased muscle tension e.g. Freedman and Sattler (1982) found that prior to sleep-onset insomniacs had higher frontalis and chin EMG than good sleepers. The failure of treatments such as biofeedback training to result in muscle tension changes whilst reporting improvements in sleep (Bootzin and Rider, 1997 review) does question the validity of physiological arousal as a maintaining factor in insomnia. More recently studies have examined other neurobiological processes e.g. Bonnet and Arand (1995) showed that insomniacs had a significantly increased oxygen use during the day/ night than good sleepers. Such research requires further investigation.

Cognitive Models

Cognitive Arousal

It has been suggested that primary insomniacs show heightened cognitive arousal which interferes with the sleep process. Cognitive arousal may be expressed in terms of worry, a "racing" mind, rumination, intrusive thoughts, planning or difficulty in controlling exciting thoughts (Morin, 1993).

One of the first cognitive-based theories of insomnia to be postulated was a performance anxiety model. This states that one of the consequences of insomnia is anxiety regarding poor sleep performance. This results in a tendency to try harder at falling asleep which leads to further anxiety when the attempt fails which further inhibits sleep. This model grew from the technique of paradoxical intention (Frankl, 1963). Numerous early case reports (e.g. Relinger, Bornstein & Mungus, 1978, Ascher and Efran, 1978, Espie and Lindsay, 1985) supported the use of paradoxical intention in the treatment of initial insomnia. Insomniacs are told that their paradoxical intention is to try and stay awake for as long as possible rather than continuing the effort to fall asleep. Such instructions are hypothesised to reduce anxiety associated with performance failure. Fogle and Dyal (1983) provided evidence that the mechanism of action of paradoxical treatments does appear to be a reduction in performance anxiety. They hypothesised that instructions to give up all efforts to sleep without trying to stay awake should also be effective as paradoxical intention is effective because it interrupts the anxious efforts of the insomniac to fall sleep. There were three conditions; instructions to give up deliberate sleep efforts without the instruction to stay awake with the aim of improving sleep, the same

instructions with the aim of increasing restedness and nighttime comfort and a self-monitoring placebo condition. All three conditions resulted in improved daily sleep estimates but only the "giving up" groups improved on a measure of performance anxiety.

Further evidence for the link between attempts at control and insomnia comes from a study by Harvey (2000b). Insomniacs and good sleepers were allocated to a suppression condition (instructions consisted of "suppress the thought most likely to dominate your thinking as you get into bed) or a non-suppression condition (instructions consisted of "think about anything as you get into bed, including the thought you would most likely think about as you go to sleep"). Individuals in the suppression condition, regardless of whether or not they had insomnia, reported longer sleep latencies and poorer sleep quality. No studies to date have directly examined whether insomniacs and good sleepers differ on a measure of performance anxiety.

Support for the importance of cognitive overarousal comes from a study by Lichstein and Rosenthal (1980). In a survey of causal attributions of insomnia, 35% of their sample of insomniacs stated that both somatic and cognitive arousal were causal factors of their insomnia whereas 55% claimed that cognitive factors were the main determinant. Both Espie et al (1989) and Harvey (2000b) found that in a sample of insomniacs the most frequently endorsed items of the Sleep Disturbance Questionnaire (a measure of causal attributions of sleeplessness, Espie et al, 1989) were cognitive items.

Experimental manipulations of cognitive arousal have been carried out and support the idea that excessive cognitive arousal leads to sleep disturbance. Gross and Borkovec (1982) found that sleep onset latency increased in 38 good sleepers when they were informed they would have to give a speech following sleep. Haynes, Adams & Franzen (1981) found that exposure to a mild stressor at bedtime led to increased sleep onset latency in 10 good sleepers.

There was then a shift in emphasis in the literature from cognitive arousal per se to the nature of the cognitions themselves/their affective valence. Kuisk, Bertelson & Walsh (1989) examined the thought content of 16 insomniacs and 8 good sleepers when lying in bed. Results showed that insomniacs reported more negatively toned thoughts in the pre-sleep period compared with normal sleepers. Borkovec, Lane and Van Oot (1981) compared insomniacs and good sleepers and found that insomniacs reported more intrusive thoughts regarding sleep itself than the control group, in addition to the intrusive thoughts being more worried and negative in content. Coyle and Watts (1991) administered an extended 30-item version of the Sleep Disturbance Questionnaire to a sample of insomniacs. Factor analysis revealed two distinct cognitive factors: "sleep attitudes" (anxiety about the sleep process) and "mental activity" (non-specific cognitive activity). Wicklow and Espie (2000) examined the thought content of 21 insomniacs by asking them to record their thoughts into a voice activated audiorecorder whilst lying in bed during the pre-sleep period. Thinking about sleep, the anticipated consequences of poor sleep and general problem-solving were the strongest predictors of objective sleep latency. Harvey

(2000b) showed that the pre-sleep cognitive activity of insomniacs was characterised by worry about not getting to sleep, general worries, solving problems, the time and noises in the house as compared to good sleepers and less characterised by "nothing in particular".

Morin, Stone, Trinkle, Mercer and Remsberg (1993) asked a sample of older adults with insomnia (N=74) and older adults who were good sleepers (N=71) to complete the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS, Morin, 1993). The DBAS taps various beliefs, attitudes, expectations and attributions about sleep and insomnia. The results showed that insomniacs hold more unrealistic expectations about their sleep requirements and have stronger beliefs about the consequences of insomnia than good sleepers. Morin concluded that dysfunctional beliefs and attitudes about sleep can trigger emotional arousal and feed into the insomnia problem. Edinger, Sullivan, Bastian, Hope & Young (2000) found that insomniacs with subjective insomnia reported more dysfunctional cognitions regarding sleep than individuals with objective insomnia. This provides evidence for the important role of dysfunctional sleep-related cognitions in the subjective complaint of insomnia.

INSERT FIGURE 1

Espie and Wicklow (2001) have put forward a cognitive model of insomnia which focuses on cognitive de-arousal. Figure 1 illustrates this model. Cognitive de-arousal is the main aim and there are four factors which lead to this. Firstly, there

must be minimal processing of information in general. Processing of information is not seen as conducive to the sleep process. Minimal information processing is achieved through reduction of wakefulness or mental activity. Secondly, there is a need for minimal cognitive drive i.e. minimal thinking about themes of emotional importance to the individual e.g. reflection upon problems. This is achieved by a reduction of the necessary work/business carried out by the brain, during the pre-sleep/sleep period. Thirdly, there must be minimal effort to sleep. Espie and Wicklow propose that insomniacs have to learn to reduce trying to control sleep. Finally, minimal affective load i.e. concern or anxiety about daytime life or about sleep itself leads to cognitive de-arousal. Reduction of anxiety is proposed to lead to minimal affective load.

Emotional Arousal

Coyle and Watts (1991) suggest that the psychological make-up of insomniacs may heighten the affective response of insomniacs to poor sleep and trigger dysfunctional sleep cognitions. There is a large literature on the psychological profile of insomniacs. In summary, many studies have administered an array of questionnaires to insomniacs and good sleepers to identify psychological factors associated with and/or potentially mediational to insomnia. The results show that insomniacs more frequently have pathological profiles characterised by elevated measures of anxiety, dysphoria, worry, somatized tension and neuroticism in general (e.g. Edinger, Stout and Hoelscher, 1988, Freedman and Sattler, 1982).

INSERT FIGURES 2 AND 3

Cognitive-Behavioural Conceptualisation

Both Espie (1991) and Morin (1993) have put forward cognitive-behavioural conceptualisations which combine the above models. These conceptualisations are shown in Figures 2 and 3 respectively. Although very similar they do differ in several ways. Espie views arousal as the main difficulty with all the factors discussed above leading to heightened arousal. Morin sees arousal as one element in perpetuating a cycle of insomnia. Espie's model is more inclusive as regards biological factors and Morin's model is more detailed regarding dysfunctional cognitions. Espie and Wicklow's (2001) model described above, however, further develops the cognitive strand of Espie's (1991) original model. Morin also discusses the consequences of insomnia as a maintaining factor in insomnia.

Nonpharmacological Treatments for Primary Insomnia

Models of primary insomnia have given rise to nonpharmacological treatments. These include relaxation training, stimulus control treatments, sleep restriction therapy, sleep hygiene education, paradoxical treatments, strategies to decrease cognitive arousal and approaches aimed at altering maladaptive cognitions. For a detailed description of these treatments see Espie (1991) and Morin (1993). Various meta-analyses and reviews (e.g. Murtagh and Greenwood, 1995, and Morin, Hauri, Espie, Spielman, Buysse et al., 1999, on behalf of the American Academy of Sleep Medicine) have demonstrated the efficacy of these treatments. Multi-component treatment studies confirming the efficacy of CBT have also been published (e.g. Espie , Inglis, Tessier & Harvey, 2001a, Morin, Culbert & Schwartz, 1994). A comprehensive review of the above treatments/ their efficacy is out with the scope of

this review. However, they are relevant to the current discussion in that the mechanisms of these treatments are not well understood e.g. relaxation training is assumed to work by reducing physiological arousal but it could also work by acting as a distracter. Stimulus control procedures are assumed to work by reestablishing discriminative stimuli for sleep. Yet, there is little evidence to support this model of insomnia. Of interest, Bootzin, Lack & Wright (1999) reported that stimulus control instructions, in addition to improving sleep, also reduced anticipatory anxiety. The outcome studies also illustrate that there are many treatments which are efficacious and if it could be shown that models of primary insomnia apply to sleep disturbance associated with depression then the treatment options for this latter population may expand.

Theories of Sleep Disturbance in Depression

Behavioural Theories

Theories of depression have been postulated which are based on conditioning principles. Skinner (1953) was the first to view depression as an extinction phenomenon (behavioural repertoire being weakened due to insufficient reinforcement relative to the effort expanded). Lewinsohn (1976) expanded this theory and stated that depression was due to a low rate of response-contingent positive reinforcement. Lewinsohn's theory is generally viewed as the most influential behavioural theory of depression. Such theories do little to explain sleep disturbance in depression.

Biological Theories

Theories Involving the Central Nervous System (CNS)

Neurochemical theories have been posited e.g. it has been suggested that deficits in central catecholaminergic systems and an increase in cholinergic activity and/or hypersensitivity to neurochemical activity may explain sleep disturbance in depression.

Circadian Rhythms- some of the clinical features of depression, such as diurnal variation in mood and early morning wakening suggest that a disruption of circadian rhythmicity may be present. Various theories have put forward to explain the nature of the disruption of the circadian system. The theories have included the following; there are two or more circadian processes which run at different periods because one or more are not entrained (desynchronisation hypothesis, Halberg, 1968), circadian rhythms are phase-advanced within the 24 hour period (phase advance theory, Wehr and Wirz-Justice, 1982), a process that normally builds up during waking and is dissipated during slow wave sleep is deficient (two process model of sleep regulation, Borbely, Tobler and Wirz-Justice, 1981b), there is a significant reduction in the amplitude of circadian rhythms (biological rhythm amplitude reduction theory, Schultz and Lund, 1983), circadian rhythms have a loss of power or rhythmicity (dysregulation hypothesis, Siever and Davis, 1985) and disrupted social rhythms lead to a disruption in circadian rhythms (Ehlers et al, 1993). A comprehensive review of these studies is out with the scope of this review.

Cognitive Models

Perhaps the most influential cognitive theory of depression is Beck's (1967) theory. Certain core beliefs regarding the world/self (formed in childhood) are dysfunctional in individuals vulnerable to depression. Critical incidents in later life activate these core beliefs. The dysfunctional beliefs lead to errors in information processing which lead to "negative automatic thoughts" which are irrational. Thus individuals with depression are likely to have emotionally laden irrational beliefs and thoughts. No specific link with sleep disturbance is made.

Sleep-based Treatments for Depression

- Chronobiological theories of sleep disruption in depression have led to treatment suggestions e.g. phototherapy, phase advance therapy, total sleep deprivation, partial sleep deprivation and REM sleep deprivation. Some evidence exists to suggest that these treatments are efficacious (e.g. Beauchemin and Hays, 1997, Wehr, Wirz-Justice, Goodwin, Duncan and Gillin, 1979, Wu and Bunney, 1990, Riemann, Konig, Hohagen, Keimen & Voderholzer, 1999, Schilgen and Tolle, 1980, Vogel, 1983). Again, a comprehensive review of sleep-based treatments for depression/ their efficacy is out with the scope of the current review.

Despite the existence of these sleep-based treatments they are rarely recommended in clinical practice. Lichstein et al (1999) in discussing secondary insomnia concludes "the traditional reluctance to treat secondary insomnia stems from the widespread belief among health professionals that direct treatment of the sleep disturbance would be fruitless as long as the primary condition provoking the

insomnia persists, and that sleep improvement will follow amelioration of the primary condition" (National Institutes of Health, 1991, p298). Similarly, a report from the American Psychiatric Association/National Institute of Mental Health DSM-IV field trial (Buysse, Reynolds, Kupfer, Thorpy, Bixler et al.,1997) investigating sleep specialists' and non-sleep specialists' treatment recommendations for insomnia diagnoses showed that sleep specialists' and non-sleep specialists' most common treatment recommendation for the diagnosis of "insomnia related to a mental disorder" was a psychiatric intervention.

Conclusions

- Theories/models of primary insomnia have included biological, psychological and environmental views. Some evidence exists for these theories/models, however, several are still poorly understood (in particular stimulus control, sleep hygiene, physiological arousal and performance anxiety models). In addition, the mechanisms of treatments (derived from these theories) are not well understood. The current study aimed to investigate the evidence for putative models of primary insomnia. Theories explaining sleep disturbance in depression have been mainly biological theories, focusing on circadian rhythms. Given that the concept of insomnia secondary to depression has questionable validity, to what extent can the wider array of models of primary insomnia inform our knowledge of insomnia associated with depression? The answers to such a question could have implications for the treatment of depression, potentially increasing available evidence-based interventions for sleep disturbance associated with depression. Another aim of the proposed study was, thus, to examine to what extent models of primary insomnia apply to insomnia

associated with depression. Only one study to data provides information regarding this question. Broman and Hetta (1994) administered the PSAS (Nicassio, Mendlowitz, Fussell and Petras, 1985) to primary insomniacs (N=22) and individuals with "insomnia associated with affective disorders" (N=18). Both groups obtained very similar mean scores (and standard deviations) on both cognitive and physiological subscales. The absence of a good sleeper control group prevents any real conclusions being made.

REFERENCES

American Psychiatric Association (1994). Diagnostic and Statistical Manual of the Mental Disorders (fourth edition). Washington, DC: American Psychiatric Association.

American Sleep Disorders Association (1995). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. Sleep, 18(4), 285-287.

Ascher, L. M and Efran, J. S. (1978). Use of paradoxical intention in a behavioural program for sleep onset insomnia. Journal of Consulting and Clinical Psychology, 46,- 547-550.

Beauchemin, K. M. and Hays, P. (1997). Phototherapy is a useful adjunct in the treatment of depressed in-patients. Acta Psychiatrica Scandinavica, 95, 424-427.

Beck, A. T. (1967). Depression, Clinical, Experimental and Theorectical Aspects. London: Hoeber Medical Division, Harper and Row.

Bonnet, M. H., and Arand, D. L. (1995). Hour metabolic rate in insomniacs and matched normal sleeepers. Sleep, 18, 581-588.

Bootzin, R. R. (1972). Stimulus control treatment for insomnia. Proceedings of the American Psychological Association, 7, 395-396.

Bootzin, R. R., Epstein, D. and Wood, J. M. (1991). Stimulus control instructions. In Case studies in insommnia, ed. PJ Hauri, 19-28. New York: Plenum Press.

Bootzin, R. R., Lack, L. and Wright, H. (1999). Efficacy of bright light and stimulus control instructions for sleep-onset insomnia. Sleep Suppl., 22, S153-154.

Bootzin, R. R. and Rider, S. P. (1997). Behavioural techniques and biofeedback for insomnia. In Understanding Sleep. The evaluation and treatment of sleep disorders, eds. M. R. Pressman and W. C. Orr, 315-328. Washington DC: American Psychological Association.

Borbely, A. A., Tobler, I. and Wirz-Justice, A. (1981b). Circadian and sleep dependent processes in sleep regulation: outline of a model and implications for depression. Sleep Research, 10, 19.

Borkovec, T. D., Lane, T. W., and Van Oot, P. H. (1981). Phenomenology of sleep among insomniacs and good sleepers. Wakefulness experience when cortically asleep. Journal of Abnormal Psychology, 90, 607-609.

Breslau, N., Roth, T., Rosenthal, L. and Andreski, P. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Social Biological Psychiatry, 39, 411-418.

Broman, J. and Hetta, J. (1994). Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. Nordic Journal of Psychiatry, 48(3), 203-207.

Buysse, D. J., Reynolds, C. F., Kupfer, D. J., Thorpy, M. J. and Bixler, E. et al. (1997). Effects of diagnosis on treatment recommendations in chronic insomnia-a report from the APA/NIMH DSM-IV Field Trial. Sleep, 20(7), 542-552.

Campbell, S. S., Dawson, D. and Anderson, M. W. (1993). Alleviation of sleep maintenace insomnia with timed exposure to bright light. Journal of the American Geriatrics Society, 41, 829-836.

Coyle, K. and Watts, F. N. (1991). The factorial structure of sleep dissatisfaction. Behaviour Research and Therapy, 29, 513-520.

Davies, R., Lacks, P., Storandt, M and Bertelson, A. D. (1986). Countercontrol treatment of sleep-maintenance insomnia in relation to age. Psychology and Aging, 1, 233-238.

Eaton, W. W., Badawi, M. and Melton, B (1995). Prodromes and precursors: epidemiological data for primary prevention of disorders with slow onset. American Journal of Psychiatry, 152, 967-972.

Edinger, J., D., Stout, A.L. and Hoelscher, T. J. (1988). Cluster analysis of insomniacs' MMPI profiles. Relation of subtypes to sleep history and treatment outcome. Psychosomatic Medicine, 50, 77-87.

Edinger, J. D., Sullivan, R. J., Bastian, L. A., Hope, T. V. and Young, M. (2000) et al. Insomnia and the eye of the beholder: are there clinical markers of objective sleep disturbances among adults with and without insomnia complaints? Journal of Consulting and Clinical Psychology, 68, 586-593.

Ehlers, C. L., Kupfer, M. D. Frank, E. and Monk, T. H. (1993). Biological rhythms and depression: the role of zeitgebers and zeitstorers. Depression, 1, 285-293.

Emilien, G. and Maloteaux, J. M. (1999). Circadian system and sleep anomaly in depression. Irish Journal of Psychological Medicine, 16(1), 18-23.

Espie, C. A. (1991). The Psychological Treatment of Insomnia. England: John Wiley.

Espie, C. A. (2002) in press. Insomnia: conceptual issues in the development, maintenance and treatment of sleep disorder in adults. Annual Review of Psychology, 53.

Espie, A. A., Brooks, D. N. and Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. Journal of Behaviour Therapy and Experimental Psychiatry, 20(2), 143-153.

Espie, C. A., Inglis, S. J., Tessier, S. and Harvey, L. (2001a). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a Sleep Clinic in general medical practice. Behaviour Research and Therapy, 39, 45-60.

Espie, C. A. and Lindsay, W. R. (1985). Paradoxical intention in the treatment of chronic insomnia: six case studies illustrating variability in therapeutic response. Behaviour Research and Therapy, 23(6), 703-709.

Espie, C. A. and Wicklow, A. (2001). Cognitive therapy for insomnia. In Sleep and Psychiatry, eds. C. Shapiro and E. P. Sloan, in press. Cambridge, England: Cambridge University Press.

Fogle, D. O and Dyal, J. A. (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. Psychotherapy, Theory, Research and Practice, 20(1), 21-30.

Ford, D. E. and Kamerow, D. B (1989). Epidemiologic study of sleep disturbance and psychiatric disorders: an opportunity for prevention? Journal of American Medical Association, 263, 1479-1484.

Frankl, V. E. (1963). Man's Search for Meaning: An Introduction to Logotherapy. New York: Washington Square Press.

Freedman, R. R. and Sattler, H. L. (1982). Physiological and psychological factors in sleep-onset insomnia. Journal of Abnormal Psychology, 91(5), 380-389.

Giles, D. E. Jarrett, R. B., Roffwarg, H. P. and Rush, A. K. (1987). Reduced REM latency: a predictor of recurrence in depression. Neuropsychopharmacology, 1, 33-39.

Giles, D. E. and Kupfer, D. J. (1994). Reduced REM latency: risk factor for first episode of depression (abstract). Sleep Research, 23, 197.

Gross , R. T. and Borkovec, T. D. (1982). Effects of cognitive intrusion manipulation on sleep-onset latency of good sleepers. Behaviour Therapy, 13, 112-116.

Halberg, F. (1968). Physiological considerations underlying rhythmometry with special reference to emotional illness. In cycles Biologiques et Psychiatrie ed. J. Ajuriaguerra. Paris: Georg.

Harvey, A. (2000a). Sleep hygiene and sleep-onset insomnia. Journal of Nervous and Mental Disease, 188, 53-55.

Harvey, A. G. (2000b). Pre-cognitive activity in insomnia: a comparison of sleep-onset insomniacs and good sleepers. British Journal of Clinical Psychology, 39, 275-286.

Hauri, P. J. (1982). The Sleep Disorders. Kalamazoo, MI: Upjohn.

Hauri, P. J. and Wisbey, J. (1992). Wrist actigraphy in insomnia. Sleep, 15, 293-301.

Haynes , S. N., Adams, A. E., and Franzen, M. (1981). The effects of presleep stress on sleep-onset insomnia. Journal of Abnormal Psychology, 80, 601-606.

Haynes, S. N., Adams, A. E., West, S., Kamens, L. and Safranek, R. (1982). The stimulus control paradigm in sleep-onset insomnia: a multi-method assessment. Journal of Psychosomatic Research, 26, 333-339.

Haynes S. N., Fitzgerald, S. G., Shute, G. and O'Meary, M. (1985). Responses of psychophysiologic and subjective insomniacs to auditory stimuli during sleep. A replication and extension. Journal of Abnormal Psychology, 94, 338-345.

Jean Louis, G., von Gizycki, H., Zizi, F., Fookson, J., Spielman, A. et al. (1996). Determination of sleep and wakefulness with the actigraphic data analysis software. Sleep, 19(9), 739-743.

Kales, A. and Kales, J. (1984). Evaluation and Treatment of Insomnia. New York: Oxford Univeristy Press.

Kazarian, S. S., Howe, M. G. and Csapo, K. G. (1979). Development of the Sleep Behaviour Self-Rating Scale. Behaviour Therapy, 10, 412-417.

Krieg, J. C., Lauer, C. J, Hermle, L., von Bardeleben, U., Pollmacher, T. and Holsboer, F. (1990). Psychometric, polysomnographic and neuroendocrine measures in subjects at high risk for psychiatric disorders: preliminary results. Neuropsychobiology, 23, 57-67.

Kuisk, L. A., Bertelson, A. D. and Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. Perceptual and Motor Skills, 69, 1219-1225.

Kupfer , D. J., Frank, E., McEachran, A. B. and Grochocinski, V. J. (1990). Delta sleep ratio: A biological correlate of early recurrence in unipolar affective disorder. Archives of General Psychiatry, 47, 1100-1105.

Lacks, P. and Rotert, M. (1986). Knowledge and practice of sleep hygiene techniques in insomniacs and good sleepers. Behaviour Research and Therapy, 24, 365-368.

Lacks, L., Wright, H. and Paynter, D. (1995). The treatment of sleep onset insomnia with morning bright light. Sleep research, 24a, 338.

Lemke, M. L., Broderick, A., Zeitelberger, M. and Wolfgang, H. (1997). Motor activity and daily variation of symptom intensity in depressed patients. Neuropsychobiology, 36(2), 57-61.

Lewinsohn, P. M., Biglan, A. and Zeiss, A. M. (1976). Behaviour Treatment of Depression in The Behavioural Management of Anxiety and Depression and Pain, ed. P. O. Davidson. New York: Brunner/Mazel.

Lichstein, K. L. and Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. Journal of Abnormal Psychology, 89, 105-107.

Monroe, L. J. (1967). Psychological and physiological differences between good and poor sleepers. Journal of Abnormal Psychology, 72(3), 255-264.

Morin, C. M. (1993). Insomnia: psychological assessment and management. New York; Guildford Press.

Morin, C. M., Culbert, J. P., and Schwartz, M. S. (1994). Non-pharmacological interventions of insomnia: a meta-analysis of treatment efficacy. American Journal of Psychiatry, 151, 1172-1180.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J. and Bootzin, R. (1999). Nonpharmacologic Treatment of Chronic Insomnia: an Amercian Academy of Sleep Medicine Review, Sleep, 22(8), 1-25.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J. and Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychology and Aging, 8(3), 463-467.

Moruzzi, G. and Magoun, H. W. (1949). Brainstem reticular formation and activation of the EEG. Electroencephalography and Clinical Neurophysiology, 1, 455-473.

Murtagh, D. R. and Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. Journal of Consulting and Clinical Psychology, 63, 79-89.

Perlis, M. L., Giles, D. E., Buysse, D. J., Tu, X and Kupfer, D. J. (1997). Self-reported sleep disturbance as a prodromal symptom in recurrent depression. Journal of Affective Disorders, 42, 209-212.

Puca, F., Bricola, A. and Turella, G. (1973). Effect of L-Dopa or amantadine on sleep spindles in parkinsonism. Electroencephalography and Clinical Neurophysiology, 35, 327-330.

Relinger, H., Bornstein, P. H. and Mungus, D. M. (1978). Treatment of insomnia by paradoxical instruction: a time series analysis. Behaviour Therapy, 9, 955-959.

Reynolds, C. F. and Kupfer, D. J. (1987). Sleep research in affective illness: state of the art circa (review). Sleep, 10(3), 199-215.

Riemann, D., Konig, A., Hohagen, F., Keimen, A., Voderholzer, U. et al. (1999). How to preserve the antidepressive effect of sleep deprivation: a comparison of sleep phase advance and sleep phase delay. European Archives of Psychiatry and Clinical Neuroscience, 249, 231-237.

Schilgen, B. and Tolle, R. (1980). Partial sleep deprivation as therapy for depression. Archives of General Psychiatry, 37, 267-271.

Schultz, H. and Lund, R. (1983). Sleep onset REM episodes are associated with circadian parameters of body temperature: a study in depressed patients and normal controls. Biological Psychiatry, 18, 1411-1426.

Siever, L. J. and Davis, K. L (1985). Overview: toward a dysregulation hypothesis of depression. Amercian Journal of Psychiatry, 142, 1017-1031.

Skinner, B. F. (1953). Science and Human Behaviour. New York: Macmillan.

Spielman, A. J., Caruso, L. S. and Glovinsky, P. B. (1987). A behavioural perspective on insomnia treatment. Psychiatric Clinics of North America, 10, 541-553.

Stepanski, E. J., Glinn, M., Fortier, J., Sicklesteel, J and Zorick, F. J. (1989). Physiological reactivity in chronic insomnia. Sleep Research, 18, 306.

Thase, M. E., Kupfer, D. J., Fasiezka, A. J., Buysse, D. J., Simons, A. D. and Frank, E. (1996). Identifying an abnormal electroencephalographic sleep profile to characterize major depressive disorder. Biological Psychiatry, 41, 964-973.

Vogel, G. W. (1983). Evidence for REM sleep deprivation as the mechanism of action of antidepressant drugs. Progress in Neuropharmacological and Biological Psychiatry, 7, 343-349.

Wehr, T. A. and Wirz-Justice, A. (1982). Circadian rhythm mechanisms in affective illness and antidepressant drug action. Pharmacopsychiatria, 15, 31-39.

Wehr, T. A., Wirz-Justice, A., Goodwin, F. K., Duncan, W. and Gillin, J. C.(1979). Phase advance of the sleep-wake cycle as an antidepressant. Science, 206, 710-713.

Wicklow, A. and Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behaviour Research and Therapy, 38, 679-693.

Wu, J. C. and Bunney, E. E. (1990). The biological basis of an antidepressant response to sleep deprivation and relapse: review and hypothesis. American Journal of Psychiatry, 147, 14-21.

Zwart, C. A. and Lisman, S. A. (1979). Analysis of stimulus control treatment of sleep-onset insomnia. Journal of Consulting and Clinical Psychology, 47, 113-119.

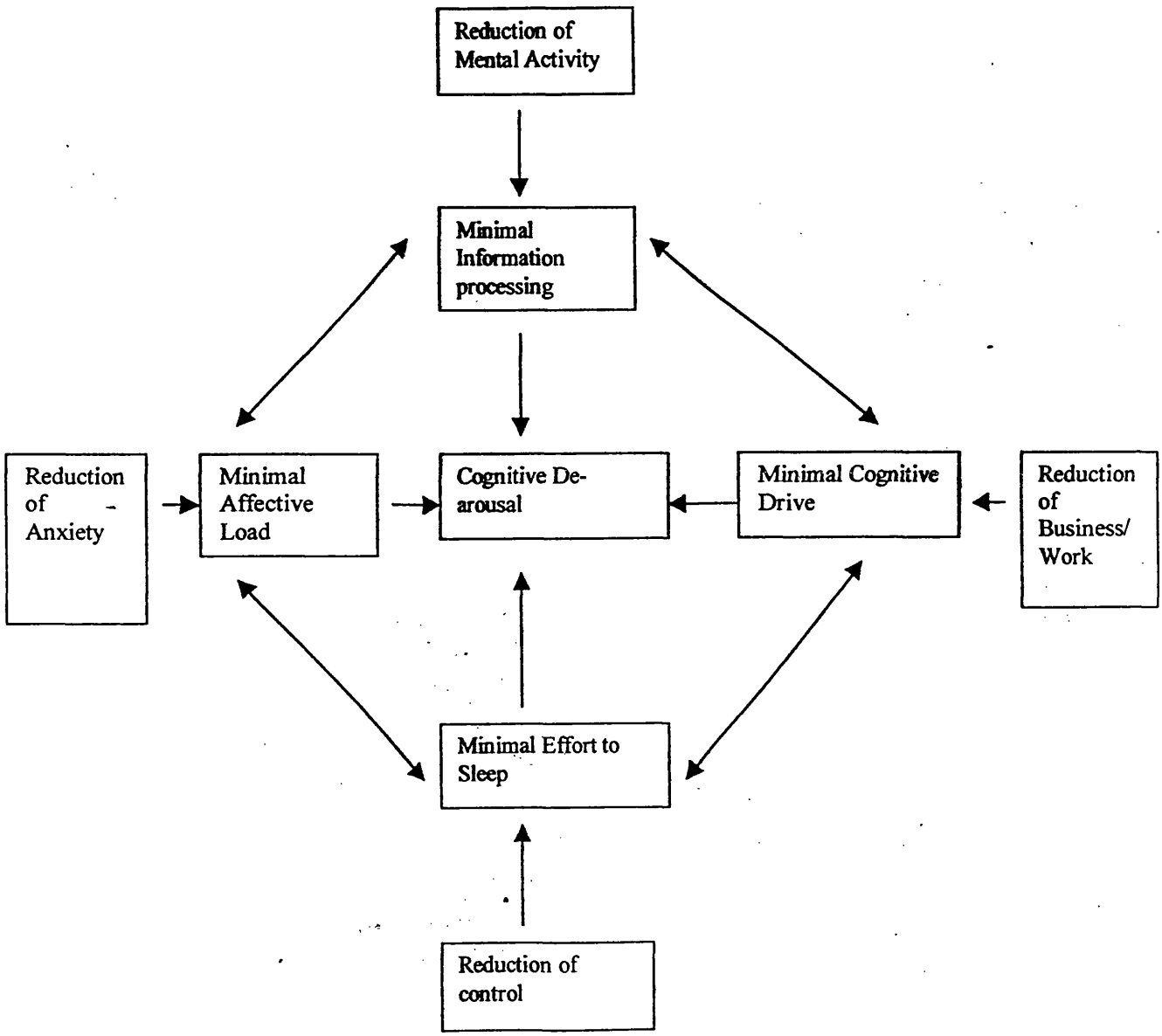

Figure 1 Espie and Wicklow (2000) model of cognitive arousal

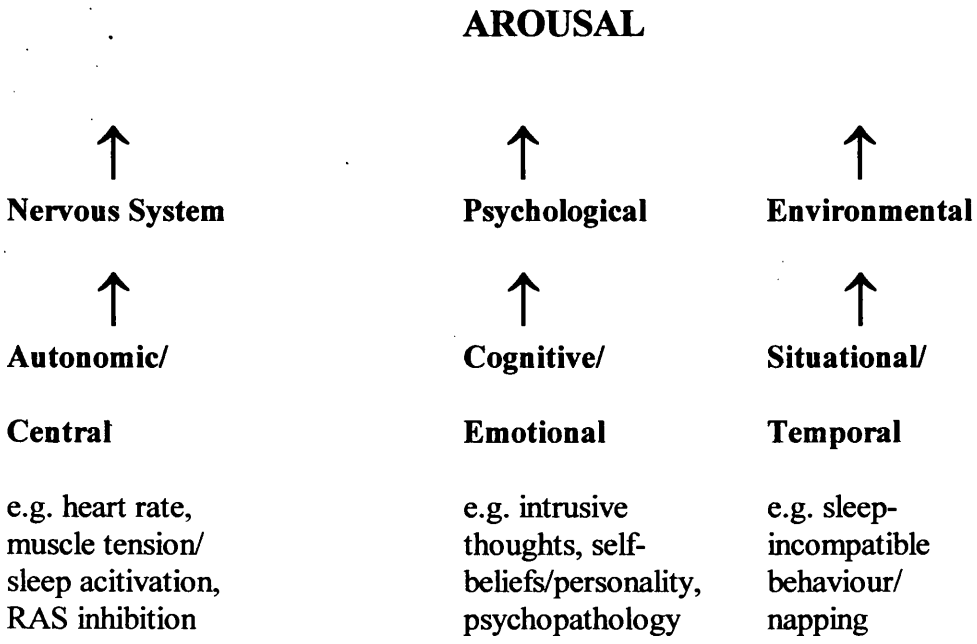

Figure 2 Espie (1991) cognitive-behavioural model

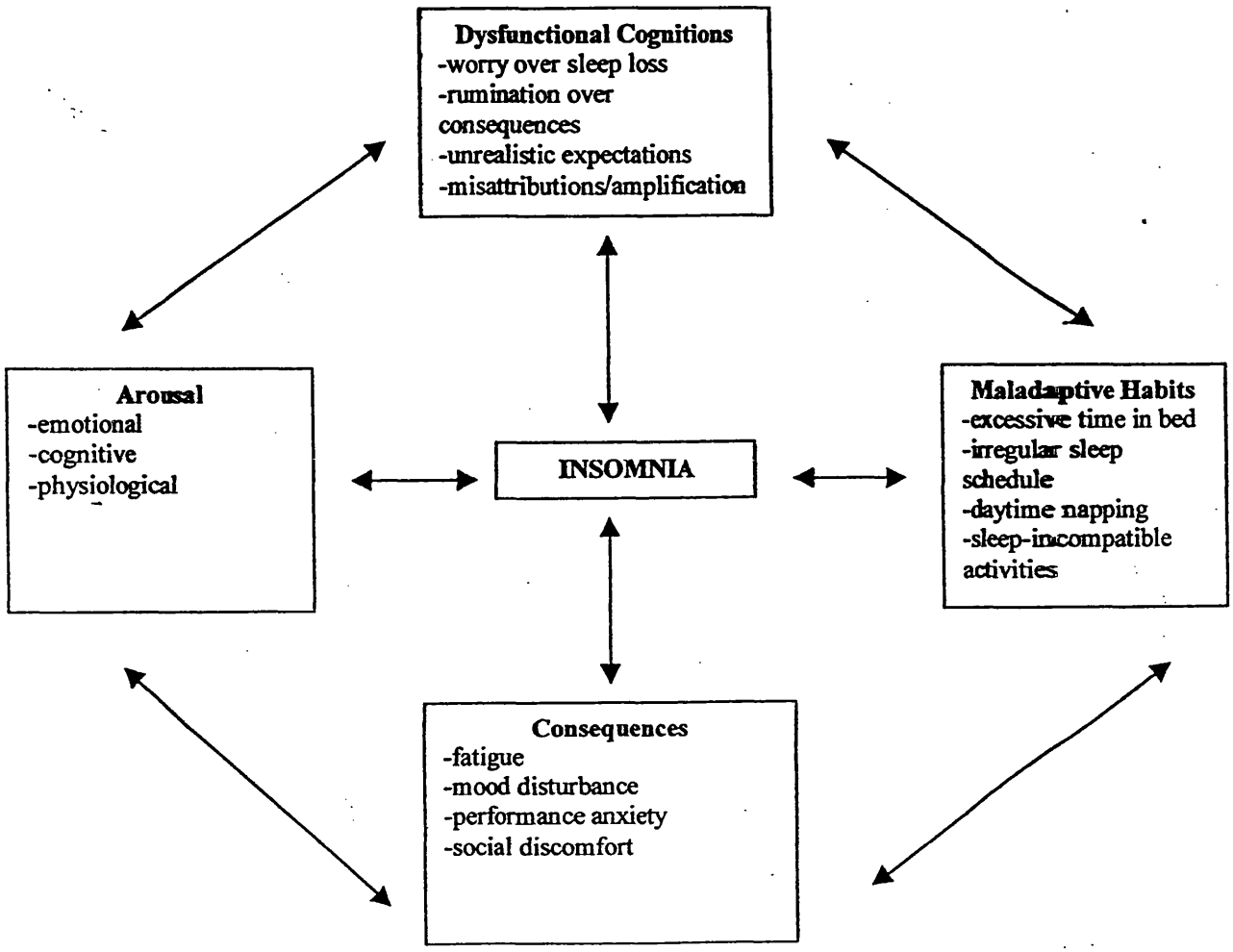

Figure 3 Morin (1993) cognitive-behavioural model

Chapter 3: Major Research Project Proposal

Models of Sleep Disturbance in Major Depressive Disorder and Primary Insomnia

(format, taken from Department of Psychological Medicine's Doctorate in Clinical Psychology course handbook, is based upon the application for a mini-project grant in Health Services Research [SOHHD-Chief Scientist Office])

SUMMARY

Although sleep disturbance secondary to depression is well documented, the theories posited to account for this sleep disturbance have been rather restricted in range, tending to focus on chronobiological theories. Theories accounting for primary insomnia, on the other hand, reflect a wide range of theoretical positions including biological, psychological and environmental views. Various non-pharmacological treatments for insomnia have grown from some of the elements of these theories and several of the treatments have been shown to be efficacious.

This study proposes to examine the extent to which theories thought to explain maintenance factors in primary insomnia can also be applied to sleep disturbance in depression. This would greatly enhance understanding of sleep disturbance in depression. In addition, the study would examine the extent to which there is a theoretical basis to suggest that the non-pharmacological treatments found to be efficacious in primary insomnia could be applied to a secondary insomnia population.

The proposed design is a between groups design. There will be three groups of participants recruited from various health-care settings in Glasgow. The groups will be:

group 1- a control group consisting of non-depressed non-insomniacs.

group 2- non-depressed primary insomniacs

group 3- depressed insomniacs

Sleep, as measured by a history questionnaire, daily logs and actigraphy will be used to help categorise participants into one of the three groups. The presence/absence of depression will be measured using the SCID-I structured interview. The three groups will be compared (using analyses of variance) on sleep measures and their responses to questionnaires assessing sleep hygiene, stimulus control, cognitive and physiological arousal and performance anxiety.

A comparison of the three groups will also allow an investigation of the mechanisms underlying the various non-pharmacological treatments for primary insomnia. The precise mechanisms of these treatments are not fully understood.

INTRODUCTION

Sleep disturbance as a symptom of major depressive disorder is well documented. Indeed one criterion included in DSM-IV (American Psychiatric Association, 1994) is "insomnia or hyposomnia nearly every day".

Over the past twenty years the specific nature of sleep disturbance in individuals suffering from depressive disorders has been investigated by utilising the technique of polysomnography. The results from various studies (e.g. Reynolds and Kupfer, 1987 -review, Kupfer et al, 1990, Thase et al, 1996, Emilien and Maloteaux, 1999) show that the most reliable and predictable sleep abnormalities associated with depression include a sleep continuity disturbance and diminished slow wave sleep. In addition to a general diminution throughout the night, the preponderance of slow wave activity is shifted from the first to the second non-REM period. A shortened REM latency and

an alteration in the temporal distribution of REM sleep (increased REM sleep in the first half of the night) is also reliably seen in depressed individuals.

In recent years attention has been given to the less intrusive and relatively inexpensive technique of actigraphy to measure sleep variables (e.g. Hauri and Wisbey, 1992). There are relatively few studies which utilise actigraphy to examine sleep disturbance associated with major depressive disorder.

Theories have been put forward to explain the sleep disturbance in depression. These have been mainly biological theories, in particular theories of abnormalities of circadian rhythms. Work on circadian pathophysiology has been dominated by the theory that endogenous depressions arise as a result of an aberrant body-clock. The best known proposal has been the phase advance hypothesis. This suggests that there is a clock pathology which drives some, but not all, internal rhythms out of phase with the environment.

Alternative theories have been posited e.g. Siever and Davis (1985) and Schulz and Lund (1985) proposed the circadian dysregulation hypothesis i.e. they proposed that circadian rhythms in depression are characterised by a loss of power or rhythmicity. Evidence for this theory comes form the mechanism of anti-depressants which purportedly work by restoring circadian rhythmicity.

In comparison, however, the theories which have been posited to explain the sleep disturbance in primary insomnia (sleep disturbance which has no apparent physical or

mental illness aetiology) have been far more eclectic, consisting of a variety of biological, psychological and environmental theories. Note that insomnia is defined as difficulty initiating and/or maintaining sleep (International Classification of Sleep Disorders-Revised, 1997).

In terms of biological theories it has been hypothesised that primary insomniacs have heightened physiological arousal. A variety of researchers has shown that compared to good sleepers insomniacs have faster heart rates, increased frontalis muscle tension and greater electrodermal conductance during sleep and in the pre-sleep period (e.g. Haynes et al, 1985). However, some studies have failed to replicate these findings (e.g. Gross and Borkovec, 1982).

It has also been suggested that primary insomniacs show heightened psychological arousal. Evans (1977) was the first to put forward this notion. Interestingly, Lichstein and Rosenthal (1980) found evidence to suggest that insomniacs gave more weight to cognitive arousal as a cause of their sleep disturbance than to physiological arousal. Experimental manipulations of cognitive arousal have also been carried out. Gross and Borkovec (1982) found that sleep onset latency was increased in a sample of good sleepers when they were informed that they would have to give a speech the next day. However, Haynes et al (1981) found that exposure to a mild stressor (arithmetic task) at bedtime decreased sleep onset in insomniacs. These two apparently contrasting pieces of evidence can be explained if the nature of the cognitions themselves are examined.

Kuisk et al (1989) looked at the content of thoughts when participants were lying in bed with the lights out. Insomniacs reported more negative thoughts than a control group. Borkovec et al (1981, 1982) compared insomniacs and good sleepers and found that insomniacs reported more intrusive thoughts regarding sleep itself than the control group, in addition to the intrusive thoughts also being more worried and negative in content. Wicklow and Espie (2000) found that in a group of insomniacs, thinking about sleep and the anticipated consequences of poor sleep and general problem-solving were the strongest cognitive predictors of objective sleep latency.

More generally, there has been the idea that insomniacs, in trait terms, have heightened cognitive arousal. Free-floating anxiety, phobic anxiety, somatic symptoms of anxiety and neurotic depression have all been shown to be elevated in the poor sleeper group (Kumar and Vaidya, 1984).

The idea that insomnia may arise as a response to environmental (situational or temporal) events or be a by-product of the interaction between the sleeper and his environment has also received considerable attention. Bootzin (1972) was the first to suggest an operant analysis of insomnia. He postulated that stimuli associated with sleep become discriminative stimuli for the occurrence of reinforcement in the form of sleep. However, the evidence for such a theory is equivocal with several studies showing that insomniacs do not differ from good sleepers in the extent to which they engage in sleep incompatible behaviours (Haynes et al, 1982). There has been no recent evidence examining this controversy.

Although heightened physiological arousal, heightened cognitive arousal and poor stimulus control can all be hypothesised to have a predisposing, precipitating and perpetuating role in insomnia, the emphasis in the literature has been to give more weight to cognitive arousal and maladaptive sleep habits as perpetuating factors. This is mainly due to the evidence which has been gathered on the efficacy of various treatments.

Various non-pharmacological therapies for the treatment of primary insomnia have been introduced over the past 40 years. These include progressive muscle relaxation training, biofeedback, cognitive therapy, paradoxical intention, stimulus control, sleep restriction, sleep hygiene and multi-component cognitive behaviour therapy. A review of the efficacy of these treatments has been carried out recently by a working group on behalf of the American Academy of Sleep Medicine (Morin et al, 1999). Following on from this review, practice parameters for the non-pharmacological treatment of chronic insomnia were then produced (Chesson et al, AASM, 1999).

The above treatments were derived from elements of the various theoretical positions previously described. For example, relaxation training was a response to the increased autonomic arousal insomniacs were thought to show. However, the actual mechanisms for the various treatments are not well understood e.g. relaxation training could also work by decreasing cognitive arousal by acting as a distractor.

The American Academy of Sleep Medicine Review concludes by stating that research is needed to evaluate the effectiveness of non-pharmacological interventions and to

validate available treatment procedures with patients seeking treatment in various clinical settings and with various co-existing illnesses (secondary insomnia).

The aim of this study is to examine the evidence for and against each of the theoretical positions thought to underlie insomnia and to compare this evidence with comparable data derived from depressed insomniacs. This will help to establish the salience of physiological versus behavioural versus cognitive factors in the presentation of insomnia across two populations compared with controls. The results of this study will also have implications for the treatment of depressed insomniacs. To date only one study has addressed such questions. Broman and Hetta (1994) administered the Pre-Sleep Arousal Scale to a sample of primary insomniacs and a sample of insomniacs with affective disorders. They found that the two groups obtained very similar scores on both the physiological and cognitive subscales of this scale. The absence of a good sleeper control group, however, prevents firm conclusions being made.

AIMS AND RESEARCH QUESTIONS

AIMS-

This is an exploratory study investigating possible maintaining factors and attributions as to the maintenance of sleep disturbance. The study will address the following research questions:

RESEARCH QUESTIONS-

Q1:

Do the sleep patterns of the three groups (depressed insomniacs, primary insomniacs and controls) differ from one another:

a) subjectively?

b) objectively?

Q2:

Do the three groups differ in the extent to which they:

a) report being physiologically aroused when they cannot sleep?

b) report being cognitively aroused when they cannot sleep?

c) experience performance anxiety regarding not being able to sleep?

d) engage in sleep-incompatible activities in bed?

e) engage in pre-bedtime behaviours which adversely affect sleep?

f) possess dysfunctional beliefs regarding the process of sleep?

PLAN OF INVESTIGATION

PARTICIPANTS-

Three groups of participants will be used:

Group 1 - good sleepers who are not depressed (age 18-65). These individuals will form an opportunistic sample.

Inclusion criteria: individuals will also show a) a sleep-onset latency (SOL) of <30 minutes occurring on at least 5 nights out of 7 per week b) sleep efficiency of >85% occurring on at least 5 nights out of 7 per week and c) nocturnal awakenings totaling less than 30 minutes of wakefulness after sleep onset occurring on at least 5 nights out of 7 per week (based on a combination of DSM-IV, ICSD criteria and those typically used in clinical research, Morin 1993).

Exclusion criteria: any axis I disorder (according to SCID-I interview) or past history of major depressive episode.

Group 2 - primary non-depressed insomniacs (age 18-65). These individuals will be recruited through GP surgeries in the Glasgow area.

Inclusion criteria: individuals will also show either a) a sleep-onset latency (SOL) of >30 minutes occurring on 3 nights out of 7 per week and/or b) sleep efficiency of <85% occurring on 3 nights out of 7 per week and/or c) nocturnal awakenings totaling more than 30 minutes of wakefulness after sleep onset occurring on 3 nights

out of 7 per week (based on a combination of DSM-IV, ICSD criteria and those typically used in clinical research, Morin 1993).

Exclusion criteria: any axis I disorder or past history of major depressive episode (according to SCID-I interview), comorbid substance abuse or physical or medical problems causing insomnia.

Group 3 - insomniacs (age 18-65) who are also depressed. These individuals will be recruited through out-patient Clinical Psychology departments in the Glasgow area.

Inclusion criteria: the same sleep criteria used for Group 2. Individuals will also meet DSM-IV criteria for major depressive disorder (according to SCID-I interview).

Exclusion criteria: psychotic depression, any other comorbid axis I disorder, comorbid substance abuse or physical or medical problems causing insomnia (as defined in ICSD-R, 1997). Individuals will also be excluded if their only sleep problem is early morning wakening.

Number of participants

Monroe (1967) was the first to compare sleep patterns of good and poor sleepers (as measured by EEG). Monroe compared 16 good sleepers and 16 poor sleepers. These numbers were sufficiently large to show significant differences between the groups at all sleep stages and in the pre-sleep period. Freedman and Sattler (1981) compared good sleepers and poor sleepers on measures of physiological and

psychological arousal. There were 12 participants in each group and, again, this number of participants was sufficient enough to show significant differences between the two groups in terms of psychological arousal (see introduction for discussion of cognitive arousal and importance of nature of cognitions). These studies are not directly comparable to the current study, however, one of the current study's main measures is the Pre-Sleep Arousal Scale (PSAS, Nicassio, 1985) which provides measures of cognitive and somatic arousal. Power calculations were based on this PSAS measure.

Nicassio et al (1985) investigated somatic and cognitive arousal in a good sleeper group as compared to a poor sleeper group using the Pre-Sleep Arousal Scale (PSAS). There were 30 participants in each group. Power calculations were carried out based on this paper. They revealed that a sample size of 13 per group would be sufficient to detect a significant difference between means (somatic arousal) for a large effect size at power of 0.80 and $p< 0.05$. However, these calculations were based on a one-tailed comparison. Re-analysing, making the analysis more sensitive, showed that 16 participants would be needed per group for a two-tailed comparison. Calculations based on a comparison of cognitive arousal showed that a sample size of 8 would be sufficient to detect a significant difference between means for a large effect size at power 0.80 and $p<0.05$ (one-tailed comparison). For a two-tailed comparison 10 participants per group would be needed.

Based on the results from the power calculations and previous analogous studies, this study will aim to recruit 18 participants in the primary insomniac group and 18 participants in the control group.

In terms of comparing a secondary insomniac group with a primary insomniac group or a good sleeper group few studies have been carried out. Broman and Hetta (1994) compared pre-sleep arousal (somatic and cognitive) in a psychophysiologic insomniac group with a "psychiatric" insomniac group. There were 22 participants in the psychophysiologic group and 18 participants in the insomniac group associated with affective disorders. No data were available from this study on which to base a power calculation. In the absence of any comparable data, this study will also aim to recruit 18 participants in the secondary insomniac group.

MEASURES-

SLEEP

-**Sleep History** questionnaire designed by Espie. Used to obtain self-report measure of history of sleep difficulties and obtain basic information regarding psychological/psychiatric history.

-**Sleep Log** kept for two weeks. Variables of interest will be sleep onset latency (SOL), number of wakenings (WAKE), wake time after sleep onset (WASO), total sleep time (TST) and sleep efficiency (SE).

-**Actigraph** worn for 5 nights and 6 days. As described by Morin (1993), an actigraph is a small monitor worn on the non-dominant wrist which records motor activity (Hauri and Wisbey, 1992). This ambulatory monitoring system uses a

microprocessor to record and store wrist activity along with actual clock time. Data are recovered and stored through microcomputer software. An algorithm is used for estimating sleep-onset latency, wake after sleep onset, total sleep time and total recording time.

DEPRESSION/AXIS I DISORDERS

-**SCID-I** structured interview. The applicant will carry out several practice interviews to become competent at using this interview. The practice interviews will be audio-taped and critiqued by an independent clinician.

-**Beck Depression Inventory** (BDI Beck, et al, 1988) as a measure of severity of depression. The BDI has been shown to have good reliability and validity (Beck et al, 1988).

-**Beck Anxiety Inventory** (BAI, Beck 1990) as a measure of severity of anxiety symptoms. It may be that individuals do not meet DSM-IV criteria for an anxiety disorder but yet show significant anxiety symptomatology. Such information may be important for analysis purposes. The BAI has been shown to have good reliability and validity.

PHYSIOLOGICAL/COGNITIVE AROUSAL

-**Sleep Disturbance Questionnaire** (SDQ, Espie, Brooks and Lindsay, 1989). This 12-item questionnaire provides a self-report measure of physiological and cognitive arousal at bedtime. The SDQ has been shown to be a reliable and valid measure (Espie et al, 2000).

-**Pre-Sleep Arousal Scale** (PSAS, Nicassio et al, 1985). This is a 16-item self-report questionnaire measuring cognitive and somatic arousal states at bedtime. It provides two scores weighing the relative contribution of intrusive cognitions and physiological factors to sleep-onset difficulties. The PSAS has also been shown to have good reliability and validity (Nicassio et al, 1985, Wicklow and Espie, 2000).

- **Dysfunctional Beliefs and Attitudes about Sleep Scale** (DBAS-10 Morin et al, 1993). This 30-item self-report measure is designed to tap cognitions relating to sleep. A 10-item short form will be utilised as it has been demonstrated to have a more robust principal component structure than the original scale (Espie et al, 2000).

SLEEP HYGIENE

-**The Sleep Hygiene Awareness and Practice Scale** (SHAPS, Lacks and Rotert, 1986). This questionnaire provides a measure of the frequency with which participants engage in activities which adversely affect sleep (poor sleep hygiene).

STIMULUS CONTROL

-**Sleep Behaviour Self-Rating Scale** (Kazarian, Howe and Csapo, 1979). Minor alterations will be made to this measure.

PERFORMANCE ANXIETY

Performance anxiety will be measured using an eight-item self-report questionnaire devised by Fogle and Dyal (1983). Participants will be asked to rate how much of a problem four key aspects of sleep performance are to them and then asked to rate the

extent to which their sleeplessness worries them at four points throughout the day/night.

PROCEDURE-

The applicant will contact heads of departments (via letter and telephone) in out-patient Clinical Psychology Departments and all the GPs in 5 GP surgeries in the West of Glasgow to gain permission to use patients in those settings who consent. A letter will firstly be sent outlining the study and requesting permission to use individuals in that particular setting. The letter will be followed up by a telephone call several weeks later to enquire if it would be possible for the applicant to visit and discuss the study in more detail, assuming the professional involved consents to the use of their patients, and to arrange exactly how the applicant will be informed of and contact potential participants. Once potential participants have been identified they will be invited to attend an individual appointment with the applicant. After giving consent, in this first session an outline of the research project will be presented (note that participants will also be asked whether they consent to any of the information they give being passed on to their GP, other medical practitioners involved in their care or to their Psychologist). Participants will then be assessed using the SCID-I interview to determine the presence/absence of depressive disorder, any other axis-I disorder or substance abuse. Participants will then be instructed in completing a daily sleep log which they will be asked to keep for one week and bring with them to the next session (approximately one week later). The actiwatch will also be introduced to participants and they will be asked to wear the watch for five nights and six days (the precise nights/days will be determined) and bring it with them to Session 2. In

Session 2 participants will be asked to complete sleep hygiene, stimulus control, arousal and performance anxiety measures and to keep a sleep diary for another week. Two 1.5 hour sessions are planned to be sufficient to collect the data for each participant.

On the basis of the results from the SCID-I interview, sleep logs and actigraphic data participants will be divided into one of the three groups or discarded from the study. The sleep measures, sleep hygiene, stimulus control, arousal and performance anxiety measures will then be compared across groups.

Following receipt of the second sleep diary the applicant will send an information leaflet to participants with sleep problems. The leaflet entitled the "Good Sleep Guide" outlines some basic techniques for ameliorating sleep problems.

SETTINGS/EQUIPMENT

The study will be carried out in various health-care settings. The applicant will travel to and book rooms in out-patients units or GP surgeries (depending on the source of the participant) in the Glasgow area. Contacts with the control sample will be conducted in the Department of Psychological Medicine.

Wrist actigraphs are currently available in the Department of Psychological Medicine.

DATA ANALYSIS

Data in the form of questionnaires and actigraphy will be stored in a locked filing cabinet in the Department of Psychological Medicine. At the analysis stage unidentifiable data will be stored on a computer and a back-up disk, again in the Department of Psychological Medicine.

As the design of the study is a between groups design and the data will be on at least an interval scale, it is proposed that the data will be analysed using Anova models.

TIME-SCALE

It is proposed that data collection will commence in June 2000 and will take approximately nine months to complete, allowing approximately five months for analysis and write-up.

ETHICAL APPROVAL

Ethical approval is needed for this study. Applications to the Greater Glasgow Primary Care NHS Trust Research Ethics Committee and to the Greater Glasgow Community and Primary Care Local Research Ethics Committee will be made in approximately May 2000.

(see appendices 3.1 and 3.2 for letters granting ethical approval).

REFERENCES

American Psychiatric Association (1993). *Diagnostic and Statistical Manual of Mental Disorders,* 4th edn., Washington, DC; American Psychiatric Association.

American Psychological Association (1997). *International Classification of Sleep disorders-revised (ICSD-R), diagnostic and coding manual.* Minnesota: American Sleep Disorders Association.

Beck, A. R. (1988). *Beck Depression Inventory.* Sidcup: Psychological Corporation.

Beck, A. R. (1990). *Beck Anxiety Inventory.* New York: Psychological Corporation.

Beck, A. T., Steer, R. A. and Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: twenty-five years of investigation. *Clinical Psychology Review, 8,* 77-100.

Bootzin, R. R. (1972). Stimulus control treatment for insomnia. *Proceedings of the American Psychological Association, 7,* 395-396.

Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psychology, 50,* 880-895.

Borkovec, T. D., Lane, T. W. and VanOot, P. H. (1981). Phenomenology of sleep among insomniacs and good sleepers: wakefulness experience when cortically asleep. *Journal of Abnormal Psychology, 6*, 607-609.

Broman, J. E. and Hetta, J. (1994). Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. *Nordic Journal of Psychiatry, 48* (3), 203-207.

Chesson, A. L., Anderson, W. McD., Littner, M., Davila, D., Hartse, K., Johnson, S., Wise, M. and Rafecas, J. (1999). Practice parameters for the nonpharmacological treatment of chronic insomnia. An American Academy of Sleep Medicine Report. *Sleep*, 22(8), 1128-1133.

Coates, T. J., Killen, J. D., Marchini, E., Silverman, S., Hamilton, S. and Thoresen, C. E. (1982a). Discriminating good sleepers from insomniacs using all-night polysomnographs conducted at home. *Journal of Nervous and Mental Disease, 170*, 224-230.

Emilien, G. and Maloteaux, J. (1999). Circadian system and sleep anomaly in depression. *Journal of Psychological Medicine, 16*(1), 18-23.

Espie, C. A. (1991). *"The Psychological Treatment of Insomnia"*. Chichester, England: Wiley.

Espie, C. A., Brooks, D. N. and Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behaviour Therapy and Experimental Psychiatry, 20*, 143-153.

Espie, C. A., Inglis, S. J., Harvey, L. and Tessier, S. (2000). Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research, 48*, 1-9.

Evans, F. J. (1977). Subjective characteristics of sleep efficiency. *Journal of Abnormal Psychology*, 86, 561-564.

Fogle, D. O. and Dyal, J. A. (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychotherapy: theory, research and practice*, 20(1), 21-30.

Freedman R. R. and Sattler, H. L. (1981). Physiological and psychological factors in sleep-onset insomnia. *Journal of Abnormal Psychology*, 91 (5), 380-389.

Gross, R. T. and Borkovec, T. D. (1982). Effects of cognitive intrusion manipulation on sleep-onset latency of good sleepers. *Behaviour Therapy, 13*, 112-116.

Hauri, P. J. and Wisbey, J. (1992). Wrist actigraphy in insomnia. *Sleep, 15*, 293-301.

Haynes, S. N., Adams, A. and Franzen, M. (1981). The effects of presleep stress on sleep-onset insomnia. *Journal of Abnormal Psychology*, *90*, 601-606.

Haynes, S. N., Adams, A. E., West, S., Kamens, L. and Safranek, R. (1982). The stimulus control paradigm in sleep-onset insomnia: A multimethod assessment. *Journal of Psychosomatic Research*, 26, 333-339.

Haynes, S. N., Fitzgerald, S. G., Shute, G. and O'Meary, M. (1985). Responses of psychophysiologic and subjective insomniacs to auditory stimuli during sleep: a replication and extension. *Journal of Abnormal Psychology*, 94, 338-345.

Kazarian, S. S., Howe, M. G., and Csapo, K. G. (1979). Development of the sleep behaviour self-rating scale. *Behaviour Therapy*, *10*, 412-417.

Kuisk, K. A., Bertelson, A. D. and Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. *Perceptual and Motor Skills*, *69*, 1219-1225.

Kumar, A. and Vaidya, A. K. (1984). Anxiety as a personality dimension of short and long sleepers. *Journal of Clinical Psychology*, *40*, 197-198.

Kupfer, D. J., Frank, E., McEachran, A. B. and Grochocinski, V. J. (1990). Delta sleep ratio. *Archives of General Psychiatry*, *47*, 1100-1105.

Lacks, P. and Rotert, M. (1986). Knowledge and practice of sleep hygiene techniques in insomniacs and poor sleepers. *Behaviour Research and Therapy, 24,* 365-368.

Lichstein, K. L. and Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology, 89,* 105-107.

Monroe, L. J. (1967). Psychological and physiological differences between good and poor sleepers. *Journal of Abnormal Psychology, 72* (3), 255-264.

Morin, C. M. (1993). *Insomnia, Psychological Assessment and Management.* New York: Guilford Press.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J. and Bootzin, R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep, 22*(8), 1-25.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J. and Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychological Ageing, 8,* 463-467.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J. and Petras, L. (1985). The phenomenology of the pre-sleep state: The development of the Pre-Sleep Arousal Scale. *Behaviour Research and Therapy, 23*, 263-271.

Reynolds, C. F. and Kupfer, D. J. (1987). Sleep research in affective illness: state of the art circa (state-of-the-art-review). *Sleep, 10*, 199-215.

Schulz, H. and Lund, R. (1985). On the origins of early REM episodes in the sleep of depressed patients: a comparison of three hypotheses. *Psychiatric Research, 16*, 65-77.

Siever, L. J. and Davis, K. L. (1985). Overview: toward a dysregulation hypothesis of depression. *American Journal of Psychiatry, 142*, 1017-1031.

Thase, M. E., Kupfer, D. J., Fasiezka, A. J., Buysse, D. J., Simons, A. D. and Frank, E. (1996). Identifying an abnormal electroencephalographic sleep profile to characterise major depressive disorder. *Biological Psychiatry, 41*, 965-973.

Wicklow, A. and Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy*, 38 , 679-693.

CHAPTER 4: MAJOR RESEARCH PROJECT PAPER

MODELS OF SLEEP DISTURBANCE IN PRIMARY INSOMNIA AND MAJOR DEPRESSIVE DISORDER

Prepared in accordance with the instructions to authors for the Journal of Consulting and Clinical Psychology (appendix 4.1)

Models of sleep disturbance in primary insomnia and major depresive disorder

Linda Harvey and Colin A. Espie

University of Glasgow

Running Head: Models of Insomnia

ABSTRACT

This study aimed to investigate the evidence for putative models of primary insomnia by comparing a primary insomniac group and a good sleeper group on measures of stimulus control, sleep hygiene practice, physiological arousal, cognitive arousal, performance anxiety and dysfunctional cognitions. Another aim of the study was to examine to what extent models of primary insomnia apply to insomnia associated with depression, given that there is evidence that insomnia may be a risk factor for the development/recurrence of depression. The above questionnaires were, thus, also administered to a group of depressed insomniacs. Sleep disturbance was measured using diaries and wrist actigraphs. The data were analysed using ANOVA models. The results provided support for sleep hygiene and cognitive models in the maintenance of primary insomnia (models of stimulus control and physiological arousal were not supported by the data). Questionnaire responses from the depressed insomniac group suggested that poor sleep hygiene and cognitive factors may also be involved in the maintenance of insomnia associated with depression. The results are discussed in the context of existing models/treatments of primary insomnia. The idea of insomnia being a prodrome to depression is also discussed.

INTRODUCTION

Insomnia is defined as "an almost nightly complaint of insufficient amount of sleep, or of not feeling rested after the sleep episode. It is accompanied by impairment of social or occupational functioning and is associated with feelings of restlessness, irritability, anxiety, daytime fatigue and tiredness" (International Clasification of Sleep Disorders [American Psychological Association], 1997, p23). Various models of primary insomnia have been postulated (see Harvey, 2001 for a review). For example, an operant analysis has been put forward. This view proposes that insomniacs have poor stimulus control regarding the process of sleep/sleep environment i.e. the bedroom environment is is not an effective cue for sleep. Insomniacs are hypothesised to frequently engage in behaviours incompatible with sleep in the bedroom environment (e.g. Bootzin, 1972). "Poor sleep hygiene" refers to a high frequency of behaviours that adversely affect sleep e.g. the heavy use of alcohol, tobacco and caffeine. It has been suggested that insomniacs exhibit poor sleep hygiene compared to good sleepers (e.g. Hauri, 1982). It has also been suggested that insomniacs display high physiological arousal in bed e.g. increased heart rate, electrodermal conductance and muscle tension which interferes with the process of sleep (e.g. Haynes, Fitzgerald, Shute and O'Meary, 1985). Various cognitively-based models have also received attention. One of the first was a model of performance anxiety (e.g. Ascher and Efran, 1978, Espie and Lindsay, 1985). This model states that one of the consequences of insomnia is anxiety regarding poor sleep performance. This leads to attempts to try harder to control the process of sleep which leads to further anxiety when the attempt fails which further inhibits sleep. It

has also been postulated that insomniacs generally display cognitive overarousal (worry, a "racing" mind, intrusive thoughts, planning, Morin, 1993) during the pre-sleep period which inhibits sleep (e.g. Lichstein and Rosenthal, 1980, Gross and Borkovec, 1982, Kuisk, Bertelson & Walsh, 1989). The content of insomniacs' thoughts has also been investigated and it has been suggested that, in addition to general cognitive overarousal, anxious thinking about the sleep process/ the anticipated consequences of poor sleep is a major contributor to the maintenance of insomnia (e.g. Coyle and Watts, 1991, Wicklow and Espie, 2000). Morin, Stone, Trinkle, Mercer & Remsberg (1993) hypothesised that insomniacs have "dysfunctional" beliefs/attitudes regarding the process of sleep e.g. regarding their sleep requirements, which maintain insomnia. Cognitive-behavioural conceptualisations that combine some of the above models have also been put forward (e.g. Espie, 1991, Morin, 1993).

These models have given rise to treatments for primary insomnia e.g. relaxation training, stimulus control treatments, sleep restriction therapy and cognitive approaches (see Espie, 1991, Morin, 1993). A number of studies have shown these treatments to be efficacious (e.g. Morin, Culbert and Schwartz 1994, Murtagh and Greenwood, 1995 and Espie, Inglis, Harvey and Tessier, 2000a).

There is some evidence to support the above models of primary insomnia (see Harvey, 2001), however, some areas remain poorly understood. Firstly, there has been a dearth of research in some areas e.g. few studies have directly examined whether insomniacs do display greater performance anxiety or poorer sleep hygiene

than good sleepers. Secondly, there is controversy surrounding certain models e.g. some studies have shown that insomniacs do exhibit greater physiological arousal than good sleepers whereas other studies have provided contradictory evidence (e.g. Haynes et al, 1985 versus Gross and Borkovec, 1982). The same is true of models of stimulus control (e.g. Kazarian, Howe & Csapo, 1979 versus Haynes, Adams, West, Kamens and Safranek, 1982). Thirdly, the mechanisms of treatments are not well understood e.g. relaxation training and stimulus control treatments have been shown to be efficacious. However, if there is little evidence to show that insomniacs do display elevated levels of physiological arousal or poor stimulus control then it is unlikely that these factors are the mechanisms responsible for change.

Some research has demonstrated that insomnia may represent a risk factor for the *development* of depression (e.g. Krieg, Lauer, Hermle, von Bardeleben, Pollmacher et al., 1990, Giles and Kupfer, 1994, Ford and Kamerow, 1989, Breslau, Roth, Rosenthal & Andreski, 1996 and Eaton, Badawi and Melton, 1995). Similarly, Perlis, Giles, Buysse, Tu & Kupfer (1997) demonstrated that greater levels of sleep disturbance were reported several weeks prior to the *recurrence* of a depressive episode in a sample of individuals with recurrent depressive disorder. EEG studies have found the presence of specific EEG abnormalities that predict recurrence in depression (e.g. Giles, Jarrett, Roffwarg & Rugg, 1987 and Buysse, Reynolds, Kupfer, Thorpy, Bixler et al., 1997). For a review of this body of literature see Harvey (2001).

EEG studies have investigated the nature of sleep disturbance in depression (e.g. Reynolds and Kupfer, 1987-review, Kupfer, Frank, McEachran & Grochocinski, 1990, Thase, Kupfer, Fasiezka, Buysse, Simons et al., 1996, Emilien and Maloteaux, 1999). They demonstrate that there is a sleep continuity disturbance and characteristic alterations in sleep stages in individuals with depression. The range of models put forward to explain sleep disturbance in depression is limited. They are primarily biological theories, in particular theories emphasising the role of circadian rhythms e.g. the desynchronisation hypothesis (Halberg, 1968) and phase advance theory (Wehr and Wirz-Justice, 1982) (see Harvey, 2001 for a review). Psychological models of depression do little to directly explain sleep disturbance in depression. Some sleep-based treatments have arisen from the circadian rhythm theories such as phase-advance therapy (e.g. Wehr, Wirz-Justice, Goodwin, Duncan and Gillin, 1979) and partial sleep deprivation therapy (e.g. Schiligen and Tolle, 1980). Some evidence is presented in these papers which suggests that the treatments are efficacious.

This study aimed, firstly, to investigate the evidence for existing models of primary insomnia. Secondly, given the growing body of literature that suggests that sleep disturbance may be a risk factor for the onset/recurrence of depression and given the limited range of models which have been posited to account for sleep disturbance in depression, this study aimed to investigate to what extent models of primary insomnia can be applied to sleep disturbance associated with depression. If it could be shown that these models are applicable then there would be a theoretical basis for suggesting that efficacious treatments for primary insomnia could be applied

to sleep disturbance in depression, thus increasing options for the treatment of depressive symptoms.

METHOD

Research Hypotheses

Before considering the main experimental hypotheses, it was hypothesised that primary insomniacs and depressed insomniacs would show greater sleep-onset latency (SOL), greater wake time after sleep-onset (WASO), decreased total sleep time (TST) and decreased sleep efficiency (SE, ratio of time asleep over total time in bed) compared with good sleepers.

The following hypotheses arise from the psychological literature and were tested in the current study:

Hypothesis 1

Based on the stimulus control model insomniacs (whether primary or depressed) will report engaging in higher levels of sleep-incompatible activities in the bedroom than will good sleepers.

Hypothesis 2

Based on the sleep hygiene model insomniacs will report a higher frequency of pre-bedtime/lifestyle factors which adversely affect sleep than will good sleepers.

Hypothesis 3

Based on the physiological hyperarousal model insomniacs will report higher levels of physiological arousal in bed than will good sleepers.

Hypothesis 4

Based on the cognitive model insomniacs will report higher levels of cognitive overarousal, sleep-related performance anxiety, and "dysfunctional" beliefs/attitudes regarding the process of sleep than will good sleepers.

Due to the model-testing nature of the study, and the limited and/or inconclusive evidence available from previous research, all hypotheses will be tested two-tailed. There is insufficient evidence comparing primary and depressed insomniacs. Therefore, this comparison is posed as a research question.

Question 1

Are there systematic differences between primary and depressed insomniacs in relation to each of the hypotheses concerning psychological models?

Design

To test the research hypotheses an independent groups design was considered appropriate. There were three groups in the study; a good sleeper (Control) group, a primary insomniac (PI) group and a depressed insomniac (DI) group. Comparing a good sleeper group and a primary insomniac group allowed an investigation of models of primary insomnia. Comparing a primary insomniac group and a good sleeper group with a depressed insomniac group allowed an examination of whether models of primary insomnia apply to sleep disturbance associated with depression.

Participants

Power calculations were based on a paper by Nicassio et al. (1985) comparing somatic/cognitive arousal in a good sleeper group with a poor sleeper group using the Pre-Sleep Arousal Scale (PSAS, Nicassio, Mendlowitz, Fussell & Petras 1985). Calculations revealed that 18 participants per group would be sufficient based on a two-tailed comparison for a large effect size at power of 0.80 and $p < 0.05$. Broman and Hetta (1994) compared pre-sleep arousal in a group of primary insomniacs (N=22) with a "psychiatric" insomniac group (N=18). Thus, a sample size of 18 in the depressed insomniac group also seemed appropriate.

Group 1

Good sleepers (opportunistic sample) showed a) a sleep-onset latency (SOL) of <30 minutes occurring on at least 5 nights out of 7 per week b) sleep efficiency of >85% occurring on at least 5 nights out of 7 per week and c) nocturnal awakenings totaling less than 30 minutes of wakefulness after sleep onset occurring on at least 5 nights out of 7 per week as measured by a sleep diary (based on a combination of DSM-IV, ICSD criteria and those typically used in clinical research, Morin 1993).

They were excluded if they met criteria for any axis-I disorder or a past major depressive episode (as assessed by the Structured Clinical Interview for DSM-IV axis-I Disorders (SCID-I clinical version, First, Spitzer, Gibbons & Williams 1997) or a Beck Depression Inventory (BDI, Beck, 1988a) score ≥20. The Beck Anxiety Inventory (BAI, Beck, 1990) was applied descriptively to assess severity of anxiety symptoms.

Inter-rater reliability studies based on an earlier version of the SCID-I interview have yielded kappa values ranging from 0.70 to 1.00 (e.g. Segal, Kabacoff and Hersen, 1995). The BDI rather than the BDI-II was used as normative BDI data from a sample of insomniacs was available from another study (Espie, Inglis, Tessier & Harvey, 2001a). The BDI has been said to possess good reliability and validity (e.g. Beck, Steer and Garbin, 1988b). The BAI has been shown to possess good reliability and validity (e.g. Beck , 1990).

No-one dropped out of the study but two participants were excluded due to temporary illness causing sleep disturbance.

Group 2

Primary insomniacs met one or more of the following criteria for insomnia (taken from Morin, 1993, p6, and based on a combination of Diagnostic and Statistical Manual of Mental Disorders [DSM-IV] criteria (American Psychiatric Association) and ICSD-R criteria) as assessed by a sleep diary:
a) a sleep-onset latency (SOL) of >30 minutes occurring on at least 3 nights out of 7 per week or b) sleep efficiency of <85% occurring on at least 3 nights out of 7 per week or c) frequent or extended nocturnal awakenings totaling more than 30 minutes of wakefulness after sleep onset occurring on at least 3 nights out of 7 per week or any combination of a, b and c.

Exclusion criteria were the same as above but also consisted of any physical or medical problems causing insomnia (as defined in ICSD-R). Seven participants failed to complete screening (a sleep history questionnaire and a sleep diary [content from Espie (1991), appendices 4.2 and 4.3 respectively], three failed to attend, no-one dropped out and two were excluded because they did not meet insomnia criteria for inclusion. Of the final sample, 15 participants formed an opportunistic sample, two were recruited through advertisements and one was recruited through inquiries to the Department of Psychological Medicine.

Group 3

- Depressed insomniacs satisfied sleep disturbance criteria (as above), met DSM-IV criteria for major depressive disorder and obtained a score of ≥ 20 on the BDI (Kendall and Sheldrick, 2000).

Individuals were excluded if they met criteria for any other axis-I disorders (with the exception of anxiety disorders other than post-traumatic stress disorder and obsessive-compulsive disorder) or if they had physical or medical problems causing sleep disturbance or if their only sleep problem was early morning wakening. Two participants failed to complete screening, two failed to attend, two failed to return measures (drop-outs) and eight were excluded (one had a mild learning disability, one a medical complaint causing insomnia, four did not meet criteria for major depressive disorder and two had additional axis-I diagnoses that merited exclusion). Of the final sample, 11 participants were recruited through Clinical Psychology Clinics, three via

the Voluntary Sector, two through Community Teams and two through General Practice Surgeries.

INSERT TABLE 1 HERE

Descriptive information regarding the sample is shown in Table 1. From Table 1 it can be seen that there were more females in the PI group than the other two groups. A Chi-square test, however, showed that this difference was not significant ($X=2.11$; d.f.=2; $p=0.35$). The mean ages of the groups were approximately late thirties/early forties. A one-way analysis of variance and post-hoc Scheffe tests were conducted and revealed that there were no significant between-group differences in terms of age. One-way analyses of variance and post-hoc Scheffe tests showed that the control group had significantly more years education than the insomniac groups (approximately 16 years for the control group and thirteen for the insomniac groups) and that the insomniac groups did not significantly differ in the duration of their sleep problem.

INSERT TABLE 2 HERE

Table 2 provides further descriptive information regarding the final sample. A one-way analysis of variance revealed that scores on the BDI were significantly higher for the DI group than the PI group which in turn were significantly higher than for the control group. A comparison with BDI data obtained from 139 primary insomniacs in a study by Espie et al., 2000a (mean: 10.94, standard deviation: 9.12)

showed that the PI group in the current study obtained very similar scores to this group and the depressed insomniac group scored higher, supporting the idea that the insomniac groups in the current study represented different populations. A one-way analysis of variance also revealed that the depressed insomniac group scored significantly higher on the BAI than both the PI and control groups who did not differ from one another. For the remaining variables in Table 2 analyses of variance were not conducted as differences were likely across the groups. Few individuals in the insomniac groups were prescribed sleeping tablets (1 PI and 2 DI participants). 15 participants from the DI group were on anti-depressant medication.

Measures

Sleep Variables

Sleep Diary (appendix 4.3): a seven-night sleep diary was used as a subjective measure of sleep pattern. Mean measures of SOL, WASO, TST and SE were calculated. Coates, Killen, George, Marchini, Silverman et al., (1982b) showed that daily morning estimates of specific sleep parameters yield a relatively reliable and valid index of insomnia.

Wrist Actigraph: participants wore a wrist actigraph for five nights and six days to provide an objective measure of sleep pattern. An actigraph is a small monitor which is attached to a strap and is worn on the non-dominant wrist to measure motor activity. It contains a microprocessor to record and store wrist activity in relation to time. Estimates of SOL, WASO, TSTand SE can be obtained by recovering data from the actigraph through computer software. The actigraph used was from the

Cambridge Neurotechnology range and was an "actiwatch plus" model. The actigraph was set at a one-minute epoch interval.

Measures Used to Test The Experimental Hypotheses

Sleep Behaviour Self-Rating Scale-adapted (SBSRS, Kazarian et al., 1979 [appendix 4.4], **SBSRS-A,** Harvey and Espie, unpublished [appendix 4.5)- the SBSRS was developed by Kazarian et al. to assess sleep-incompatible behaviours associated with a person's bedroom/ bed. It is the only such published scale. Test-retest correlations ($r=0.88$, $p<0.01$) for the scale indicated that it possessed good reliability. It has also been shown to possess good discriminant validity (Kazarian et al., 1979). The SBSRS was adapted in the current study. Several items were added, based on Bootzin's (1991) stimulus control instructions, to fully address stimulus control principles. The wording of the scale was changed slightly to make it more concise and several items were combined as they were very similar conceptually.

Sleep Hygiene Awareness and Practice Scale (SHAPS, Lacks and Rotert, 1986 - appendix 4.6) - the SHAPS is a 19-item questionnaire which is said to provide a measure of sleep hygiene. It was used as it is the only measure of sleep hygiene practice available in the literature.

Pre-Sleep Arousal Scale (PSAS, Nicassio et al., 1985 - appendix 4.7) - the PSAS is a 16-item questionnaire which provides a measure of physiological and cognitive arousal (two subscale scores). Both scales have been shown to possess internal consistency ($\alpha=0.84$ and 0.81 for good sleepers and insomniacs respectively

[physiological subscale]; α=0.67 and 0.76 [cognitive subscale], Nicassio et al., 1985). Evidence for the construct validity of the PSAS was found in its significant relationship with other theoretically relevant variables.

Sleep Disturbance Questionnaire (SDQ, Espie, Brooks and Lindsay, 1989 - appendix 4.8) - the SDQ is a 12-item questionnaire which "seeks to identify causal attributions concerning the perceived sources of the sleep problem" (Espie , Inglis, Harvey and Tessier, 2000b, p6). Two principal components analyses have been carried out on the SDQ (Espie et al., 1989 and Espie et al., 2000b), yielding a three factor solution (accounting for 68.3% of the variance) and a four factor solution (61%) respectively. Only one factor has reliably emerged in both studies; "attributions concerning mental overactivity". As a result only this factor was considered in the current study, providing a "mental overactivity" score. Espie et al. (2000b) have shown the reliability of the SDQ to be satisfactory (internal consistency [α] = 0.67).

Sleep Anxiety Scale (SAS, Fogle and Dyal, 1983 - appendix 4.9) - Fogle and Dyal developed an eight item questionnaire to measure performance anxiety associated with sleep. The SAS is the only such published questionnaire. The evidence presented for the scale's reliability and validity is unconvincing.

Sleep Performance Anxiety Scale (SPAQ, Espie, Broomfield and Harvey, unpublished - appendix 4.10)- the SPAQ was developed in response to the poor face

validity of the SAS. The SPAQ is hypothesised to measure the various elements involved in a general model of performance anxiety.

Dysfunctional Beliefs and Attitudes about Sleep Scale-10 (DBAS-10, Morin et al., 1993, Espie et al., 2000b - appendix 4.11) -Morin et al. published a 30-item scale (DBAS, 1993) which was reported to measure salient, affect-laden, irrational beliefs concerning the process of sleep. Espie et al. (2000b) found the internal consistency of the various subscales of the original 30-item scale to be poor and proposed a revised 10-item version. Espie et al. found the validity and reliability of this revised version to be acceptable e.g. internal consistency (α) =0.69.

Procedure

Potential participants were sent an information leaflet (appendix 4.12), asked to sign a consent form (appendix 4.13) and complete screening measures. An individual appointment with the researcher was then arranged. At this appointment, actigraphic recording started for six days and five nights and participants were given another sleep diary to start completing the following day. Questionaire measures were taken home to complete (a freepost return envelope was provided). The opportunity of receiving written advice regarding sleep problems, in the form of "the good sleep guide," was given to participants who completed the study. The "good sleep guide" (reproduced from Scottish Health Service Advisory Council report "Management of anxiety and insomnia", 1994, appendix 4.14) is a leaflet with simple, general advice covering the basic elements of psychological treatments of primary insomnia. Limitations on time favoured advice in written format.

RESULTS

SCID-I Diagnoses

To determine the reliability of the diagnoses an independent rater (Consultant Clinical Psychologist) familiar with administration of the SCID-I interview listened to a random sample (determined using random tables) of three audiotaped interviews for each group. The rating for each item in each interview was compared across raters. Cohen's kappa coefficients showed that overall the ratings made by the two raters were identical (kappa=1.000, p=0.000).

Six individuals in the DI group had a comorbid anxiety diagnosis of Generalised Anxiety Disorder, 6 had a diagnosis of Social Phobia, 7 had a diagnosis of Agoraphobia with Panic Disorder and 2 had a diagnosis of Specific Phobia.

Approach to Data Analyses

As the data were on an interval scale and between-group differences were being investigated, Analysis of Variance (Anova) models were considered appropriate. Before proceeding, the other assumptions of Anova models were examined. Frequency distributions of the eight sleep variables and eight variables from the "experimental measures" were plotted separately for each group. Tests of skewness and kurtosis were carried out (appendix 4.15). Using a cut-off range of [-1.96→+1.96] (Hair, Anderson, Tatham and Black, 1998, pp 71-73), there was significant skewness and kurtosis for several of the sleep variables. There was significant kurtosis of the SHAPS distribution for the Control group. However,

Hinkle, Wiersma and Jurs (1994, p337) state that "Anova is robust with respect to violations of the assumptions except in the case of unequal variances with unequal sample sizes".

The Levene Statistic (a test of homogeneity of variances across groups) revealed that many of the variances of the groups were not equal (appendix 4.15). Tabachnick and Fidell (1996, p48) state that "departures from homogeneity of variance only become a significant problem in parametric tests when there are unequal group sizes". Only the SAS variable had slightly unequal group sizes (17 in the DI group due to missing data) and showed heterogeneity of variance. It was, thus, considered appropriate to apply Anova models to the data.

Sleep Variables

Four nights data was obtained for two Control and two DI participants. Three nights data was obtained from, 1 PI and two DI participants. For these individuals mean values for SOL, WASO, TST and SE were calculated for four nights/three nights respectively.

A multivariate analysis of variance (Manova) was applied to confirm that the sleep pattern of the Control group was different from the insomniac groups and to compare the insomniac groups. A Manova was chosen to allow an investigation of the effect of the independent variable on the means of a joint distribution of dependent variables. The four sleep diary variables (SOL, WASO, TST, SE) were entered into a General Linear Model (GLM) Multivariate with "group" as a factor

variable. The procedure was repeated for the four actigraphic variables. There was a significant effect of the factor variable (Wilks' Lambda) for both sleep diary and actigraphic variables : $F(8,96) = 4.22$; $p=0.00$ and $F(8,96) = 2.64$; $p = 0.01$ respectively.

INSERT TABLE 3 HERE

Eight one-way analyses of variance were used to investigate between-group differences as regards the specific sleep variables. Table 3 shows the results. The control group had significantly lower SOL (diary), WASO (diary) and significantly greater TST(diary) and SE (diary) than the PI and DI groups. There were no significant differences between the PI and DI groups. There were no significant between-group differences for WASO (actigraph) or TST (actigraph). The DI group had a significantly greater SOL (actigraph) than the control group and the PI group had a significantly higher SE (actigraph) than the DI group.

Pearson product-moment correlations were carried out to investigate the overall relationship between the sleep diary variables and the respective actigraphic variables. Only the correlation coefficient for SOL reached significance, taking $p=0.05$ (SOL: $r=0.62$, $p<0.01$; WASO: $r=0.04$, $p>0.10$; TST: $r=0.17$, $p>0.10$; SE=0.13, $p>0.10$).

The preliminary research hypothesis was thus supported by the data when sleep diary variables were considered but not actigraphic variables.

INSERT TABLE 4 HERE

Main Experimental Hypotheses

One participant failed to complete the SBSRS-A, one the SAS and three the DBAS-10. To investigate the main experimental hypotheses eight one-way analyses of variance were applied with the following dependent variables: SBSRS-A total score, SHAPS total score, PSAS (physiological sub-scale) score, PSAS (cognitive sub-scale) score, SDQ ("mental overactivity") score, SAS total score, SPAQ total score and DBAS total score. The results are shown in Table 4.

Hypothesis 1 was not supported by the data. There were no significant differences between insomniacs and controls on the SBSRS (measure of frequency of engagement in sleep-incompatible activities in bedroom). Hypothesis 2 was supported by the data. Insomniacs reported more frequent engagement in lifestyle factors which adversely affect sleep (as measured by the SHAPS). Hypothesis 3 was partially supported by the data. The control group and the PI group did not differ and experienced significantly less physiological arousal than the DI group (as measured by the PSAS-physiological subscale). Hypothesis 4 was supported by the data. The insomniac groups reported greater cognitive overarousal than the control group as measured by the PSAS (cognitive subscale) and the SDQ. Both insomniac groups scored significantly higher than the control group on the SAS and SPAQ (performance anxiety measures) and on the DBAS-10 (measure of dysfunctional cognitions regarding sleep).

Returning to research question 1 the DI group scored significantly higher than the PI group on the SHAPS, the PSAS (physiological and cognitive subscales) and the SPAQ.

DISCUSSION

Sleep Patterns

The results confirmed that the sleep pattern of the control group on all variables was significantly different (and in the predicted direction) from that of the insomniac groups (who did not differ from one another) when self-report data was considered. The correlation between subjective and objective measures was poor. This is perhaps not surprising given that they measure different aspects of the experience of sleep. Self-report data are still considered to be the main assessment measure of insomnia (American Sleep Disorder Association practice parameters, 1995).

Models of Primary Insomnia

As regards the behavioural model of primary insomnia (i.e. insomniacs more frequently engage in behaviours in the bedroom environment which are incompatible with sleep) the results from this study do not support such a model. Means from the SBSRS-A showed that there were no between group differences, with all three groups achieving moderate scores (see Table 4, maximum score 60). The results support results from a previous study (Haynes et al, 1982) and suggest that previous controversy in this area can, perhaps, be explained by sample differences. A study

that did find that insomniacs and good sleepers differed included "psychiatric" patients in their sample (Kazarian et al., 1979). The results also support the findings from a study by Zwart and Lisman (1979) who showed that although stimulus control instructions were effective in treating insomnia the mechanism responsible for change was not that the bed/bedroom had become a discriminative stimulus.

The results from this study support the sleep hygiene model of primary insomnia (i.e. insomniacs more frequently engage in behaviours/lifestyle factors that adversely affect sleep) as the PI group scored significantly higher on the SHAPS than the control group. This supports the few studies in the literature which show that primary insomniacs engage in a higher frequency of behaviours that adversely affect sleep than good sleepers.

The model of physiological arousal (i.e. insomniacs are more physiologically aroused than good sleepers) was not supported by the data. The finding from the current study that the PI group and the Control group did not differ on a measure of physiological arousal is consistent with findings from previous studies (e.g. Gross and Borkovec, 1982). Such findings have implications for our knowledge of the mechanisms of effective treatments. For example, relaxation has been shown to be effective (e.g. Morin, Hauri, Espie and Spielman, Buysse et al., 1999) and was originally assumed to work by reducing physiological arousal. The findings from the current study and previous studies suggest that the mechanism of relaxation is not the reduction of physiological arousal.

The model of cognitive overarousal (i.e. that insomniacs are generally more overaroused cognitively than good sleepers) was supported by the data. The finding that the PI group reported significantly greater levels of cognitive overarousal than the control group is supportive of the research literature in this area (see Harvey, 2001) and highlights the importance of cognitive overarousal as a maintaining factor in primary insomnia.

The performance anxiety model of primary insomnia (i.e. anxiety resulting from insomnia results in a tendency to try harder at falling asleep which further inhibits sleep) received support from the current study. On both measures of performance anxiety the PI group scored significantly higher than the control group suggesting that performance anxiety may be a maintaining factor in insomnia. Research examining the role of performance anxiety in insomnia has focused on the effectiveness of paradoxical treatments (assumed to work by reducing performance anxiety). It is thought that the current study makes a useful contribution to this area by directly investigating the validity of a model of performance anxiety. A word of caution is, perhaps, required though. It may be that the construct being measured is simply "effort to sleep". A Pearson product-moment correlation showed that the correlation between the two performance anxiety measures was acceptable ($r=0.89$, $p=0.01$) indicating that they were measuring the same construct. Future research is needed to investigate the structure of these scales.

The cognitive model which states that insomniacs have more dysfunctional beliefs regarding the process of sleep than good sleepers also received support. The PI group endorsed such beliefs to a greater extent than the control group.

As models of stimulus control and physiological arousal were not supported by the data it is unlikely that they are the responsible mechanisms of change for treatments such as relaxation and stimulus control instructions. Given these findings (which are supported by previous studies [Harvey, 2001 review]) it would seem more likely that the mechanisms for these treatments are cognitively-based. The evidence in the literature for differences in cognitive arousal between insomniacs and good sleepers and the effectiveness of cognitive interventions give further weight to this argument. Relaxation could work by decreasing cognitive overarousal or focusing on relaxation (and succeeding) may be a paradoxical treatment in itself, reducing performance anxiety. Stimulus control instructions could also act in these ways and, in addition, could lead to a cognitive shift away from dysfunctional beliefs (Espie, 2002). Being told by a professional, who is ostensibly knowledgeable about sleep, that it is best to get out of bed if you cannot sleep may lead to a shift from the belief that it is important to stay in bed and try hard to sleep.

Models of Sleep Disturbance in Depression

Sleep hygiene models and models of cognitive arousal were also supported by the data for sleep disturbance associated with depression, suggesting that poor sleep hygiene and cognitive factors may also be important in the maintenance of sleep disturbance associated with depression. Examining the results from the DI group

showed that they reported more physiological arousal in bed than both the PI group and the control group suggesting that a model of physiological overarousal may be involved in the maintenance of sleep disturbance in depression. It is perhaps not surprising that physiological factors appear to be more important in the DI group as somatic symptoms are well noted in depression.

The DI group actually scored significantly higher than the PI group on a measure of sleep hygiene practice and several cognitive measures suggesting that such models may be particularly relevant to sleep disturbance associated with depression. Examination of items in the SHAPS indicates why this might be. More individuals in the DI group were prescribed sleeping tablets. Given the nature of depressive illness it could be hypothesised that individuals in the DI group were less likely to exercise during the day or set aside time to relax. It may also be that individuals with depression are more likely to self-medicate with substances that adversely affect sleep such as alcohol or even cigarettes. The pattern of results was consistent with these hypotheses.

The DI group reported higher levels of cognitive overarousal than the PI group (as measured by the PSAS but not the SDQ, note). Insomniacs thoughts during the pre-sleep period have been described as being worried and negative in content (Borkovec, Lane & Van Oot, 1981). Individuals with depression may appraise things even more negatively, resulting in greater rumination of past events or future events during the pre-sleep period. On the SPAQ the DI group scored significantly higher

than the PI group. Although on the SAS there were no significant between-group differences for the DI group and the PI group.

The findings of greater cognitive and physiological arousal (as measured by the PSAS) in depressed insomniacs, as compared to primary insomniacs, are inconsistent with Broman and Hetta's (1994) findings. In their study the two groups of insomniacs scored very similarly on the subscales.

The results are, to some extent, consistent with the idea that insomnia is a prodrome to depression in that the DI group scored significantly higher on several variables than the PI group who scored higher than the Control group. In addition, the similarity between these groups in their responses to measures is consistent with the idea of insomnia as a risk factor for depression.

There were some limitations to this study. There were significant between group differences in BAI scores between the DI group and the PI/Control group (Table 1). It had been hoped that an Analysis of Covariance (Ancova) could be applied to investigate to what extent anxiety could contribute to any observed between-group differences as regards the main experimental hypotheses. Tests of the assumptions of Ancova models (Tabachnick and Fiddell, 1996) revealed that although the data did not violate the assumption of colinearity of variables ($p>0.05$ for all correlations, Field, 2000) the assumption of homogeneity of regression slopes was violated ($p<0.05$ for all variables, Field, 2000) making the application of an Ancova untenable. A word of caution is, therefore, necessary. The results, as

regards the DI group, can only be said to apply to the specific population in this study i.e. depressed insomniacs who also have comorbid anxiety disorders. However, the well documented high comorbidity of anxiety disorders with depression (Rapaport and Mason, 1992; Chambless and Gillis, 1993; Brown and Barlow, 1992) suggests that the sample in the current study was representative of the population of individuals with depression.

Years of education was higher in the control group than the other two groups. This was not considered to be problematic, though, as there was no evidence that this affected the PI/DI groups' participation in the study e.g. ability to competently complete measures.

Medication usage (hypnotics/ anti-depressant medication) was not considered an exclusion criteria for the current study. The effects of hypnotics are generally stated as reducing SOL and increasing TST although they have been reported to suppress REM sleep at high doses (see Espie, 1991 for a review). It may be that individuals prescribed hypnotics showed less sleep disturbance and, as hypnotic usage differed between the two insomniac groups, any comparison of sleep pattern between the two groups was confounded. However, the data did not support a trend of hypnotic usage leading to less severe sleep disturbance. One of the documented side-effects of some tricyclic antidepressants is "movement disorders" (British National Formulary, 2002). It is possible that drugs that affect movement (as a side-effect) could have affected actigraphic recording, resulting in less correlation between subjective/objective measures of sleep in some individuals. However, the correlation

was generally poor. It is also unlikely that such a side-effect adversely affected the subjective experience of sleep, as there were no between-group differences in subjective sleep measures between the insomniac groups.

As few studies have examined the validity of stimulus control and sleep hygiene practice models replication of the current findings is needed. Future research should examine whether cognitive factors are indeed the responsible mechanisms for change in effective interventions and if so, what specific cognitive factors are involved. There is a need for a thorough investigation of the application of models of primary insomnia to sleep disturbance in depression. Already inconsistency exists within the field with the contradictory findings from the current study and Broman and Hetta's (1994) study. Future research needs to attempt to address such inconsistencies.

REFERENCES

American Psychiatric Association (1994). Diagnostic and Statistical Manual of the Mental Disorders (fourth edition). Washington, DC: American Psychiatric Association.

American Psychological Association (1997). International Classification of Sleep Disorders-revised (ICSD-R), diagnostic and coding manual. Minnesota: American Sleep Disorders Association.

American Sleep Disorders Association (1995). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. Sleep, 18(4), 285-287.

Ascher, L. M and Efran, J. S. (1978). Use of paradoxical intention in a behaviouralprogram for sleep onset insomnia. Journal of Consulting and Clinical Psychology, 46, 547-550.

Beck, A. T. (1988a). Beck Depression Inventory (BDI). Sidcup: Psychological Corporation.

Beck, A. T. (1990). Beck Anxiety Inventory (BAI). New York: Psychological Corporation.

Beck, A. T., Steer, R. A. and Garbin, M. G. (1988b). Psychometric properties of the Beck Depression Inventory-twenty five years of investigation. Clinical Psychology Review, 8, 77-100.

Blazer, D. G., Kessler, R. C., McGonagle, K. A. and Swartz, M. S. (1994). The prevalence and distribution of major depression in a national community sample: the national comorbidity survey. American Journal of Psychiatry, 151, 979-986.

Bootzin, R. R. (1972). Stimulus control treatment for insomnia. Proceedings of the American Psychological Association, 7, 395-396.

Bootzin, R. R., Epstein, D. and Wood, J. M. (1991). Stimulus control instructions. In Case studies in insommnia, ed. PJ Hauri, 19-28. New York: Plenum Press.

Borkovec, T. D., Lane, T. W., and Van Oot, P. H. (1981). Phenomenology of sleep among insomniacs and good sleepers. Wakefulness experience when cortically asleep. Journal of Abnormal Psychology, 90, 607-609.

Breslau, N., Roth, T., Rosenthal, L. and Andreski, P. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Social Biological Psychiatry, 39, 411-418.

British National Formulary (BNF, 2002). British Medical Association and Royal Pharmaceutical Society of Great Britain.

Broman, J. and Hetta, J. (1994). Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. Nordic Journal of Psychiatry, 48(3), 203-207.

Brown, T. A. and Barlow, D. H. (1992). Comorbidity among anxiety disorders; implications for treatment and DSM-IV. Journal of Consulting and Clinical Psychology, 60, 835-844.

Buysse, D. J., Reynolds, C. F., Kupfer, D. J., Thorpy, M. J. and Bixler, E. et al. (1997). Effects of diagnosis on treatment recommendations in chronic insomnia-a report from the APA/NIMH DSM-IV Field Trial. Sleep, 20(7), 542-552.

Chambless, D. L. and Gillis, M. M. (1993). Cognitive therapy of anxiety disorders. Journal of Consulting and Clinical Psychology, 61, 248-260.

Coates, T. J., Killen, J. D., George, J., Marchini, E., Silverman, S. and Thoresen, C. E. (1982b). Estimating sleep parameters: a multitrait-multimethod analysis. Journal of Consulting and Clinical Psychology, 50, 345-352.

Coyle, K. and Watts, F. N. (1991). The factorial structure of sleep dissatisfaction. Behaviour Research and Therapy, 29, 513-520.

Eaton, W. W., Badawi, M. and Melton, B (1995). Prodromes and precursors: epidemiological data for primary prevention of disorders with slow onset. American Journal of Psychiatry, 152, 967-972.

Emilien, G. and Maloteaux, J. M. (1999). Circadian system and sleep anomaly in depression. Irish Journal of Psychological Medicine, 16(1), 18-23.

Espie, C. A. (1991). The Psychological Treatment of Insomnia. England: Wiley.

Espie, C. A. (2002) in press. Insomnia: conceptual issues in the development, maintenance and treatment of sleep disorder in adults. Annual Review of Psychology, 53.

Espie, A. A., Brooks, D. N. and Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. Journal of Behaviour Therapy and Experimental Psychiatry, 20(2), 143-153.

Espie, C. A., Inglis, S. J, Harvey, L. and Tessier, S. (2000b). Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. Journal of Psychosomatic Research, 48, 141-148.

Espie, C. A., Inglis, S. J., Tessier, S. and Harvey, L. (2001a). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a Sleep Clinic in general medical practice. Behaviour Research and Therapy, 39, 45-60.

Espie, C. A. and Lindsay, W. R. (1985). Paradoxical intention in the treatment of chronic insomnia: six case studies illustrating variability in therapeutic response. Behaviour Research and Therapy, 23(6), 703-709.

Espie, C. A. and Wicklow, A. (2001). Cognitive therapy for insomnia. In Sleep and Psychiatry, eds. C. Shapiro and E. P. Sloan, in press. Cambridge, England: Cambridge University Press.

Field, A. (2000). Discovering stastistics: using SPSS for windows. London: Sage.

First, M. B., Spitzer, R. L., Gibbon, M. and Williams, J. B. W. (1997). Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), clinician version. Washington: American Psychiatric Press.

Fogle, D. O and Dyal, J. A. (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. Psychotherapy, Theory, Research and Practice, 20(1), 21-30.

Ford, D. E. and Kamerow, D. B (1989). Epidemiologic study of sleep disturbance and psychiatric disorders: an opportunity for prevention? Journal of American Medical Association, 263, 1479-1484.

Frankl, V. E. (1963). Man's Search for Meaning: An Introduction to Logotherapy. New York: Washington Square Press.

Giles, D. E. Jarrett, R. B., Roffwarg, H. P. and Rush, A. K. (1987). Reduced REM latency: a predictor of recurrence in depression. Neuropsychopharmacology, 1, 33-39.

Giles, D. E. and Kupfer, D. J. (1994). Reduced REM latency: risk factor for first episode of depression (abstract). Sleep Research, 23, 197.

Gross, R. T. and Borkovec, T. D. (1982). Effects of cognitive intrusion manipulation on sleep-onset latency of good sleepers. Behaviour Therapy, 13, 112-116.

Hair, J. F., Anderson, R. E., Tatham, R. L. and Black, W. C. (1998). Multivariate data analysis (fifth edition). New Jersey: Prentice Hall.

Halberg, F. (1968). Physiological considerations underlying rhythmometry with special reference to emotional illness. In cycles Biologiques et Psychiatrie ed. J. Ajuriaguerra. Paris: Georg.

Harvey, A. G. (2000b). Pre-cognitive activity in insomnia: a comparison of sleep-onset insomniacs and good sleepers. British Journal of Clinical Psychology, 39, 275-286.

Harvey, L. (2001). Models of sleep disturbance in primary insomnia and major depressive disorder- a review of the literature (unpublished).

Hauri, P. J. (1982). The Sleep Disorders. Kalamazoo, MI: Upjohn.

Haynes, S. N., Adams, A. E., West, S., Kamens, L. and Safranek, R. (1982). The stimulus control paradigm in sleep-onset insomnia: a multi-method assessment. Journal of Psychosomatic Research, 26, 333-339.

Haynes S. N., Fitzgerald, S. G., Shute, G. and O'Meary, M. (1985). Responses of psychophysiologic and subjective insomniacs to auditory stimuli during sleep. A replication and extension. Journal of Abnormal Psychology, 94, 338-345.

Hinkle, D. E., Wiersma, W. and Jurs, S. G. (1994). Applied statistics for the behavioural sciences, third edition. Boston: Houghton Mifflin Company.

Kazarian, S. S., Howe, M. G. and Csapo, K. G. (1979). Development of the Sleep Behaviour Self-Rating Scale. Behaviour Therapy, 10, 412-417.

Kendall, P. C. and Sheldrick, R. C. (2000). Normative data for normative comparisons. Journal of Consulting and Clinical Psychology, 68(5), 767-773.

Krieg, J. C., Lauer, C. J, Hermle, L., von Bardeleben, U., Pollmacher, T. and Holsboer, F. (1990). Psychometric, polysomnographic and neuroendocrine measures in subjects at high risk for psychiatric disorders: preliminary results. Neuropsychobiology, 23, 57-67.

Kuisk, L. A., Bertelson, A. D. and Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. Perceptual and Motor Skills, 69, 1219-1225.

Kupfer , D. J., Frank, E., McEachran, A. B. and Grochocinski, V. J. (1990). Delta sleep ratio: a biological correlate of early recurrence in unipolar affective disorder. Archives of General Psychiatry, 47, 1100-1105.

Lacks, P. and Rotert, M. (1986). Knowledge and practice of sleep hygiene techniques in insomniacs and good sleepers. Behaviour Research and Therapy, 24, 365-368.

Lichstein, K. L. and Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. Journal of Abnormal Psychology, 89, 105-107.

Morin, C. M. (1993). Insomnia: psychological assessment and management. New York; Guildford Press.

Morin, C. M., Culbert, J. P., and Schwartz, M. S. (1994). Non-pharmacological interventions of insomnia: a meta-analysis of treatment efficacy. American Journal of Psychiatry, 151, 1172-1180.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J. and Bootzin, R. (1999). Nonpharmacologic treatment of chronic insomnia: an Amercian Academy of Sleep Medicine Review, Sleep, 22(8), 1-25.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J. and Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychology and Aging, 8(3), 463-467.

Murtagh, D. R. and Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. Journal of Consulting and Clinical Psychology, 63, 79-89.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J. and Petras, L. (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. Behaviour Research and Therapy, 23(3), 263-271.

Perlis, M. L., Giles, D. E., Buyesse, D. J., Tu, X and Kupfer, D. J. (1997). Self-reported sleep disturbance as a prodromal symptom in recurrent depression. Journal of Affective Disorders, 42, 209-212.

Rapaport, M. H. and Mason, J. D. (1992). Secondary depression: definition and treatment. Pharmacology Bulletin, 28, 27-33.

Reynolds, C. F. and Kupfer, D. J. (1987). Sleep research in affective illness: state of the art circa (review). Sleep, 10(3), 199-215.

Reynolds, C. F., Kupfer, D. J., Buysse, D. J., Coble, P. and Yeager, A. (1991). Subtyping DSM-III-R primary insomnia: a literature review by the DSM-IV Work Group on Sleep Disorders. American Journal of Psychiatry, 148, 432-438.

Robins, L. N. and Regier, D. A. (1991). Psychiatric disorders in america; the epidemiological catchment area study. New York: Free Press.

Schilgen, B. and Tolle, R. (1980). Partial sleep deprivation as therapy for depression. Archives of General Psychiatry, 37, 267-271.

Scottish Health Service Advisory Council (SHSAC,1994). A report on the management of anxiety and insomnia. Scottish Office Home and Health Department.

Segal, D. L, Kabacoff, R. I. and Hersen, M. (1995). Update on the reliability of diagnosis in older psychiatric outpatients using the Structured Clinical Interview for DSM-III-R. Journal of Clinical Geropsychology, 1, 313-321.

Tabachnick, B. G. and Fidell, L. S. (1996). Using multivariate statistics. Harper Collins.

Thase, M. E., Kupfer, D. J., Fasiezka, A. J., Buysse, D. J., Simons, A. D. and Frank, E. (1996). Identifying an abnormal electroencephalographic sleep profile to characterize major depressive disorder. Biological Psychiatry, 41, 964-973.

Wehr, T. A. and Wirz-Justice, A. (1982). Circadian rhythm mechanisms in affective illness and antidepressant drug action. Pharmacopsychiatria, 15, 31-39.

Wehr, T. A., Wirz-Justice, A., Goodwin, F. K., Duncan, W. and Gillin, J. C.(1979). Phase advance of the sleep-wake cycle as an antidepressant. Science, 206, 710-713.

Wicklow, A. and Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behaviour Research and Therapy, 38, 679-693.

Zwart, C. A. and Lisman, S. A. (1979). Analysis of stimulus control treatment of sleep-onset insomnia. Journal of Consulting and Clinical Psychology, 47, 113-119.

Table 1 Relevant descriptive information regarding the final sample.

	Control Group	**Primary Insomniac (PI) Group**	**Depressed Insomniac (DI) Group**	**df**	**F**	**p**	**Post hoc (Scheffe) tests**
Males, females	8m, 10f	4m, 14f	7m, 11f	/	/	/	/
Age	Mean: **36.06** SD: **13.23** Range: **25-61**	Mean: **44.39** SD: **14.91** Range: **21-62**	Mean: **39.94** SD: **13.37** Range: **21-64**	2, 51	1.63	0.21	N/A
Years of Education	Mean: **16.11** SD: **2.85** Range: **11-20**	Mean: **13.83** SD: **2.96** Range: **10-20**	Mean: **12.83** SD: **2.60** Range: **11-19**	2, 51	6.46	0.00	C>PI, DI
Duration of sleep Problem (years)	N/A	Mean: **9.47** SD: **10.39** Range: **1-44**	Mean: **9.47** SD: **9.06** Range: **0.5-31**	2, 51	8.5	0.00	DI, PI>C

Table 2 Further relevant descriptive information regarding the final sample

	Control Group	**Primary Insomniac (PI) Group**	**Depressed Insomniac (DI) Group**	**df**	**F**	**p**	**Post hoc (Scheffe) tests**
BDI score	Mean: **1.44** SD: **2.43** Range: **0-9**	Mean: **8.44** SD: **7.11** Range: **0-19**	Mean: **28.56** SD: **8.13** Range: **20-47**	2, 51	87.28	0.00	DI>PI>C
BAI score	Mean: **2.00** SD: **2.72** Range: **0-9**	Mean: **5.61** SD: **5.36** Range: **0-19**	Mean: **25.56** SD: **14.09** Range: **3-48**	2, 51	37.04	0.00	DI>PI,C
Number currently prescribed antidepressant medication	0	1	15	/	/	/	/
Number currently prescribed sleeping tablets	0	1	2	/	/	/	/
Number currently attending Mental Healt Services (NHS professional, voluntary sector)	0	0	15	/	/	/	/

Table 3 Results from the one-way ANOVAs ("group"= the independent variable, sleep variables=the dependent variables). * represents p<0.05, **p<0.01, ***p<0.001

	Control Group	Primary Insomniac Group	Depressed Insomniac Group	df	F	p	Post hoc (Scheffe) tests
Sleep Onset Latency (diary)	mean: **11.02** sd: **9.14**	mean: **41.40** sd: **27.24**	mean: **52.22** sd: **40.47**	2, 51	9.99	0.000***	DI, PI>C
Wake time After Sleep Onset (diary)	mean: **4.72** sd: **5.04**	mean: **60.75** sd: **49.48**	mean: **55.64** sd: **70.28**	2, 51	6.99	0.002**	DI, PI>C
Total Sleep Time (diary)	mean: **446.18** sd: **59.43**	mean: **343.74** sd: **86.34**	mean: **381.94** sd: **120.70**	2, 51	5.66	0.006**	C>PI
Sleep Efficiency (diary)	mean: **95.87** sd: **3.01**	mean: **77.63** sd: **11.68**	mean: **75.79** sd: **18.31**	2, 51	13.84	0.000***	C>PI, DI
Sleep Onset Latency (actigraph)	mean: **8.11** sd: **9.86**	mean: **13.58** sd: **10.41**	mean: **25.99** sd: **31.40**	2, 51	3.81	0.03*	DI>C
Wake time After Sleep Onset (actigraph)	mean: **50.87** sd: **27.20**	mean: **41.47** sd: **17.68**	mean: **63.70** sd: **40.09**	2, 51	2.53	0.09	N/A
Total Sleep Time (actigraph)	mean: **376.78** sd: **43.46**	mean: **417.76** sd: **81.25**	mean: **388.37** sd: **75.56**	2, 51	1.70	0.19	N/A
Sleep Efficiency (actigraph)	mean: **86.81** sd: **5.36**	mean: **87.38** sd: **4.90**	mean: **80.06** sd: **12.97**	2, 51	4.05	0.02*	PI>DI

Table 4 Results from one-way Anovas investigating the effect of "group" on each of the main measures. * represents p<0.05, **p<0.01, ***p<0.001

	Control Group	Primary Insomniac Group	Depressed Insomniac Group	df	F	p	Post hoc (Scheffe) tests
Sleep Behaviour Self Rating Scale-Adapted	**mean:** 33.65 **sd:** 6.54	**mean:** 34.11 **sd:** 7.63	**mean:** 38.89 **sd:** 9.07	2, 50	2.44	0.10	N/A
Sleep Hygiene Awareness and Practice Scale	**mean:** 21.11 **sd:** 10.39	**mean:** 32.89 **sd:** 9.22	**mean:** 43.39 **sd:** 14.65	2, 51	16.45	0.00***	DI>PI>C
Pre-Sleep Arousal Scale (physiological subscale)	**mean:** 8.67 **sd:** 0.91	**mean:** 11.89 **sd:** 4.30	**mean:** 20.44 **sd:** 8.28	2, 51	22.79	0.00***	DI>PI, C
Pre-Sleep Arousal Scale (cognitive subscale)	**mean:** 14.00 **sd:** 3.80	**mean:** 24.94 **sd:** 8.19	**mean:** 30.83 **sd:** 4.29	2, 51	39.46	0.00***	DI>PI>C
Sleep Disturbance Questionnaire score	**mean:** 7.17 **sd:** 1.79	**mean:** 12.33 **sd:** 3.50	**mean:** 13.11 **sd:** 2.08	2, 51	28.50	0.00***	DI, PI>C
Sleep Anxiety Scale	**mean:** 9.17 **sd:** 1.62	**mean:** 17.61 **sd:** 3.63	**mean:** 18.82 **sd:** 2.51	2, 50	66.54	0.00***	DI, PI>C
Sleep Performance Anxiety Questionnaire	**mean:** 7.50 **sd:** 0.62	**mean:** 13.39 **sd:** 3.11	**mean:** 15.39 **sd:** 1.61	2, 51	71.79	0.00***	DI>PI>C
Dysfunctional Belief and Attitudes About Sleep Scale-10	**mean:** 30.07 **sd:** 13.71	**mean:** 47.73 **sd:** 15.92	**mean:** 54.85 **sd:** 21.88	2, 48	12.25	0.00***	DI, PI>C

Chapter 5: Single Subject Research Design (abstract)

The Effects of Distraction On Anxiety During Exposure

Abstract

The purpose of the study was to investigate, using single subject methodology, the effects of two differentially demanding cognitive distracters on anxiety levels during exposure. The research hypotheses were that a dual-task cognitive distracter would inhibit anxiety reduction during exposure whereas a single-task cognitive distracter and an exposure-only condition would result in a decrease in anxiety in a patient with claustrophobia. A study was designed comparing the following experimental conditions: an exposure-only condition, a single task cognitive distractor condition and dual task cognitive distractor condition. Baseline conditions preceded and followed each experimental condition. Conditions were presented in two different orders; the original order and the reverse order. Measures included general anxiety ratings and heart rate. Visual inspection of the data revealed that the trend was for anxiety (measured both subjectively and objectively) to increase or remain at a high level during a single-task and dual-task cognitive distracter compared to a baseline condition. Anxiety during exposure-only tended to remain at low baseline levels but only when conditions were carried out in the original order. Interrupted time-series analyses generally supported these observed trends. The results are discussed in light of theories of anxiety reduction which suggest a negative influence of distraction during exposure and in light of inconsistent findings from studies which vary in terms of the type of distracter used.

CHAPTER 6: APPENDICES

APPENDICES FOR SMALL-SCALE RESEARCH PROJECT (CHAPTER 1)

Appendix 1.1

Notes for Contributors

Journal of Mental Health welcomes original communications and articles which have relevance to the field of mental health. Papers are accepted on the understanding that their contents have not been published elsewhere.

Manuscripts should be sent to the Executive Editor, Professor Ray J. Hodgson, Centre for Applied Public Health Medicine, Lansdowne Hospital, University of Wales College of Medicine, Cardiff CF1 8UL, United Kingdom.

To expedite assessment, 3 complete copies of each manuscript should be submitted. All submissions should be in the style of the American Psychological Association (*Publication Manual*, Fourth edition, 1994). Papers should be typed on one side of the paper, double spaced (including the references), with margins of at least 2.5 cm (1 inch). The first sheet should include the full title of the paper, a short title not exceeding 45 characters (for a running title at the head of each page), names of authors (to include full first name) and the address where the work was carried out. All pages must be numbered. Significant delays may occur to manuscripts that do not conform to journal style. Each article should be accompanied by an abstract of not more than 150 words. Manuscripts should not exceed 6000 words in total, unless previously agreed by the Editor. The full postal address, telephone and fax numbers of the author who will check proofs and receive correspondence and offprints should also be included. Footnotes should be avoided where possible.

To expedite blind reviewing the names of authors should not be displayed on figures, tables or footnotes. The title page is removed before sending to referees.

In order to improve accuracy and expedite publication, authors are requested to submit the *final* and *revised* version of their manuscript on disk. The disk should contain the paper saved in Microsoft Word (preferably for Macintosh), rich text format (RTF) or as a text or ASCII (plain) text file. The disk should be clearly labelled with the author(s) name, paper title, file names and the software used. A good quality copy of the manuscript is *always* required.

References should follow the style of the American Psychological Association. All publications cited in the text should be listed following the text; similarly, all references listed must be mentioned in the text. Within the text references should be indicated by the author's name and year of publication in parentheses, e.g. (Folkman, 1992) or (Sartory & Stern, 1979), or if there are more than two authors (Gallico *et al.*, 1985). Where several references are quoted consecutively, or within a single year, within the text the order should be alphabetical, e.g. (Mawson, 1992; Parry & Watts, 1989) and (Grey, 1992; Kelly, 1992; Smith, 1992). If more than one paper from the same author(s) and year are listed, the date should be followed by (a), (b), etc., e.g. (Cobb, 1992a).

References should be listed alphabetically by author on a separate sheet(s) (double spaced) in the following standard form, capitalisation and punctuation:

a) For periodical articles (titles of journals should *not* be abbreviated):

Rachman, S., Cobb, J., Grey, S.J., McDonald, B., Mawson, D., Sartory, G. & Stern, R. (1979). The behavioural treatment of obsessive-compulsive disorders, with and without clomipramine. *Behaviour Research and Therapy*, 17, 467–478.

b) For books:

Powell, T.J. & Enright, S.J. (1990). *Anxiety and Stress Management*. London: Routledge.

c) For chapters within multi-authored books:

Hodgson, R.J. & Rollnick, S. (1989). More fun, less stress: How to survive in research. In G. Parry & F. Watts (Eds.), *A Handbook of Skills and Methods in Mental Health Research* (pp. 75–89). London: Lawrence Erlbaum.

Journal titles should not be abbreviated and unnecessary references should be avoided. Names of all authors are required.

Clear, grammatical and tabular presentation is strongly encouraged.

Illustrations should not be inserted in the text. Each should be provided separately, and numbered on the back with the figure number and title of the paper. Three copies of all figures must be submitted. All photographs, graphs and diagrams should be referred to as 'Figures' and should be numbered consecutively in the text in Arabic numerals (e.g. Fig. 3). The appropriate position of each illustration should be indicated in the text. A list of captions for the figures should be submitted on a separate sheet and should make interpretation possible without reference to the text. Captions should include keys to symbols. It would help to ensure greater accuracy in the reproduction of figures if the values used to generate them were supplied. Figures should be provided, on disk, in Microsoft Excel.

Tables should be typed on separate sheets and their approximate position in the text should be indicated. Units should appear in parentheses in the column heading but not in the body of the table. Words and numerals should be repeated on successive lines; 'ditto' or 'do' should *not* be used.

Proofs are supplied for checking and making essential corrections, not for general revision or alteration. Proofs should be corrected and returned within 3 days of receipt.

Offprints Fifty offprints of each paper are supplied free. Additional copies may be purchased and should be ordered when the proofs are returned. Offprints, together with a complete copy of the relevant journal issue, are sent approximately 3 weeks after publication.

Copyright. It is a condition of publication that authors vest copyright in their articles, including abstracts, in Shadowfax Publishing and Taylor & Francis Limited. This enables us to ensure full copyright protection and to disseminate the article, and the journal, to the widest possible readership in print and electronic formats as appropriate. Authors may, of course, use the article elsewhere *after* publication providing that prior permission is obtained from Taylor & Francis Ltd.

Authors are themselves responsible for obtaining permission to reproduce copyright material from other sources.

Typeset by Infotype Ltd, Oxfordshire
Printed and Bound in Great Britain by Polestar AUP Aberdeen Ltd, Aberdeen

APPENDICES FOR LITERATURE REVIEW (CHAPTER 2)

Appendix 2.1

Instructions to Authors

Style of manuscripts. Authors should prepare manuscripts according to the *Publication Manual of the American Psychological Association* (4th ed.). Typing instructions (all copy must be double-spaced) and instructions on preparing tables, figures, references, metrics, and abstracts appear in the *Manual.* Also, all manuscripts are subject to masked review and editing for sexist language.

Publication policies. APA policy prohibits an author from submitting the same manuscript for concurrent consideration by two or more publications. In addition, it is a violation of APA Ethical Principles to publish "as original data, data that have been previously published" (Standard 6.24). As this journal is a primary journal that publishes original material only, APA policy prohibits as well publication of any manuscript that has already been published in whole or substantial part elsewhere. Authors have an obligation to consult journal editors concerning prior publication of any data upon which their article depends. In addition, APA Ethical Principles specify that "after research results are published, psychologists do not withhold the data on which their conclusions are based from other competent professionals who seek to verify the substantive claims through reanalysis and who intend to use such data only for that purpose, provided that the confidentiality of the participants can be protected and unless legal rights concerning proprietary data preclude their release" (Standard 6.25). APA expects authors submitting to this journal to adhere to these standards. Specifically, authors of manuscripts submitted to APA journals are expected to have available their data throughout the editorial review process and for at least 5 years after the date of publication.

Authors will be required to state in writing that they have complied with APA ethical standards in the treatment of their sample, human or animal, or to describe the details of treatment. A copy of the APA Ethical Principles may be obtained by writing the APA Ethics Office, 750 First Street, NE, Washington, DC 20002-4242.

Abstracts. Manuscripts of regular articles must be accompanied by an abstract containing a maximum of 960 characters and spaces (which is approximately 120 words). Manuscripts of Brief Reports must be accompanied by an abstract of 75–100 words. All abstracts must be typed on a separate sheet of paper.

Brief Reports. The *Journal of Consulting and Clinical Psychology* will accept Brief Reports of research studies in clinical psychology. The procedure is intended to permit the publication of soundly designed studies of specialized interest or limited importance that cannot now be accepted as regular articles because of lack of space. Several pages in each issue may be devoted to Brief Reports.

An author who submits a Brief Report must agree not to submit the full report to another journal of general circulation. The Brief Report should give a clear, condensed summary of the procedure of the study and as full an account of the results as space permits. Brief Reports should be limited to four printed pages and prepared according to the following specifications:

For Brief Reports, length limits are exact and must be strictly followed. In preparing your manuscript, set the character/space limit at 60 characters per line and do not exceed 410 lines for text and references. These limits do not include the title page, abstract, author note, footnotes, tables, or figures. For Brief Reports, as for regular manuscripts, do not exceed 960 characters/spaces in the abstract.

This journal no longer requires an extended report. However if one is available, the Brief Report must be accompanied by the following footnote:

Correspondence concerning this article (and requests for an extended report of this study) should be addressed to (give the author's full name and address).

Submitting manuscripts. Manuscripts should be submitted in quadruplicate, and all copies should be clear, readable, and on paper of good quality. A dot matrix or unusual typeface is acceptable only if it is clear and legible. Dittoed and mimeographed copies are not acceptable and will not be considered. In addition to addresses and phone numbers, authors should supply electronic mail addresses and fax numbers, if available, for potential use by the editorial office and later by the production office. Authors should keep a copy of the manuscript to guard against loss. Effective in January 1996, the Incoming Editor is receiving all submissions to the journal. Submissions that are accepted will be published in the 1997 volume. Mail manuscripts to the Incoming Editor, Philip C. Kendall, *Journal of Consulting and Clinical Psychology*, Department of Psychology, Weiss Hall, Temple University, Philadelphia, PA 19122.

APPENDICES FOR MAJOR RESEARCH PROJECT PROPOSAL (CHAPTER 3)

Appendix 3.1

Ref: AmcM/0002

GREATER GLASGOW
PRIMARY CARE
NHS TRUST

23 June, 2000

Ms L Harvey
Academic Centre
Gartnavel Royal Hospital
1055 Gt Western Road
Glasgow
G12 0XH

Dear Ms Harvey

PROJECT: Models of sleep disturbance in insomnia and major depressive disorder

Many thanks for sending the above named submission to the Research Ethics Committee - it was discussed at our meeting on Thursday, 8 June, 2000. I am pleased to be able to tell you that the Committee has no objections from an ethical point of view, to this project proceeding and ethical approval is formally granted. Before your project commences you will also require to obtain management approval via the Research & Development Directorate, Gartnavel Royal Hospital.

The Committee require however that some minor alterations are made to the proposal -

Part B of the consent form should be removed and the last paragraph greatly reduced with the section on "research number" being removed.

The Committee would also like to bring to your own and the Department's attention the concern that the same patients were perhaps being asked to participate in too many trials

I would also like to take this opportunity to remind you that you should notify the Committee if there are any changes, or untoward developments, connected with the study – the Committee would then require to further reconsider your application for approval. The Committee expect to receive a brief regular update every 6 months, and then a brief final report on your project when the study reaches its conclusion. (Failure to keep the Committee abreast of the status of the project can eventually lead to ethical approval being withdrawn)

May I wish you every success with your study.

Yours sincerely

A W McMAHON
Administrator – Research Ethics Committee

cc B Rae

GREATER GLASGOW COMMUNITY/PRIMARY CARE LOCAL RESEARCH ETHICS COMMITTEE

27 July 2000

Linda Harvey
Trainee Clinical Psychologist
Department of Psychological Medicine
Academic Centre
Gartnavel Royal Hospital
1055 Great Western Road
Glasgow
G12 OXH

Study – Models of sleep disturbance in insomnia and major depressive disorder.

Thank you for your letter dated 17th July 2000 and would advise that the screeners on our committee have now looked at your submission and agree that it does not require ethics approval.

Yours sincerely

Hazel Illingworth
Administrator

APPENDICES FOR MAJOR RESEARCH PROJECT PAPER
(CHAPTER 4)

Appendix 4.1

Instructions to Authors

Style of manuscripts. Authors should prepare manuscripts according to the *Publication Manual of the American Psychological Association* (4th ed.). Typing instructions (all copy must be double-spaced) and instructions on preparing tables, figures, references, metrics, and abstracts appear in the *Manual.* Also, all manuscripts are subject to masked review and editing for sexist language.

Publication policies. APA policy prohibits an author from submitting the same manuscript for concurrent consideration by two or more publications. In addition, it is a violation of APA Ethical Principles to publish "as original data, data that have been previously published" (Standard 6.24). As this journal is a primary journal that publishes original material only, APA policy prohibits as well publication of any manuscript that has already been published in whole or substantial part elsewhere. Authors have an obligation to consult journal editors concerning prior publication of any data upon which their article depends. In addition, APA Ethical Principles specify that "after research results are published, psychologists do not withhold the data on which their conclusions are based from other competent professionals who seek to verify the substantive claims through reanalysis and who intend to use such data only for that purpose, provided that the confidentiality of the participants can be protected and unless legal rights concerning proprietary data preclude their release" (Standard 6.25). APA expects authors submitting to this journal to adhere to these standards. Specifically, authors of manuscripts submitted to APA journals are expected to have available their data throughout the editorial review process and for at least 5 years after the date of publication.

Authors will be required to state in writing that they have complied with APA ethical standards in the treatment of their sample, human or animal, or to describe the details of treatment. A copy of the APA Ethical Principles may be obtained by writing the APA Ethics Office, 750 First Street, NE, Washington, DC 20002-4242.

Abstracts. Manuscripts of regular articles must be accompanied by an abstract containing a maximum of 960 characters and spaces (which is approximately 120 words). Manuscripts of Brief Reports must be accompanied by an abstract of 75–100 words. All abstracts must be typed on a separate sheet of paper.

Brief Reports. The *Journal of Consulting and Clinical Psychology* will accept Brief Reports of research studies in clinical psychology. The procedure is intended to permit the publication of soundly designed studies of specialized interest or limited importance that cannot now be accepted as regular articles because of lack of space. Several pages in each issue may be devoted to Brief Reports.

An author who submits a Brief Report must agree not to submit the full report to another journal of general circulation. The Brief Report should give a clear, condensed summary of the procedure of the study and as full an account of the results as space permits. Brief Reports should be limited to four printed pages and prepared according to the following specifications:

For Brief Reports, length limits are exact and must be strictly followed. In preparing your manuscript, set the character/space limit at 60 characters per line and do not exceed 410 lines for text and references. These limits do not include the title page, abstract, author note, footnotes, tables, or figures. For Brief Reports, as for regular manuscripts, do not exceed 960 characters/spaces in the abstract.

This journal no longer requires an extended report However if one is available, the Brief Report must be accompanied by the following footnote:

Correspondence concerning this article (and requests for an extended report of this study) should be addressed to (give the author's full name and address).

Submitting manuscripts. Manuscripts should be submitted in quadruplicate, and all copies should be clear, readable, and on paper of good quality. A dot matrix or unusual typeface is acceptable only if it is clear and legible. Dittoed and mimeographed copies are not acceptable and will not be considered. In addition to addresses and phone numbers, authors should supply electronic mail addresses and fax numbers, if available, for potential use by the editorial office and later by the production office. Authors should keep a copy of the manuscript to guard against loss. Effective in January 1996, the Incoming Editor is receiving all submissions to the journal. Submissions that are accepted will be published in the 199- volume. Mail manuscripts to the Incoming Editor, Philip C. Kendall, *Journal of Consulting and Clinical Psychology*, Department of Psychology, Weiss Hall, Temple University, Philadelphia, PA 19122.

Appendix 4.2
Sleep History Questionnaire

Name:

Date of Birth:

Male/Female :

1. **How long have you had your sleep problem? (Try and be as specific as possible)**

2. **Has your sleep problem been constant over the years or does it come in spells?**

3. **Please describe your sleep problems**

4. **Do you ever sleepwalk, sleeptalk or engage in any other unusual behaviours while asleep? If yes, please give details.**

5. **Do you ever experience unpleasant or strange sensations in your legs while you are in bed? If yes please give details.**

6. **Do you often experience limb movements while trying to sleep? If yes, please give details.**

6. **Do you snore?**

7. **Do you ever wake up with difficulty breathing? If yes, please give details.**

8. **Do you feel excessively tired during the day?**

4. **Do you know the cause of your sleep problems? If so, please explain e.g. were you undergoing a major life event such as death of a loved one, illness, change of jobs at the time of the initial episode of insomnia**

5. **Are you currently taking any medication to help you sleep?**

6. **If you are taking medication, did the doctor prescribe it or did you buy it over-the-counter from a chemist shop?**

7. **What is the name of the medication you take? How often do you take it? Do you know the dosage?**

8. **Have you seen any professional other than your doctor regarding your sleep difficulties? If yes, what kind of professional did you see?**

9. **In the past have you ever experienced any psychological or psychiatric problems?**

10. **If yes, it would be very helpful if you could possibly give a brief outline of the difficulties you experienced?**

11. **Did you see any professionals regarding these difficulties. If yes, please give brief details.**

12. **Are you currently experiencing any psychological or psychiatric problems?**

13. **If yes, again it would be helpful if you could give a brief outline of your current difficulties?**

14. **Are you currently receiving any help from any professionals with these difficulties. If yes, please give brief details.**

15. **Please could you list <u>all</u> the medications you are currently prescribed?**

Appendix 4.3
Sleep Diary

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
1. At what time did you rise from bed this morning?							
2. At what time did you go to bed last night?							
3. How long did it take you to fall asleep (minutes)?							
4. How many times did you wake up during the night?							
5. How long were you awake during the night (in total)?							
6. About how long did you sleep altogether (hours/mins)?							
7. How much alcohol did you take last night?							
8. Did you take any sleeping pills to help you sleep?							

Appendix 4.4
Sleep Behaviour Self Rating Scale (SBSRS)

Please read each item and indicate to what extent you engage in that behaviour in your bed or bedroom either during the day or around sleeping time.

Behaviour	Never	Rarely	Sometimes	Often	Very Often
Reading during the day					
Reading around sleeping time					
Eating during the day					
Eating around sleeping time					
Watching television around sleeping time					
Pleasant conversation during the day					
Pleasant conversation around sleeping time					
Unpleasant conversation during the day					
Unpleasant conversation around sleeping time					
Positive thoughts during the day					
Negative thoughts during the day					
Negative thoughts around sleeping time					
Sex during the day					
Sex around sleeping time					
Listening to the radio during the day					
Listening to the radio around sleeping time					
Talking on the phone during the day					
Talking on the phone around sleeping time					
Smoking during the day					
Smoking around sleeping time					

Appendix 4.5
Sleep Behaviour Self Rating Scale -Adapted (SBSRS-A)

Please indicate how often you do the following things in your bed before falling asleep or while in your bedroom. Complete the form by considering what you would do in an average week.

Behaviour	Never	Rarely	Sometimes	Often	Very Often
Read a book or magazine					
Watch TV					
Listen to the radio					
Have a conversation with someone					
Speak on the telephone					
Eat or drink					
Smoke					
Please also answer the following questions: I take naps during the day or evening					
I feel sleepy when I go to bed					
I switch the light off as soon as I get into bed					
I spend a lot of time lying awake in bed at night					
If I can't get to sleep within approx. 20 minutes I get out of bed and move to another room until I feel sleepy again					
I set myself a regular rising time each morning					
If I have a bad night's sleep I still get up at my usual time					

Appendix 4.6
Sleep Hygiene Awareness and Practice Scale (SHAPS)

For each of the following behaviours state the number of days per week (0-7) that you engage in that activity or have that experience. Base your answers on what you would consider an average week for yourself.

Indicate the number of days or nights in an average week you:

1. Take a nap:

2. Go to bed hungry:

3. Go to bed thirsty:

4. Smoke more than one packet of cigarettes a day:

5. Use sleeping medications (prescribed or over-the -counter):

6. Drink beverages containing caffeine (e.g. coffee, tea, cola) within 4 hours of bedtime:

7. Drink more than 3 ounces of alcohol (e.g. 3 mixed drinks, 2 beers or 3 glasses of wine) within two hours of bedtime:

8. Take medications/drug with caffeine within 4 hours of bedtime:

9. Worry as you prepare for bed about your inability to sleep:

10. Worry during the day about your inability to sleep at night:

11. Use alcohol to facilitate sleep:

12. Exercise strenuously within 2 hours of bedtime:

13. Have your sleep disturbed by light:

14. Have your sleep disturbed by noise:

15. Have your sleep disturbed by your bedpartner (put N/A if no partner):

16. Sleep approximately the same length of time each night:

17. Set aside time to relax before bedtime:

18. Exercise in the afternoon or early evening:

19. Have a comfortable night-time temperature in your bed/bedroom:

Appendix 4.7
Pre-Sleep Arousal Scale (PSAS)

For each of the following 16 symptoms please rate how intensely you generally experience each symptom as you attempt to fall asleep in your bedroom.

1 = not at all
2 = slightly
3 = moderately
4 = a lot
5 = extremely

1.	Heart racing, pounding or beating irregularly	Rating:
2.	A jittery, nervous feeling in your body	Rating:
3.	Shortness of breath or laboured breathing	Rating:
4.	A tight, tense feeling in your muscles	Rating:
5.	Cold feeling in your hands, feet or your body in general	Rating:
6.	Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas etc.	Rating:
7.	Perspiration in palms of your hands or other parts of your body	Rating:
8.	Dry feeling in mouth or throat	Rating:
9.	Worry about falling asleep	Rating:
10.	Review or ponder events of the day	Rating:
11.	Depressing or anxious thoughts	Rating:
12.	Worry about problems other than sleep	Rating:
13.	Being mentally alert, active	Rating:
14.	Can't shut off your thoughts	Rating:
15.	Thoughts keep running through your head	Rating:
16.	Being distracted by sounds in the environment (e.g. ticking of clock, house noises, traffic)	Rating:

Appendix 4.8
Sleep Disturbance Questionnaire (SDQ)

Please put a X in the appropriate box, depending upon how true you feel each of the following statements is for your typical sleep pattern.

On the nights when I don't sleep well the problem seems to be that:

	Never True	Seldom True	Sometimes True	Often True	Very Often True
1. I can't get into a comfortable position in bed					
2. My mind keeps turning things over					
3. I can't get my sleep into a proper routine					
4. I get too "worked up" at not sleeping					
5. I find it hard to physically "let go" and relax my body					
6. My thinking takes a long time to unwind					
7. I don't feel tired enough at bedtime					
8. I try too hard to get to sleep					
9. My body is full of tension					
10. I am unable to empty my mind					
11. I spend time reading/watching TV in bed when I should be sleeping					
12. I worry that I won't cope tomorrow if I don't sleep well.					

Appendix 4.9
Sleep Anxiety Scale (SAS)

The following questions relate to your sleep. For each of the four sleep problems listed below please tick a box to show how much of a problem they are to you.

1. For you is difficulty falling asleep a

☐	☐	☐
major problem	minor problem	not a problem

2. For you is difficulty getting back to sleep after wakening up at night a

☐	☐	☐
major problem	minor problem	not a problem

3. For you is waking up too early in the morning a

☐	☐	☐
major problem	minor problem	not a problem

4. For you is not feeling rested the next day a

☐	☐	☐
major problem	minor problem	not a problem

The following four questions relate to how much your sleeplessness worries you. For each of the four questions below please tick a box to show how much your sleeplessness worries you.

1. Does your sleeplessness worry you when you go to bed

☐	☐	☐
very much	some	not at all

2. Does your sleeplessness worry you when you wake up at night

☐	☐	☐
very much	some	not at all

3. Does your sleeplessness worry you when you wake up early in the morning

☐	☐	☐
very much	some	not at all

4. Does your sleeplessness worry you when you think about it during the day

☐	☐	☐
very much	some	not at all

Appendix 4.10
Sleep Performance Anxiety Scale (SPAQ)

The following seven statements relate to your night-time sleep pattern. Please indicate by circling one response how true each statement, *in general*, is for you.

1. I put too much effort into sleeping at night when it should come naturally

Very much To some extent Not at all

2. I feel I should be able to control my sleep at night

Very much To some extent Not at all

3. I put off going to bed at night for fear of not being able to sleep

Very much To some extent Not at all

4. I worry about not sleeping if I am in my bed at night and can't sleep

Very much To some extent Not at all

5. I am no good at sleeping

Very much To some extent Not at all

6. I get anxious about sleeping before I go to bed at night

Very much To some extent Not at all

7. I worry about the long term consequences of not sleeping at night

Very much To some extent Not at all

Appendix 4.11
The Dysfunctional Beliefs and Attitudes about Sleep Scale-10 (DBAS-10)

Statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement place a mark (X) along the line wherever your PERSONAL rating falls. Try to use the whole scale rather than placing your marks at one end of the line. Please answer all questions

Example:- *If I sleep too much I don't perform as well the next day*

Strongly disagree _____ Strongly agree

1. *I need 8 hours of sleep to feel refreshed and function well during the day*

Strongly disagree _____ Strongly agree

2. *When I don't get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer*

Strongly disagree _____ Strongly agree

3. *After a poor night's sleep, I know that it will interfere with my daily activities on the next day*

Strongly disagree _____ Strongly agree

4. *When I feel irritable, depressed or anxious during the day, it is mostly because I did not sleep well the night before*

Strongly disagree _____ Strongly agree

5. *When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before*

Strongly disagree _____ Strongly agree

6. *I am concerned that chronic insomnia may have serious consequences on my physical health*

Strongly disagree _____ Strongly agree

7. *I am worried that I may lose control over my abilities to sleep*

Strongly disagree _____ Strongly agree

8. *When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week*

Strongly disagree _____ Strongly agree

9. *When I have trouble getting to sleep, I should stay in bed and try harder*

Strongly disagree _____ Strongly agree

10. *I get overwhelmed by my thoughts at night and often feel I have no control over the racing mind*

Strongly disagree _____ Strongly agree

Appendix 4.12
Participant Information Leaflet

GREATER GLASGOW PRIMARY CARE NHS TRUST

RESEARCH PROJECT: MODELS OF SLEEP DISTURBANCE IN INSOMNIA AND MAJOR DEPRESSIVE DISORDER

PARTICIPANT INFORMATION SHEET

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Purpose of Study

This research study is hoping to investigate and compare the sleep patterns of people who have problems sleeping and who are depressed, people who have problems sleeping who are not depressed and people who have no problems sleeping. The results from the study will hopefully help us understand sleep problems better and will help us to provide better treatments for people with sleep problems.

Why have I been chosen?

You have been chosen because it is thought that you may fall into one of the categories of people that we are hoping to study. In total the study needs 54 people to take part.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. This will not affect the standard of care you receive. However, if you do decide to participate then I would like to thank-you in advance for your time and cooperation.

What you will be asked to do

If you agree to take part in this research you will be required to meet with the researcher on one occasion, lasting approximately one to one and a half hours. The researcher will ask you questions relating to any psychological problems you are experiencing and will ask you to fill in a "sleep diary" every day for one week. A sleep diary is a form which asks things such as when you went to bed, when you woke up, how long it took you to get to sleep etc. It takes a few minutes each morning to fill in. The researcher will also ask you to wear a special wrist watch for five nights which tells us when you were actually awake and when you were asleep. You will then be given some questionnaires about your sleep pattern to take home

with you and fill in. The researcher will ask you to return the special watch and the questionnaires in a freepost envelope. You are entitled to travelling expenses for your visit to and from the appointment with the researcher.

Are there any benefits of taking part?

When the researcher has received your wrist watch and questionnaires, an information leaflet called the "Good Sleep Guide" will be posted to you. It is hoped that this leaflet will help you with your sleeping problems. In addition, the results from this study should hopefully help people with sleep problems in the future.

Will my taking part in this study be kept confidential?

All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you which leaves the clinic will have your name and address removed so that you cannot recognise it. If you wish, information collected about you can be passed on to your GP, any other medical practitioner or Psychologist if you feel it would be helpful for your treatment.

What will happen to the results of the study?

This study will hopefully be published at the start of the year 2002. If you would like a copy of the published results please write to the researcher at the contact address given below.

Who is organising and funding the research?

This research forms part of the researcher's Doctorate degree in Clinical Psychology based at the Department of Psychological Medicine, Gartnavel Academic Centre. The researcher is funded on this training by the NHS in Scotland.

Who has reviewed this study?

The Greater Glasgow Primary Care NHS Trust Research Ethics Committee has reviewed this study and granted ethical approval. The Greater Glasgow Community and Primary Care Local Research Ethics Committee has seen a proposal for this study. They concluded that the study does not need to be presented before their committee.

Contact for Further Information

For further information please contact the researcher, Linda Harvey, at:
Department of Psychological Medicine
Gartnavel Academic Centre
1055 Great Western Road
Glasgow
G12 0XH
Tel: 0141 211 3577

Appendix 4.13
Consent Form

GREATER GLASGOW PRIMARY CARE NHS TRUST

RESEARCH STUDY: MODELS OF SLEEP DISTURBANCE IN INSOMNIA AND MAJOR DEPRESSIVE DISORDER

CONSENT FORM

I confirm that I have read and understood the information sheet and have been provided with my own copy of the information sheet.

I confirm that I have been given the opportunity to discuss the research project with the researcher and have had any questions answered.

I understand that I am free to withdraw from this study at any time without having to give a reason and I understand that my health-care or treatment would not be affected in any way if I was to withdraw from the study.

I understand that the information I give to the researcher is confidential.

I consent to participate in this study.

Signed.....Date.....

Name (printed).....

Witness signature.....

Name (printed).....

OPTIONAL

I consent to any useful information that may arise from taking part in this study to be passed on to my GP/medical practitioners and to my Psychologist.

Signed.....

Appendix 4.14

THE GOOD SLEEP GUIDE

DURING THE EVENING

1. Put the day to rest. Think it through. Tie up "loose ends" in your mind and plan ahead. A notebook may help.
2. Take some light exercise early in the evening. Generally try to keep yourself fit.
3. Wind down during the course of the evening. Do not do anything that is mentally demanding within 90 minutes of bedtime.
4. Do not sleep or doze in the armchair. Keep your sleep for bedtime.
5. Do not drink too much coffee or tea and only have a light snack for supper. Do not drink alcohol to aid your sleep - it usually upsets sleep.
6. Make sure your bed and bedroom are comfortable - not too cold and not too warm.

AT BEDTIME

1. Go to bed when you are "sleepy tired" and not before.
2. Do not read or watch TV in bed. Keep these activities for another room.
3. Set the alarm for the same time every day - 7 days a week, or at least until your sleep pattern settles down.
4. Put the light out when you get into bed.
5. Let yourself relax and tell yourself that "sleep will come when it's ready". Enjoy relaxing even if you don't at first fall asleep.
6. Do not try to fall asleep. Sleep is not something you can switch on deliberately but if you try to switch it on you can switch it off!

Appendix 4.15
Skewness, Kurtosis and Homogeneity of Variance information for data distributions

	Skewness			Kurtosis			Homogeneity of Variance ($p<0.05$ represents significant heterogeneity)
	C group	PI group	DI group	C group	PI group	DI group	
SOL (diary)	2.07*	0.67	0.95	4.46*	-0.26	0.28	0.00
WASO (diary)	1.74	2.16*	2.59*	3.97*	5.64*	7.53*	0.01
TST (diary)	-1.11	-0.17	-1.32	1.31	-0.94	2.04*	0.03
SE (diary)	-2.04*	-1.40	-1.47	5.13*	1.87	1.70	0.00
SOL (actigraph)	2.60*	1.06	1.51	8.09*	0.91	1.50	0.00
WASO (actigraph)	0.42	0.86	1.02	-0.77	0.58	1.91	0.09
TST (actigraph)	-0.64	1.55	0.21	-0.50	4.93*	-1.15	0.16
SE (actigraph)	0.20	-0.84	-0.94	-1.12	2.37*	0.58	0.00
SBSRS-A	-0.55	0.48	-0.10	-0.24	-0.27	-0.51	0.50
SHAPS	1.11	0.30	0.64	2.40*	0.45	0.16	0.26
PSAS (physio.)	1.30	1.43	0.36	1.08	1.38	-1.40	0.00
PSAS (cog.)	0.48	-0.56	0.07	-0.50	-0.64	-1.1	0.00
SDQ	-0.21	-1.55	-1.00	-0.89	1.89	-0.21	0.06
SAS	1.57	-0.1	-0.61	1.90	-0.24	0.61	0.02
SPAQ	0.84	0.96	0.04	-0.10	0.96	-0.91	0.00
DBAS-10	-0.88	0.51	0.11	0.43	-0.96	-0.21	0.14

* represents significant skewness or kurtosis