RAPID PUBLICATION

Randomized Clinical Effectiveness Trial of Nurse-Administered Small-Group Cognitive Behavior Therapy for Persistent Insomnia in General Practice

Colin A. Espie, PhD[1]; Kenneth M. A. MacMahon, PhD[1]; Heidi-Louise Kelly, DClinPsy[1]; Niall M. Broomfield, PhD[1]; Neil J. Douglas, MD[2]; Heather M. Engleman, PhD[2]; Brian McKinstry, MD[3]; Charles M. Morin, PhD[4]; Andrew Walker, PhD[5]; Philip Wilson, FRCGP[6]

[1]University of Glasgow Sleep Research Laboratory, Scotland, UK; [2]Department of Sleep & Respiratory Medicine, University of Edinburgh, Scotland, UK; [3]Community Health Sciences: General Practice Section, University of Edinburgh, Scotland, UK; [4]Ecole de Psychologie, Universite Laval, Quebec City, Canada; [5]Robertson Centre for Biostatistics, University of Glasgow, Scotland, UK; [6]Section of General Practice and Primary Care, University of Glasgow, Scotland, UK

Study Objectives: Persistent insomnia, although very common in general practice, often proves problematic to manage. This study investigates the clinical effectiveness and the feasibility of applying cognitive behavior therapy (CBT) methods for insomnia in primary care.

Design: Pragmatic randomized controlled trial of CBT versus treatment as usual.

Setting: General medical practice.

Participants: Two hundred one adults (mean age, 54 years) randomly assigned to receive CBT (n = 107; 72 women) or treatment as usual (n = 94; 65 women).

Intervention: CBT comprised 5 sessions delivered in small groups by primary care nurses. Treatment as usual comprised usual care from general practitioners.

Measurements and Results: Assessments were completed at baseline, after treatment, and at 6-month follow-up visits. Sleep outcomes were appraised by sleep diary, actigraphy, and clinical endpoint. CBT was associated with improvements in self-reported sleep latency, wakefulness after sleep onset, and sleep efficiency. Improvements were partly sustained at follow-up. Effect sizes were moderate for the index variable of sleep efficiency. Participants receiving treatment as usual did not improve. Actigraphically estimated sleep improved modestly after CBT, relative to no change in treatment as usual. CBT was also associated with significant positive changes in mental health and energy/vitality. Comorbid physical and mental health difficulties did not impair sleep improvement following CBT.

Conclusion: This study suggests that trained and supervised nurses can effectively deliver CBT for insomnia in routine general medical practice. Treatment response to small-group service delivery was encouraging, although effect sizes were smaller than those obtained in efficacy studies. Further research is required to consider the possibility that CBT could become the treatment of first choice for persistent insomnia in primary healthcare.

Keywords: Insomnia, sleep, treatment, primary care, psychological intervention

Citation: Espie C; MacMahon KMA; Kelly HL et al. Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *SLEEP* 2007;30(5):574-584

SLEEP DISTURBANCE IS THE MOST COMMON SYMPTOM OF MENTAL ILLNESS, BEING MORE COMMON THAN WORRY AND TWICE AS COMMON AS ANXIETY or depressive symptoms. Moreover, in a recent UK psychiatric morbidity study,[1] this finding held for men and women of any age or ethnic group in any region. Epidemiologic studies report the prevalence of insomnia disorder at 10% to 12%, with older adult rates at greater than 20%.[2,3] One fifth of patients consulting in primary care have insomnia.[4] Typically, difficulty initiating or maintaining sleep, or both initiating and maintaining sleep, is associated with reduced daytime alertness and productivity, poorer quality of life, impaired relationships, and increased ill health.[5-9] Two meta-analyses have reported preexisting sleep disturbance as the largest, potentially treatable, risk factor for first-episode depression and for recurrence of depression.[10,11]

Despite such findings, persistent insomnia often goes unrecognized, and care management is poorly developed.[12] Benzodiazepine hypnotics and sedative antidepressants are commonly prescribed in clinical practice, although long-term outcome data are relatively sparse,[13,14] and, although the benzodiazepine receptor agonists confer some advantages in the management of acute insomnia, there is thus far limited evidence that they are preferable for the treatment of persistent insomnia.[15] In short, the management of chronic insomnia represents a very significant gap in the clinical armamentarium.

Cognitive behavior therapy (CBT) offers 1 promising approach. Insomnia often arises from psychological factors such as conditioned arousal, maladaptive sleep habits and sleep schedules, dysfunctional thinking about sleep and its consequences, and sleep preoccupation.[16] This behavioral phenotype may be similar whether insomnia is primary or presenting in the context of psychiatric problems.[2,17] Although 3 meta-analyses have demonstrated clear benefit,[18-20] CBT efficacy trials have recruited largely among media-solicited participants, perhaps excluding patients with complex presentations. Such studies have conformed more to the traditions of clinical efficacy research, where there is an

Disclosure Statement

This is not an industry supported study. Dr. Douglas has received industry support from ResMed and is on the International Medical Advisory Board of ResMed. Dr. Morin has received research support from Sanofi-Aventis; has been a consultant to Sepracor, Pfizer, Neurocrine, Takeda, and Shire Biochem; and has participated in speaking engagements for Takeda, Sanofi-Aventis, and Merck. Dr. Walker has participated in speaking engagements for Schering-Plough, Novartis, and Boehringer-Ingelheim. Drs. Espie, MacMahon, Kelly, Broomfield, Engleman, McKinstry, and Wilson have indicated no financial conflicts of interest.

Submitted for publication August, 2006
Accepted for publication December, 2006

Address correspondence to: Professor Colin A. Espie, University of Glasgow Sleep Research Laboratory, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, Scotland, UK; Tel: +44 (0)141-232-7700; Fax: +44 (0)141-232-7697; Email: c.espie@clinmed.gla.ac.uk

Table 1—Inclusion and Exclusion Criteria Based Primarily Upon ICSD-R/ DSM-IV

Inclusion Criteria
- Aged ≥ 18 years
- Referred by general practitioner
- Living in the community in Glasgow or Edinburgh area
- Difficulty initiating and/or maintaining sleep, comprising SOL ≥ 30 minutes and/or WASO ≥ 30 minutes, 3 or more nights per week
- Present sleep complaint for at least 6 months
- Negative complaint of insomnia impact (eg, fatigue, impaired mood)

Exclusion Criteria
- Deteriorating health or dementia
- Incapacitating pain or illness
- Untreated mental health problems
- Untreated other sleep problems

ICSD-R refers to the *International Classification of Sleep Disorders-Revised*[5]; DSM-IV, *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*[6]; SOL sleep-onset latency; WASO, wake time after sleep-onset.

emphasis on sample homogeneity, the exclusion of comorbidities, measurement reliability, and the management of other factors that are known to influence extraneous variance. Consequently, we do not know whether it is clinically effective or feasible to translate CBT to primary care. Effectiveness studies, by way of contrast, emphasize validity and generalizability to "real-world" settings by accessing populations and following procedures that reflect more typical clinical practice. Results from a preliminary clinical effectiveness study have suggested that improvements with CBT delivered by primary care nurses may be obtained.[21] The present report is a formal intention-to-treat evaluation of this model.

METHODS

Aims and Objectives

The aim of the study was to test the effectiveness of CBT for the treatment of persistent insomnia in the "real-world" primary care setting. The major research questions were "Is CBT superior to treatment as usual (TAU) in reducing chronic sleep disturbance?", "Are observed changes in sleep pattern and sleep quality durable?", and "Are there predictors of good outcome, or contraindications to the application of CBT, for insomnia in general practice?"

Design

The study conformed to a pragmatic, randomized trial design following CONSORT guidelines. CBT was compared with TAU, this being an appropriate control for a clinical effectiveness study. Major assessments were at baseline, after treatment, and at follow-up 6 months later.

Participants

Potential participants were patients attending an appointment with their general practitioner (GP), or who were on their GP's prescribing list for a sleep medication, during the period June 2001 to July 2003. One hundred and four GPs in 19 practices in Glasgow, West Lothian, and Edinburgh identified participants. Eligibility criteria are described in Table 1. Participants had to satisfy criteria based primarily upon International Classification of Sleep Disorders -revised/Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition[5,6] and standard quantitative criteria. Consistent with this type of trial, exclusions were limited to new, untreated, or serious disorders or substance abuse problems that would make participation impractical or clinically inadvisable. Patients with physical or psychological problems were not excluded. Similarly, being on sleep (or other) medications was not an exclusion criterion.

Potential participants were notified of the study by their GP and through posters in clinic waiting areas and explanatory leaflets. Some GPs conducted record searches before circulating information to patients with known insomnia problems. All prospective participants were then "referred" by their GP using a simple form on which GPs marked (√) against each study criterion (Table 1). Participants had the opportunity to discuss the research with a member of the research team and to reconsult with their GP prior to consenting. All gave written informed consent. The protocol was approved by local research ethics committees. Based on previous work,[21] a sample size of 240 would have 90% power to show a 0.5 SD difference between treatments at an α level of 0.05. This was based on CBT achieving a reduction, in minutes of total wake time, at least 50% more than TAU.

After baseline assessment and anonymization, participants were randomly assigned by an administrator in an independent research group using a computer-generated random list of numbers. Allocation was strictly in order of completion of baseline data, independent of participating GP practice or location. Randomization information was kept in a locked cabinet and was inaccessible to researchers. Because of the nature of the intervention, it was not possible to blind participants or therapists to CBT and TAU allocations.

Measures

Potential participants were screened by telephone interview. Data were collected on sleep history, including diagnostic criteria, as well as medical and psychiatric history. A comprehensive face-to-face interview then obtained a more detailed history. Interview was supplemented by completion of the Pittsburgh Sleep Quality Index (PSQI),[22] the Hospital Anxiety and Depression Scale (HADS),[23] and the Epworth Sleepiness Scale (ESS).[24]

Subjective sleep pattern was assessed using a sleep diary,[25] completed for 2 weeks at each of 3 assessment points; baseline, posttreatment, and follow-up. Such diaries are the staple tool of insomnia-assessment practice[16] and offer a valid relative index of sleep disturbance, particularly when used as repeated measures.[26,27] Fourteen nights is an adequate sampling period.[28] Items "how long did it take you to fall asleep last night" (sleep-onset latency: SOL) and "how long were you awake in total last night, after you first fell asleep?" (wake time after sleep onset: WASO) assessed the central insomnia dimensions of difficulty initiating and maintaining sleep. Participants were advised to estimate WASO between initial sleep onset and rising from bed. The diary also inquired about bedtime and rising time, from which total time in bed (TIB), and then sleep efficiency percentage (SE) was

Table 2—Summary Content of the Cognitive Behavior Therapy Program

Session 1 Sleep Information
Aim: To learn about normal sleep processes and about sleep disorders
- to understand the need for sleep and its functions
- to understand sleep pattern and how it varies during the lifetime
- to understand sleep as a process with stages and phases
- to understand factors that adversely affect sleep pattern and sleep quality
- to understand the effects of sleep loss
- to understand the concept of insomnia and how it can be measured
- to understand personal sleep histories and patterns in the above context
- to begin to correct misunderstandings about sleep and sleeplessness

Session 2 Sleep Hygiene & Relaxation
Aim: To introduce practical steps toward developing a healthy sleep pattern without recourse to drugs
- to create a bedroom environment that is comfortable for sleep
- to take regular exercise that promotes fitness and enhances sleep
- to develop a stable and appropriate diet
- to reduce the undesirable effects of caffeine upon sleep
- to moderate alcohol consumption and eliminate "night caps"
- to learn relaxation skills to apply at home and in bed

Session 3 Sleep Scheduling
Aim: To reshape sleep patterns to correspond with individual sleep needs and to strengthen sleep rhythms
- to develop a good presleep routine
- to distance waking activities (eg, watching TV) from the bedroom environment
- to establish a strong bed-sleep connection
- to eliminate wakefulness from bed (rising if not asleep within around 15 minutes)
- to define restricted parameters for the individual's sleep period
- to increase sleep efficiency through scheduling sleep in relation to current total sleep
- to eliminate daytime napping
- to establish a stable night-to-night sleep pattern, rising at the same time every day
- to encourage and support people in changing their sleep routines

Session 4 Cognitive approaches
Aim: To learn ways of reducing mental alertness, repetitive thoughts, and anxiety that interfere with sleep
- to identify thought patterns that interfere with sleep
- to develop accurate beliefs and attitudes about sleep
- to prepare mentally for bed by putting the day to rest
- to learn thought distraction and imagery techniques
- to reduce efforts to control sleep and allow it to happen naturally
- to utilize these techniques to combat intrusive thoughts
- to encourage and support people in changing their mental approach
- to further adjust sleep schedules to maintain sleep efficiency

Session 5 Developing a strong & natural sleep pattern
Aim: To integrate advice from previous sessions and to maintain implementation at home
- to systematically rehearse elements of program
- to address implementation problems experienced
- to further adjust sleep schedules to maintain sleep efficiency
- to encourage and support people in maintaining their new sleep routines
- to encourage and support people in maintaining their new mental approach
- to learn relapse-prevention approaches if a sleep problem recurs

calculated (100 – [{SOL + WASO/ TIB} × 100]). Participants were trained to complete sleep diaries using established accuracy criteria.[29]

Because movement correlates with wakefulness and lack of movement with sleep,[30,31] wrist actigraphy was used to objectively estimate sleep for 14 nights before and after treatment. Actigraphs are small nonintrusive devices that record movement information by means of an accelerometer-microprocessor link. In this study, actigraphs (Cambridge Neurotechnology®, AW-4; Cambridge Neurotechnology Ltd., Cambridge, UK) were worn 24 hours per day on the nondominant wrist. An algorithm (maximum sampling frequency 32 Hz, recording all movement over 0.05 g., filters set 3-11 Hz) enabled proprietary Sleepwatch® software (Cambridge Neurotechnology Ltd) to estimate the sleep parameters SOL, WASO, and SE using 1-minute epochs. In the United States, these same hardware and software products are distributed by Minimitter Co. Inc. (Mini Mitter Co., Inc., Bend, Ore). Validity data are available on the following websites www.camntech.com and www.minimitter.com.

Several other clinical outcomes were assessed. These comprised global PSQI score, nighttime use of hypnotic medications, generic quality of life assessed using the Short Form-36,[32] and appraisal of clinical endpoints[33] (SOL and WASO ≤ 30 minutes; SE ≥ 85%) at posttreatment and follow-up.

Interventions

Cognitive Behavior Therapy

Participants assigned to CBT attended 5, weekly, 1-hour treatment sessions. These were conducted in groups of 4 to 6 participants in local general practice premises during the early afternoon or early evening. The content, aims, and objectives of each CBT session are summarized in Table 2 (further descriptions in Morin & Espie, 2003[16] or available from the first author of this paper). As can be seen in Table 2, the intervention included the common CBT components such as stimulus control, sleep restriction, and cognitive therapy strategies.

Therapists

To test a potentially generalizable model of insomnia care, we delivered CBT at "grass-roots" level, not in a specialized center or by a specialist psychologist or behavioral sleep medicine expert. Accordingly, 7 health visitors were trained to deliver CBT. In the UK, health visitors are community nurses with postqualification training and certification, who are generally based in primary care teams. They have a specific health education role and commonly encounter sleep disorders in their practice. We followed a model of "training-to-criterion" standards. That is, the health visitors had to demonstrate competence in the

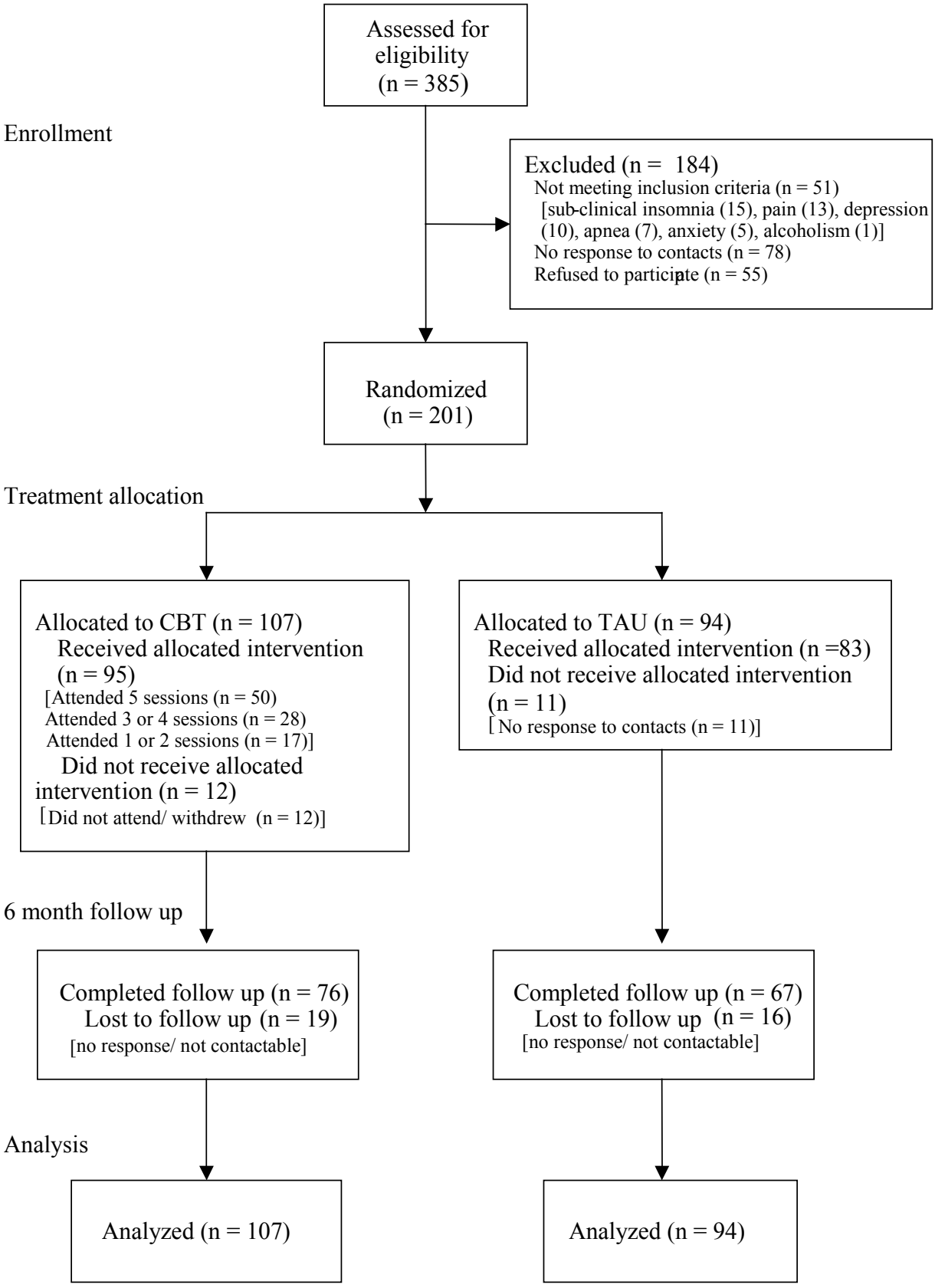

Figure 1—Participant Flowchart. CBT refers to cognitive behavior therapy; TAU treatment as usual.

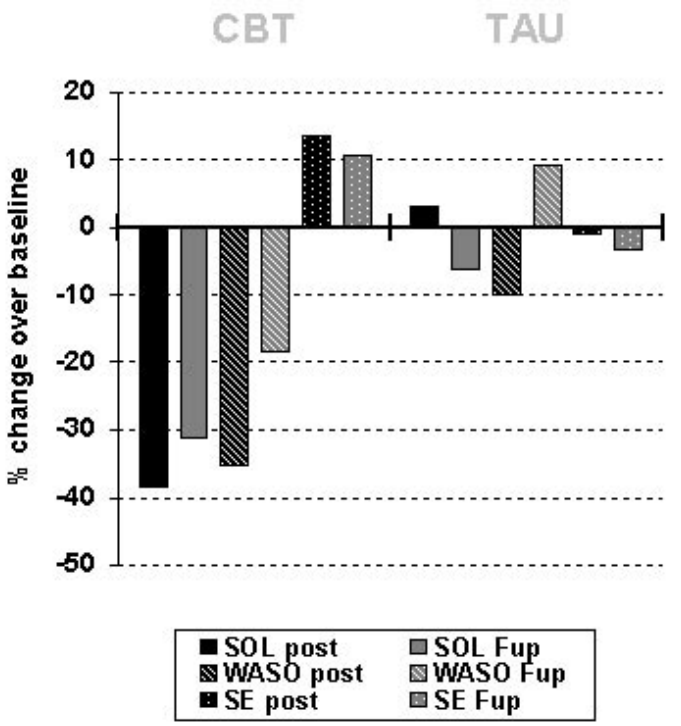

Figure 2—Sleep-diary changes at posttreatment and 6-month follow-up, expressed as percentage change from baseline values for the cognitive behavior therapy (CBT) and treatment as usual (TAU) groups. SOL refers to sleep-onset latency; WASO, wake after sleep onset; SE, sleep efficiency; post, posttreatment; Fup, follow-up

delivery of the CBT program. This was ensured by using a manualized therapy approach, participation in a short CBT course, apprenticeship learning opportunities, ongoing mentoring by an experienced clinical psychologist, and evaluation of audiotapes from randomly selected therapy sessions. Specific aspects of the training in CBT are summarized in the section on integrity/fidelity of treatment allocation.

Treatment as Usual

Effectiveness studies should replicate real clinical practices and reflect validity and generalizability.[34] Because we intended to recruit primary care patients with chronic insomnia, we expected concurrent physical and psychological symptoms, as well as concurrent treatments. The TAU comparison group thus represented normal clinical practice, in which GPs were free to offer appointments, to prescribe, and to maintain or discontinue prescriptions. What this meant in effect was that participants allocated to TAU received no additional help with their insomnia therapy, resulting from their participation in the study, but that their GPs were free to do whatever they would normally do. Indeed, in this respect, CBT was, in reality, a CBT plus TAU condition because the trial protocol explicitly permitted GPs (and other physicians and health professionals) to continue their health care provision uninterrupted with all the participants. TAU participants completed assessments as for the CBT condition but received no insomnia advice from the trial team or from our therapists. At the end of the protocol, the TAU group was provided with a booklet "The Good Sleep Guide," prepared by the first author for the National Medical Advisory Committee.[35]

Table 3—Demographic and Clinical Information on the Sample

Characteristic	CBT (n = 107)	TAU (n = 94)
Age, y[a]	54.4 ± 15.4	54.1 ± 14.4
Sex		
Women	72	65
Men	35	29
Civil status		
Partner	54	46
No partner	53	48
Working		
Yes	54	47
No	53	47
Location		
Glasgow	71	66
Edinburgh	36	28
Carstairs Deprivation Category		
1-2	37	18
3-4	23	24
5-7	47	52
Insomnia duration, y[a]	11.6 ± 9.79	10.6 ± 12.2
Insomnia presentation		
Constant	80	69
Episodic	27	23[b]
Sleep medication		
Yes	54	41
No	53	53
PSQI score[a]	12.7 ± 3.75	12.3 ± 3.55
ESS score[a]	6.05 ± 4.69	5.00 ± 4.26
Comorbid problems		
None	34	28
Physical health	11	12
Mental health	30	36
Physical and mental health	32	18
HADS-Anxiety score[a]	9.99 ± 4.10	9.63 ± 4.60
HADS-Depression score[a]	6.73 ± 3.66	7.07 ± 4.58

Data are presented as number, unless otherwise noted. CBT refers to cognitive behavior therapy; TAU: treatment as usual; PSQI: Pittsburgh Sleep Quality Index; ESS: Epworth Sleepiness Scale; HADS, Hospital Anxiety and Depression Scale.
[a]Data are presented as mean ± SD.
[b]2 missing cases.

Integrity-Fidelity of Treatment Allocation

The integrity of the treatment allocations was ensured as follows: (1) Nurse therapists attended a 12-hour, 2-day course on sleep disorders, working with groups, CBT principles, and instruction on the CBT program. (2) Therapists "sat in" on existing CBT groups and maintained an informal peer support network. (3) An experienced psychologist with training in behavioral sleep medicine acted as a mentor/consultant but did not work directly with participants. (4) The CBT program was manualized, following a recent study.[21] It comprised therapist notes, PowerPoint presentations (15 slides per session), worksheets for "break-off" times, and take-home notes with implementation guidelines. (5) Therapy sessions were audio-recorded, allowing appraisal of fidelity of CBT administration. (6) TAU participants were not seen for assessment at times when or in places where CBT assessment or intervention was operating. (7) GPs were advised of TAU allocations but were not provided with copies of CBT materials. (8)

CBT participants were asked to not make copies of materials. To strengthen this instruction, all materials were prominently marked as copyrighted.

Statistical Methods

Data analyses followed a conservative intention-to-treat model, with all allocated participants who provided baseline data included in a series of 2 (group: CBT, TAU) × 3 (time: baseline, posttreatment, follow-up) repeated-measures analyses of variance (ANOVA). Missing values were replaced using last observation carried forward, consistent with the methodology applied by Jacobs et al[57] in their intention-to-treat, placebo-controlled, trial of insomnia treatments. This approach was preferred to HLM analysis, which would have been more appropriate if the participants had been randomly assigned as groups (a "cluster" randomized trial). Participants were randomly assigned individually between the therapies. Consequently, the analysis undertaken reflects the nature of the underlying randomization (and the associated permutation test). Significant Group × Time interactions were explored posthoc by within and between sample t-tests to locate the effect. Percentage change over baseline and relative effect sizes ($d = M_1 - M_2 / \sqrt{[\{\sigma_1^2 + \sigma_2^2\}/2]}$) were computed to estimate treatment impact.[36] Consideration was paid subsequently to potential predictors of outcome using linear regression methods.

Role of the Funding Sources

Neither funding source (Chief Scientist Office, Scottish Executive; Dr. Mortimer and Theresa Sackler Foundation) participated in the study design, data analysis, or writing of this report.

RESULTS

Participant Flow

Three hundred and eighty-five adults were assessed for eligibility of whom 51 (35 women and 16 men; mean ± SD age 53.1 ± 11.2 years) were excluded, largely because of unidentified or untreated problems (Figure 1). A further 133 (87 women and 46 men; mean age 51.5 ± 17.9 years) did not complete the baseline sleep diary, leaving 201 who met criteria and were randomly assigned to treatment.

Demographic and clinical characteristics of this sample are presented in Table 3. Participants were typically middle-aged and had had insomnia for more than 10 years. Two thirds were women. Half were on sleep medication, primarily using benzodiazepine hypnotics (1 in 3 of those on hypnotic medication was on a benzodiazepine receptor agonist). Only one third of the sample had no comorbid problems. Almost 60% of the participants had some degree of mental health problem (most commonly depressive symptoms and generalized anxiety). Indeed, consistent with this finding, on the HADS, 118 (59%) scored in the clinical "caseness" range (> 10) for anxiety and 86 (43%) for depression.[23] More than one third of the sample had comorbid physical disorders, either alone (11%) or concurrent with a mental health problem (25%). Disorders of the cardiovascular (eg, high blood pressure), musculoskeletal (eg, arthritis, pain) and endocrine systems (eg, diabetes) were among the most common. Concurrent pharmacologic treatments, therefore, ranged through antidepressant, anxiolytic, β-blocker, antiinflammatory, and analgesic medications, either singly or in combination.

Participants were drawn from across the socioeconomic spectrum. The only baseline difference (Table 3 data) was a modest overrepresentation of higher socioeconomic status (DepCat group 1-2) in CBT (34.5%) relative to TAU (19%) [$\chi^2 = 6.02$, df = 2, P = 0.049]. Compared with Scottish population data,[37,38] our sample had a lower representation of midband 3 to 4 (23.5% vs 45%), a higher representation of band 1 to 2 (27.5% vs 10%), and a similar representation of the lowest band 5 to 7 (49% vs 45%).

Nineteen CBT groups were run by the therapists. Three quarters of CBT participants attended 3 or more therapy sessions. For missed sessions, participants "caught up" via discussion at the end of the subsequent attended session. The 23 participants lost from CBT/TAU during the treatment phase (Figure 1) did not respond to 2 subsequent letters or phone calls and did not differ from completers on presenting characteristics. Respectively, 80% and 81% of those receiving CBT and TAU provided data at 6-month follow-up. Thus, "drop-out" rates for CBT and TAU were similar during both intervention and follow-up. Baseline variables for noncompleters did not differ significantly from those for completers. Likewise, preliminary analyses of dependent variables revealed no differences across sites. Data therefore were pooled. No adverse events were reported anecdotally with either CBT or TAU.

Self-reported Sleep

Summary data (mean ± SD) for CBT and TAU at each assessment point are provided in Table 4. Visual inspection of these data suggests that the TAU group slept somewhat better than the CBT group at baseline. However, there were no statistically significant baseline differences in either self-reported or actigraphic sleep. Difficulty initiating sleep (SOL) declined more following CBT than TAU, the significant Group × Time interaction ($F_{2,198} = 6.64$, P = 0.002) being explained by a between-group difference at posttreatment (t = 2.74, df = 199, P = 0.004). Differences between CBT and TAU failed to maintain statistical significance at follow-up (P = 0.079). Repeated-measures ANOVA on difficulty maintaining sleep (WASO) also yielded a significant interaction term ($F_{2,198} = 7.12$, P = 0.001). However, independent samples t-tests revealed no significant differences between CBT and TAU at posttreatment (P = 0.10). SE is a measure of sleep continuity across the night. ANOVA yielded a significant Group × Time interaction ($F_{2,198} = 8.07$, P < 0.001), which was accounted for by higher posttreatment SE in the CBT group relative to TAU (t = 1.70, df = 199, P = 0.045). At follow-up, this difference was not statistically significant (P = 0.06). Total sleep time increased by 12 minutes after CBT and by 21 minutes at 6 months, compared with a 5-minute reduction for TAU. These differences, however, were not statistically significant. The interaction terms for SOL, WASO, and SE remained significant after correction for multiple comparisons on sleep-diary measures (P = 0.05/4 = 0.0125). In order to be conservative, we also repeated the above analyses entering baseline values as covariates. This did not alter any of the above findings.

Figure 2 presents percentage-change data for CBT and TAU at posttreatment and follow-up. These data illustrate marked improvement following CBT with some loss of effect at 6 months. Little change was observed with TAU. In the CBT arm, posttreat-

Table 4—Sleep Data, Before and After Intervention, and at 6-Month Follow-Up for Cognitive Behavior Therapy and Treatment as Usual Groups

SLEEP OUTCOMES	CBT	TAU
Sleep Diary		
Sleep-onset latency, min		
Baseline	60.5 ± 50.5	54.0 ± 41.1
Posttreatment	37.2 ± 42.9	55.7 ± 42.2
6-month follow-up	41.7 ± 45.5	50.7 ± 33.0
Wake after sleep onset, min		
Baseline	101.9 ± 88.2	85.0 ± 71.4
Posttreatment	66.1 ± 50.3	76.6 ± 53.1
6-month follow-up	83.0 ± 76.3	92.8 ± 63.8
Sleep efficiency, %		
Baseline	68.0 ± 19.1	73.5 ± 16.7
Posttreatment	77.1 ± 15.6	72.7 ± 16.7
6-month follow-up	75.3 ± 15.7	71.1 ± 16.7
Total sleep time, h		
Baseline	5.54 ± 1.69	5.93 ± 1.46
Posttreatment	5.74 ± 1.19	5.91 ± 1.44
6-month follow-up	5.89 ± 1.27	5.85 ± 1.21
Actigraphy		
Sleep-onset latency, min		
Baseline	23.3 ± 29.7	21.4 ± 23.3
Posttreatment	22.7 ± 22.8	20.7 ± 22.2
Wake after sleep onset, min		
Baseline	73.6 ± 37.1	56.1 ± 20.3
Posttreatment	59.0 ± 25.3	53.8 ± 23.7
Sleep efficiency, %		
Baseline	80.9 ± 9.62	84.0 ± 6.03
Posttreatment	82.7 ± 5.71	84.3 ± 4.00

CBT refers to cognitive behavior therapy; TAU, treatment as usual.

ment SOL reduction was 39% (23 minutes; d = 0.58) and was 31% (19 minutes) at follow-up (d = 0.36). For WASO, these CBT changes were 35% (36 minutes) and 19% (19 minutes), respectively (both d = 0.35). This maintained effect size at posttreatment was influenced by an increase in WASO of 10% (8 minutes) with TAU. The relative SE increase over baseline was 13% at posttreatment (d = 0.68) and was 11% at follow-up (d = 0.57) for the CBT group. These changes represented absolute increases in SE of 9.1% and 7.3% respectively. TAU was associated with a slight tendency to reduced SE

Actigraphic Estimates of Sleep

Data from 126 participants were available; 69 participants were allocated to CBT (45 women and 24 men; mean age 54.7 ± 14.6 years) and 57 to TAU (38 women and 19 men; mean age 54.7 ± 13.7 years). Demographic and clinical characteristics were similar to those of the full study sample. No effect of treatment was observed on actigraphy-derived SOL or SE (Table 4). For WASO, both Group ($F_{1,124}$ = 11.84, P = 0.001) and Time ($F_{1,124}$ = 9.55, P =0.002) main effects were significant, as was the Group × Time interaction ($F_{1,124}$ = 5.01, P = 0.027). Accordingly, a pretreatment-posttreatment change score was calculated, and an independent samples t-test conducted. This revealed a significantly greater reduction in WASO following CBT, as compared with TAU (t = 2.28, P = 0.024).

Actigraphic scores for SOL and WASO were lower and, for SE, were higher than sleep-diary estimates. Intercorrelations of weekly mean data for SOL (r = 0.340, P < 0.001), WASO (r = 0.182, P = 0.041), and SE (r = 0.275, P =0.001) were modest.

Table 5—Other Clinical Outcomes for Cognitive Behavior Therapy and Treatment as Usual Groups

OTHER CLINICAL OUTCOMES	CBT	TAU
Pittsburgh Sleep Quality Index		
Baseline	12.7 ± 3.75	12.3 ± 3.55
Posttreatment	9.84 ± 4.17	11.3 ± 3.68
6-month follow-up	8.40 ± 4.14	11.2 ± 3.24
Medication use per night		
Baseline	0.48 ± 0.92	0.61 ± 0.85
Posttreatment	0.30 ± 0.60	0.53 ± 0.68
6-month follow-up	0.26 ± 0.49	0.47 ± 0.70
SF-36		
Physical functioning		
Baseline	67.1 ± 26.5	68.5 ± 24.7
Posttreatment	71.8 ± 18.7	71.4 ± 17.9
Social functioning		
Baseline	61.9 ± 26.4	60.3 ± 28.2
Posttreatment	65.0 ± 20.0	62.4 ± 25.4
Physical role limitation		
Baseline	59.8 ± 28.9	61.7 ± 27.4
Posttreatment	62.5 ± 19.7	60.2 ± 21.2
Emotional role limitation		
Baseline	60.6 ± 23.7	62.8 ± 24.2
Posttreatment	67.0 ± 17.4	62.8 ± 21.9
Mental health		
Baseline	45.0 ± 12.9	46.4 ± 14.7
Posttreatment	50.2 ± 8.20	47.8 ± 14.2
Energy/vitality		
Baseline	38.4 ± 16.0	42.3 ± 15.1
Posttreatment	45.8 ± 12.0	43.9 ± 14.1
Pain		
Baseline	57.5 ± 22.9	59.8 ± 22.8
Posttreatment	59.1 ± 21.5	60.8 ± 19.8
General health perceptions		
Baseline	55.0 ± 22.0	56.2 ± 20.1
Posttreatment	60.6 ± 16.8	58.1 ± 18.1

Data are presented as mean ± SD; for medication use, this is mean ± SD number of tablets taken per night. CBT refers to the cognitive behavior therapy; TAU, treatment as usual; SF-36: Short Form-36.

Other Clinical Outcomes

Global sleep disturbance reduced by at least 4 PSQI points (more than 1 SD) at 6-month follow-up under CBT, compared with a 1-point change under TAU (Table 5). The Group × Time interaction effect was significant ($F_{2,198}$ = 3.83, P = 0.023), accounted for by PSQI reductions both at posttreatment (t = 1.68, df = 199, P = 0.048) and follow-up (t = 2.97, df = 199, P = 0.002) in the CBT group, compared with TAU. There was a nonsignificant reduction of hypnotic consumption in both CBT and TAU (Time main effect $F_{2,198}$ = 2.68, P = 0.074) but no significant interaction.

Following CBT, 32 participants (30%) achieved a SOL of 30 minutes or less, with 35 (33%) achieving this endpoint at 6 months. In TAU, the comparable figures were 17 (18%) and 21 (22%), respectively. These effects represent a significant improvement following CBT relative to TAU at posttreatment (χ^2 = 4.67, df =

1, P = 0.022; Fisher exact Test) but not at follow-up (χ^2 = 2.17, df = 1, P = 0.094). Fourteen participants (13%) had WASO of 30 minutes or less after CBT, with 20 (19%) obtaining this cut-off at follow-up, compared with 10 (11%) and 11 (12%), respectively, following TAU. The achievement of SE of at least 85% was also included as a conventional threshold value for normal sleep.[55,56] This criterion was achieved by 28 (26%) of CBT participants after therapy and by 21 (20%) at follow-up. For TAU, these outcomes were obtained by 16 (17%) and 13 (14%), respectively. These WASO and SE indexes of change were not significantly different upon statistical analysis.

The SF-36 was completed before and after treatment (see Table 5). Higher values indicate better perceived health. Time main effects were observed in 4 domains: physical functioning (F = 5.82, P = 0.017), mental health (F = 12.9, P = 0.001), energy/vitality (F = 15.7, P < 0.001) and general health (F = 8.89, P = 0.003) (all df 1,199). Significant Group × Time interactions, suggesting better treatment response after CBT, were obtained for 2 domains: mental health (F = 4.29, P = 0.040) and energy/vitality (F = 7.92, P < 0.005). A nonsignificant effect was observed for emotional role limitation to respond better to CBT (F = 3.43, P = 0.066).

Predictors of Outcome

It is important also to investigate if there were any factors specifically associated with better or poorer treatment response. Demographic (eg, sex, age, socioeconomic status, location of group), clinical (eg, comorbidities, psychopathology), sleep (eg, duration of insomnia, medication) and treatment-related data (eg, attendance rate, therapist, group attended) were available to inform such analyses. At the conceptual level, such independent variables are divisible into "moderator (present at baseline) and "mediator" (treatment-related) influences upon outcome. SE change from before to after treatment was selected as the dependent variable because SE is a recognized summary index of sleep disturbance and because SE demonstrated the largest posttreatment effect size. Stepwise linear regression revealed that only 2 variables contributed significantly to the prediction of SE change for the CBT group (F = 26.12, P < 0.001). Baseline SE (a moderator variable) entered on the first step (Adj. R^2 = 0.433; β = 0.611; P < 0.001) and frequency of attendance at CBT sessions (a mediating variable) contributed a small amount of additional explanatory variance (Adj. R^2 = 0.469; β = -.218; P = 0.033).

DISCUSSION

Insomnia is a problem with population prevalence and comorbidity so high that a clinical effectiveness study is required to establish if a promising intervention like CBT can be translated into a community-based treatment. CBT is normally regarded as a complex and specialized intervention; therefore, as well as effectiveness, there is the issue of feasibility. This study, therefore, investigated the impact of manualized, nurse-administered, small-group CBT on relatively unselected "real-world" participants with severe and persistent insomnia.

Our intention-to-treat data offer some support for the clinical effectiveness of CBT for insomnia. Significant reductions, totalling around 60 minutes per night, in symptom measures of SOL and WASO were observed with CBT, and SE increased by 9%. TAU did not yield comparable benefits. ANOVA models suggest that these posttreatment improvements were more convincing for sleep latency than for wakefulness during the night, and this is confirmed by effect-size data (d = 0.58 and d = 0.35, respectively). Effect size was greatest for SE (d = 0.68) indicating that, under CBT, participants were reliably sleeping through a greater proportion of their time in bed. Global sleep quality, as measured on the PSQI, also improved following CBT. Follow-up data, however, suggest some loss of therapeutic effect, particularly in WASO, although the WASO effect size was sustained, perhaps because TAU participants were somewhat more wakeful at 6 months. Effect sizes for SOL and WASO at follow-up were small to medium but remained relatively robust for SE (d = 0.57). Mean CBT reductions for SOL and WASO, in previous efficacy studies, have been about 30 minutes each,[33] similar to our findings. However, average effect sizes have been 0.88 and 0.65, respectively, considerably larger than our results. This may reflect the more severe and complex presentation of our patient group. There is also some suggestion that our study may have been underpowered to detect between-group differences at 6-month follow up, when nonsignificant probabilities for SE (P = 0.06) and SOL (P = 0.079) were obtained. Posthoc power calculations indicate that sample sizes of 235 (for SE) and 300 (for SOL) would have been required to achieve an α value of 0.05 at 80% power. We had originally planned to enrol 240 participants but achieved only 201. These data may be indicative of the considerably larger sample sizes required for effectiveness research, relative to efficacy research, because of the greater within and between-subject variability in clinical samples.

Nevertheless, and consistent with other recent data,[39] it is encouraging that factors such as the chronicity of the insomnia disorder, the absence or presence of physical and mental health comorbidities, and participant age and sex did not emerge from regression analysis as explanatory factors associated with therapeutic response to CBT. As might be expected from the law of initial values, baseline SE was the main predictor of SE improvement, explaining 43% of variance in the treated group. Thus, baseline data may moderate treatment response but, from our data, primarily in the sense that high baseline values provide greater room for improvement. Attendance rate at CBT sessions added a further 3.6% of explanatory variance and was the only treatment-related mediator of outcome. People who attended more often were likely to do better. This too is an important finding, reinforcing the importance of motivational aspects of CBT and of helping patients to conceptualize the program as a course of treatment. The interaction of insomnia severity with the likelihood of committing to achieving and sustaining change in sleep-related behavior and cognition appears worthy of further dedicated research effort.

Sleep self-report data were not mirrored by actigraphically estimated sleep, either in terms of capturing the baseline complaint of insomnia or in terms of outcome. Only WASO data demonstrated treatment-related impact, whereas, on sleep diary reports, CBT was associated with greater reduction in SOL and increase in SE. These findings parallel other recent work showing limited impact of CBT on actigraphically determined sleep.[40-42] Only 1 study, on insomnia patients with chronic pain, has demonstrated both subjective and actigraphic sleep improvement.[43] Indeed, a systematic review and practice parameters statement, published since we began this research, suggests a limited role for actigraphy applied to insomnia intervention research.[44,45] Studies have found that actigraphy consistently produces different estimates

of sleep time and number of awakenings and lower estimates of sleep latency than do sleep diaries,[46,47] and correlation between self-report and actigraphy has been generally poor.[48-50] However, such limited correlation between entirely different modes of assessment should not be unexpected. In considering the role of actigraphy in sleep assessment, Tryon[51] makes the point that the observed modest coefficients of validity of actigraphy (in relation to polysomnography) actually exceed those associated with many medical and psychological tests. Consequently, the significant impact of CBT upon actigraphically estimated WASO in the present study is interesting in part because it contrasts with the sleep-diary improvements that were observed primarily in the SOL and SE domains.

We used conservative criteria to investigate clinical endpoints. Of CBT treated. 20% to 30% achieved these endpoints (below threshold for insomnia disorder), compared with 10% to 20% of TAU participants. Compared with absolute reductions in SOL, WASO, and SE complaints, these outcomes are relatively disappointing for CBT and may reflect the initial severity of the sleep disorders in this study. That is, although CBT was associated with greater symptomatic improvement, many participants remained in the clinical range at follow-up. Certainly, if even more stringent criteria were applied (eg, reduction in symptom score of at least 0.5 SD plus 30 minutes or less of SOL or WASO), we would have obtained very few responders to CBT. Likewise, although significant PSQI-based sleep-quality improvement was achieved and sustained in the long term following CBT, the 6-month follow-up mean value of 8.4 remained considerably higher than the cut-off of 5 used for normal sleep.

Such results raise the long-standing issue in insomnia outcome research concerning the relative paucity of treated participants who endorse becoming normal sleepers after treatment. In the context of this particular study, one possible explanation for the disappointing clinical endpoints is that there may have been insufficient treatment offered through the CBT-group program, at least for a proportion of the patients. Whereas this model of care may be sufficient for some, it may not be sufficient for the majority of clinical insomnia cases. The use of more sessions, more highly skilled therapists, tailored CBT interventions, or a combination of CBT and pharmacotherapy treatments, along with proven methods to achieve and to sustain high levels of patient assimilation of treatment information and adherence to treatment protocol may all make a difference to outcomes in insomnia clinics. Although work is steadily advancing in these areas, we do not yet have the algorithms to enable us to make these judgements in an informed manner.

Nevertheless, our CBT participants demonstrated more than mere sleep-symptom change. On the SF-36, health-related quality-of-life improvement was found in domains reflecting mental health and vitality. These results suggest that CBT for insomnia may be associated with generalized benefits to everyday functioning. This is consistent with contemporary understanding of insomnia not only as a disorder that impairs the sleep experience, but also one that negatively impacts the day.[5,6] Our findings of quality-of-life change following CBT for insomnia parallel other recent data using the SF-36.[52,53]

Notwithstanding our earlier comments about sampling adequacy, our sample size (n = 201) was considerably larger than that of any previous report of insomnia treatment. Efficacy studies typically have comprised 40 to 100 participants.[33] More importantly, our participants were clinically identified, and 70% had comorbid mental or physical health problems. Only 51 of 385 potential participants were excluded, largely because of suspected untreated disorders. However, a further 133 who met criteria and consented to participate in the study, withdrew prior to random allocation to treatment. Unfortunately, we have limited data on the reasons for these withdrawals. Delays in processing individuals may have contributed to some nonresponses. Also, we suspect that many individuals who were routine users of hypnotics on prescription, who initially expressed interest when contacted by their GPs, thereafter withdrew. This may reflect the importance of patient "readiness" to adopt a CBT approach in the real-world clinical setting. Nevertheless, 50% of randomly assigned participants were on hypnotics. The personal, socioeconomic, and clinical profiles of our participants suggest that we did identify our target community population and that they had severe and chronic insomnia. Total wakefulness per night was around 2.5 hours, and baseline SE was around 70%.

We wanted to test a potentially generalizable model of care that had proven to be beneficial in a more limited previous study.[21] From our experience, it is feasible for nurses based in primary care to learn, and to deliver, a CBT program, and our results are promising for what might be regarded as a "first-line" insomnia intervention. By providing group treatment using a trained nurse, the "per patient" costs may be minimized. Crucially, however, the use of a manual ensured treatment integrity and fidelity, and, alongside training, supervision and case review, would seem a crucial component of any program "roll-out." We have summarized elsewhere how a skilled clinical psychologist or behavioral sleep medicine specialist could operate an insomnia "triage" system, allowing nurse-led group-based CBT to complement individual therapies.[54] This model, of course, requires further evaluation. Moreover, our approach would need to be tailored to the operational characteristics of other healthcare systems. Perhaps the National Health Service in Scotland is more amenable to this type of intervention in primary care because of the established health-provision role of primary care nurses (health visitors) and the day-to-day interactions between clinical psychologists and GP services. In the UK, generally, the services provided by the National Health Service are all government funded, using revenue from the taxation system. The common co-occurrence of insomnia with depressive and/ or anxiety symptoms in primary mental health care also highlights the need for investigation of CBT for insomnia as an adjunct to existing community treatment for these disorders. The intervention described here might be readily adapted for that purpose.

In interpreting our findings, several factors merit consideration. First, the dataset are limited to self-report and actigraphic estimation of sleep. Although the former in particular is appropriate to the clinical-effectiveness question, appraisal of effects upon polysomnographically defined sleep at home would be informative. Second, CBT did not specifically target hypnotic drug use. Reductions in use were observed with both CBT and TAU. This may reflect an implicit focus upon non-pharmacologic management. Nevertheless, other recent work has demonstrated that CBT can be effectively applied to hypnotic reduction as a primary outcome.[52] Third, the use of TAU as the control condition of choice for clinical effectiveness study also imposes limitations. Important among these is the fact that CBT does not control for the additional time, attention, and demand characteristics associated with provision of a therapy. Although such factors have been

controlled for in previous CBT trials, there remains the possibility that such nonspecific treatment factors played a part in the response to the CBT arm of the present study. Finally, both groups in effect had "treatment as usual," and, so, other factors may have influenced our results, even though participants were appropriately randomly assigned to a treatment group. Efficacy trials ensure better control over variation, whereas effectiveness studies give indications about potential for service implementation. Clearly, both methodologies are required to test the important possibility that CBT could become the treatment of first choice for persistent insomnia in primary care.

ACKNOWLEDGMENTS

This study was supported by the Chief Scientist Office, Scottish Executive Health Department (CZH/4/Z) and the by the Dr. Mortimer and Theresa Sackler Foundation

The authors gratefully acknowledge the administrative and practical support of Colette Fulton and Lucy McCloughan (Lothian Primary Care Network), Bridie Fitzpatrick (WestNet, Glasgow) and Brian Rae (Primary Care Division R & D, NHS Greater Glasgow). We are also grateful to Simon Dixon (University of Sheffield) for his assistance with analysis of the SF-36 data and to Lauren M. Marchetti and Carolyn J. Espie for their work on data entry. Most of all we thank the seven Health Visitors who acted as CBT therapists for the study, the General Practitioners who identified participants and, of course, the patients who participated in the trial.

TRIAL REGISTRATION

NCT00170417

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