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Long-term effectiveness of a short-term cognitive-behavioral group treatment for primary insomnia

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Abstract The long-term effectiveness of a short-term cognitive-behavioral therapy was evaluated. The structured group treatment consisted of six weekly sessions and included progressive muscle relaxation, cognitive relaxation, modified stimulus control with bedtime restriction, thought stopping and cognitive restructuring. Twenty patients with chronic primary insomnia took part in the study. All patients were referred by physicians for diagnosis and therapy of insomnia. During a waiting period of six weeks prior to treatment, patients did not experience any change of their sleep parameters. After therapy, patients improved their total sleep time and sleep efficiency and reduced their sleep latency and negative sleep-related cognitions. Furthermore, depression scores decreased. Most of the treatment effects were significant at the end of the treatment and remained stable over the long-term follow-up, which was evaluated after a mean of almost three years (35 ± 6.7 months). The subjective estimated total sleep time improved from 298 ± 109 min prior to therapy to 351 ± 54 min at the end of treatment, to 376 ± 75 min at the 3-month follow-up, to 379 ± 58 min at the 12-month follow-up and to 381 ± 92 min. at the long-term follow-up.

Key words Primary insomnia · Cognitive-behavioral therapy · Group therapy

Introduction

Difficulties in initiating and maintaining sleep are among the most common health problems. Thus, in the general population prevalence rates of insomnia according to DSM-IV were found of 19 % [17] and 12.7 % [29], respectively. Among patients in general practices about 19 % suffered from insomnia corresponding to the criteria of DSM-III-R [12]. According to operationalized diagnostic criteria like the DSM-IV, DSM-III-R or the ICD–10, insomnia is defined as a complaint of prolonged sleep latency, increased frequency of nocturnal awakenings, or no refreshing sleep for a duration of at least four weeks. The criteria include furthermore a relevant daytime impairment due to the insomnia like tiredness, problems with concentration and efficiency, or mood disturbances. Primary insomnia is not caused by a physical disease or a psychiatric disorder, but has multiple causes and maintaining conditions. In many patients disturbed sleep is characterized by hyperarousal including physical and cognitive tension in bed, worrying and ruminating about sleep, fear of the consequences of insomnia, and unfavorable sleep habits, e. g. excessive time spent in bed.

The most common treatment is the pharmacological therapy with benzodiazepines and the benzodiazepine-analogues like zolpidem or zopiclone. Nowell et al. [28] found, that benzodiazepines and zolpidem are effective treatments for primary insomnia. They conclude, however, that there are insufficient data to prove treatment effectiveness for longer durations (> 7 days) beyond the discontinuation of hypnotic medication. Furthermore, the treatment with benzodiazepines bears the risk of adverse effects, rebound insomnia, and dependence [13–15]. Different non-pharmacological treatment strategies like relaxation training, stimulus control, sleep restriction and sleep hygiene have been developed and evaluated. Two meta-analyses [24–26] indicated the effectiveness of most of these non-pharmacological treatments.

The aim of this study was to evaluate the long-term effectiveness (after a mean of 35 ± 6.7 months) of a short-term cognitive-behavioral group therapy program for patients with primary insomnia. In a previous study [33] we analyzed the effectiveness of a group treat-

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ment consisting of eleven weekly sessions. The previous therapy program consisted of sleep-related treatment elements (relaxation, sleep-wake structuring, cognitive techniques) and daytime related therapy techniques (e. g. problem solving, stress management). Most of the improvements were already found after the first six sessions (sleep-related techniques), while the second half of therapy (daytime-related techniques) had only minimal additional effects. Therefore, the daytime-related therapy techniques were omitted and the program was reduced from eleven to six sessions. The structured treatment program of the present study is symptom-focused and combines various, effective treatment strategies. The therapy includes relaxation techniques, psychoeducation (information on sleep, sleep hygiene and chronobiology), stimulus control, restriction of time in bed, and cognitive techniques like thought stopping and cognitive restructuring. In this exploratory study we sought to evaluate the effectiveness of the treatment in a natural setting. Thus only patients were included who were referred by their physicians to our sleep disorders outpatient clinic for insomnia diagnosis and therapy; we did not recruit patients through newspaper advertisements.

Methods

Sample

Twenty-one patients meeting the criteria of primary insomnia according to DSM-IV participated in the study. According to these criteria patients had suffered at least four weeks from problems of sleep onset, sleep maintenance and / or no refreshing sleep and – as a consequence of their sleep disorder – had daytime impairments such as tiredness or problems with concentration and efficiency. No patient had a comorbid psychiatric or a sleep-relevant somatic disorder. Three patients (15 %) had sleep onset insomnia (defined as thirty minutes or more for sleep onset), six patients (30 %) maintenance insomnia (defined as thirty minutes or more of wakefulness after sleep onset and / or three or more awakenings during the night) and 10 patients (50 %) had mixed insomnia (meaning problems with sleep onset and sleep maintenance). One patient did not feel refreshed after awakening according to the criteria of DSM-IV.

One female patient dropped out after the second session due to personal reasons. All patients gave their informed consent after the therapy was fully explained. The mean age of the twenty patients who completed the study was 43.0 (± 12.2) years. Thirteen of the patients were female (65 %). All patients suffered from a chronic insomnia: the mean duration of insomnia was 10.7 (± 10.6) years with a minimum of one year and a maximum of 38 years.

All patients were recruited from the outpatient sleep disorders clinic of the Department of Psychiatry and Psychotherapy of the University of Freiburg and had been referred by a physician for diagnostic evaluation and treatment of insomnia. Patients were examined by an experienced physician to rule out any psychiatric disorders and physical diseases, which might affect sleep. Afterwards patients underwent two nights of polysomnography to exclude clinically relevant sleep apneas (> 5/h) and nocturnal periodic leg movements with EEG arousal (> 5/h). Twelve patients were taking sleep medication at the beginning of therapy: six patients used benzodiazepines or benzodiazepine-like hypnotics, two patients herbal medications, two patients sedative antidepressants, and two patients other hypnotic medications. Patients were allowed to continue their sleep medication during the therapy but received recommendations and encouragement to discontinue drug usage.

Description of the short-term cognitive-behavioral therapy

The structured treatment included six weekly sessions of 90 min each. The patients were treated in groups of four to eight. Each therapy group was led by one of two experienced psychologists (first and senior author) who had worked for several years in the sleep outpatient clinic and the sleep laboratory of the Department for Psychiatry and Psychotherapy of the University of Freiburg. To improve the therapists' fidelity to the treatment model, they met regularly for peer supervision.

The treatment program has been published in German as a manual for therapists [31]. To reinforce the self-management of insomnia problems the patients were supplied with a self-help manual [3], describing the different therapy techniques. They were encouraged to use this manual not only during the six weeks of treatment but also afterwards, especially if sleep problems continued or reoccurred. Three months after the end of the therapy the patients were invited to join another group meeting for a follow-up, where the group participants could discuss their changes in sleep patterns. This offer often motivated the patients to continue practicing the techniques which they had acquired during the therapy once the treatment had finished. The treatment consisted of the following sessions:

1st session: Progressive muscle relaxation (PMR): The patients learned a version of PMR which lasted about 15–20 min. The PMR was also fully described in the self-help manual; additionally all patients received an audiocassette with the instructions. Patients were to practice the PMR regularly once or twice a day, but during the first weeks of therapy not in bed until they were confident with it. This was to avoid early disappointment with PMR, as the technique requires time and practice to develop its positive effects.

2nd session: Cognitive relaxation with positive imagery [16]: Patients were instructed to choose their individually positive images, which were to be associated with relaxation and well-being. Patients were instructed to practice the cognitive relaxation during the PMR a minimum of once a day.

3rd session: Rules for a good night sleep:

- Information on sleep and sleep hygiene: Patients were given basic information about sleep and its changes over a lifetime (e. g. lessening of deep sleep with age) as well as information about common sleep myths (e. g. 'eight hours of sleep are necessary for everyone'). Sleep hygiene rules included, for example, avoiding alcohol and coffee particularly in the evening (details are described in the manual [3], for an overview see also e. g. [10]).
- Bedtime restriction (sleep-wake-rhythm structuring): Patients were asked how much total sleep time they individually needed as the minimum to feel refreshed and efficient the next day (not: how long they would like to sleep). Most of the insomnia patients replied that six to seven hours of sleep were sufficient to meet this minimum. Patients were instructed to limit their bedtime to their individual total sleep time needed plus 30 minutes.
- Stimulus-control technique [4]

4th session: Cognitive techniques I: The cognitive techniques focus on how to combat sleep-disturbing cognitive processes. The "worry chair" approach instructed patients not to take problems to bed with them, but rather to focus on them during the evening for at least one or two hours prior to bedtime and perhaps list them in writing and devise solutions. "Thought stopping" is a technique to block out sleep-disturbing cognitions, during the process of falling asleep, by saying or thinking 'stop' when the patients realized that they were worrying/ruminating in bed. They should then shift their attention from disturbing cognitions to neutral or positive topics. After three weeks of daily practice in PMR and positive imagery, patients were then encouraged to use these techniques in bed.

5th session: Cognitive techniques II: The process of identifying dysfunctional sleep cognitions, particularly misconceptions of insomnia causes, had started in the third session and was repeated and intensified in the fifth session. Patients were encouraged to question

generalized negative conceptions of their sleep problem and work out alternative cognitions. They learned to reattribute their misattributions of insomnia consequences, their anger and feelings of helplessness about the insomnia (for overview see [22]).

6th session: The final session summarized all of the above-mentioned techniques. In this session the therapist and the patients reflected on the individually relevant causes and maintaining conditions for their insomnia as well as on those techniques which were most suitable for them. For relapse prevention, which steps should be taken when sleep disturbances reappear, were discussed. The patients were encouraged to carry on with the therapy techniques for a few months even after their insomnia had stopped.

Design of the study

All patients spent a waiting period without psychological insomnia treatment of six weeks before entering the group therapy, allowing a comparison between the waiting time and treatment. The study consisted of the following questionnaire time points: T0 (prior to waiting time), T1 (end of waiting time = prior to therapy), T2 (after therapy), T3 (3-month follow-up), T4 (12-month follow-up) and T5 (long-term follow-up after at least 2 years, mean = 35 ± 6.7 months, range 25–42.5 months). At the beginning and the end of the waiting period, patients filled out the German version of the Pittsburgh Sleep Quality Index [6]. Prior to and after therapy, as well as at the follow-ups, patients received the PSQI and a questionnaire about sleep and insomnia-related negative cognitions [11]. Furthermore the Beck Depression Inventory (BDI) and the trait anxiety scale of the State-Trait-Anxiety Inventory (STAI) were used. At the 12-month and the long-term follow-up, patients received the questionnaires by mail with a prepaid return envelope. Patients who failed to answer within three weeks received another letter requesting them to return the questionnaires. At the long-term follow up, two patients did not answer, so that the last evaluation included only 18 patients.

Statistics

ANOVAs with repeated measurements were used to determine variances over the different time points with $p < 0.05$ one tailed. In addition, only if the ANOVAs were significant were single comparisons calculated using t-tests with Bonferroni corrections. For the statistical analysis the following variables were used: PSQI: sleep quality (PSQI global score), total sleep time (minutes), sleep onset latency (minutes) and sleep efficiency in percent (wake time after sleep onset was indirectly measured by sleep efficiency); FEPS: scores for 'focusing on sleep' and 'worrying/ruminating about insomnia'; BDI: total score; STAI: trait score (stanine). To analyze differences in treatment outcome for patients who did or did not respectively use hypnotics, a 2 (groups) x 4 (measure points) ANOVA was calculated.

Results

Waiting period

No significant changes in the sleep parameters occurred during the waiting period. The PSQI global score was 13.6 ± 3.5 at T0 and 13.0 ± 3.2 at T1 (t-test: t-value = 1.24, df = 19, p = 0.115), see Fig. 1. The total sleep time was 309.0 minutes ± 114.7 at T0 and 297.7 minutes ± 108.9 at T1 (t-value = 0.69, df = 19, p = 0.248). Sleep onset latency changed from 52.4 minutes ± 55.6 at T0 to 66.7 ± 82.4 at T1 (t-value = –0.85, df = 19, p = 0.202) and sleep efficiency from 63.4 % ± 19.2 at T0 to 59.1 % ± 22.8 at T1 (t-value = 1.03, df = 19, p = 0.158).

Therapy and follow-ups

During therapy all sleep parameters improved significantly and most of them stabilized over the follow-up time. The ANOVA for the PSQI global score over the therapy and follow-up time points was significant (F=20.1, df=3, 57, p=0.000), see Fig. 1 *for means and standard deviations*. Single t-tests between T1 and the subsequent time points showed significant improvements (T1-T2: t=5.51, df=19, p=0.000; T1-T3: t=5.60, df=19, p=0.000; T1-T4: t=5.15, df=19, p=0.000, T1-T5: t=4.19, df=17, p=0.019).

The ANOVAS were significant for the total sleep time (F=10.8, df=3, 57, p=0.000), the sleep onset latency (F=3.86, df=3, 57, p=0.014) and sleep efficiency (F=20.6, df=3, 57, p=0.000). Because of missing data from two patients at the last follow-up, the ANOVAs were repeated for the 18 patients whose data were available. All sleep parameters changed significantly: the ANOVA for the PSQI global score was significant (F=10.4, df=4, 68, p=0.000) as well as for total sleep time (F=5.8, df=4, 68, p=0.000), sleep onset latency (F=2.5, df=4, 68, p=0.050) and sleep efficiency (F=11.2, df=4, 68, p=0.000). Single t-tests were significant for all sleep parameters between T1 and the following time points: T2, T3, T4. For T5 (long-term follow-up) total sleep time and sleep efficiency were still significantly better than at baseline. Exact values and statistics of t-tests are given in Table 1.

Participation in the therapy program led to a significant reduction of focusing on and ruminating about sleep. ANOVAs were significant for focusing (F = 11.3, df = 3, 57, p = 0.000) as well as for ruminating (F = 2.8, df = 3, 57, p = 0.047). For T1 to T5 (n = 18 patients) the ANOVAs were also significant for the two variables focusing (F = 12.3, df = 4, 68, p = 0.000) and ruminating (F = 2.6, df = 4, 68, p = 0.043). The patients decreased focusing on sleep significantly over all time points, suggesting that the effect was stable at the follow-ups. Single t-tests were significant between T1 and all other time points (T2: t = 6.90,

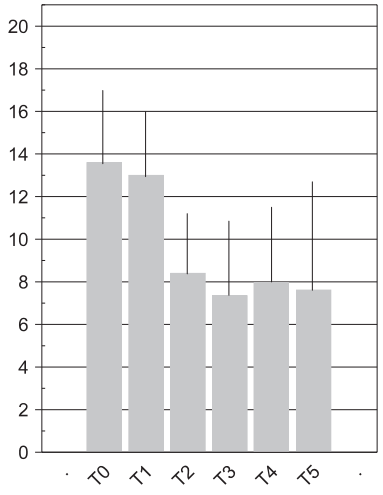

Fig. 1 Pittsburgh Sleep Quality Index (Global Score).

Tab. 1 Changes in different sleep parameters (PSQI), BDI and STAI

	T1 Prior to therapy	T2 After therapy	T3 3-month follow-up	T4 12-month follow-up	T5 Long-term follow-up	t-test T1 T2			t-test T1 T3			t-test T1 T4			t-test T1 T5		
						t	df	p	t	df	p	t	df	p	t	df	p
SOL (min)	66.7 (+82.4)	30.4 (+27.1)	25.9 (+23.8)	34.0 (+29.1)	42.0 (+48.3)	2.11	19	0.024	2.16	19	0.021	1.77	19	0.046	1.24	17	0.116
TST (min)	297.7 (+108.9)	351.0 (+54.2)	375.8 (+74.5)	379.0 (+57.7)	381.1 (+92.4)	–2.60	19	0.009*	–4.77	19	0.000*	–3.94	19	0.001*	–2.59	17	0.009*
SE (%)	59.2 (+22.8)	77.2 (+8.2)	81.8 (+12.8)	79.6 (+11.3)	78.7 (+17.6)	–4.31	19	0.000*	–5.51	19	0.000*	–4.73	19	0.000*	–3.66	17	0.001*
BDI	9.3 (+5.4)	6.8 (+4.4)	5.1 (+4.2)	5.9 (+6.8)	6.0 (+8.0)	2.62	19	0.008*	4.16	19	0.000*	2.21	19	0.020	1.76	17	0.048
STAI-T (stanine)	6.5 (+1.9)	6.1 (+2.2)	5.8 (+2.2)	5.9 (+2.0)	5.6 (+2.3)	1.57	19	0.067	3.29	19	0.002*	2.35	19	0.015*	2.06	17	0.027

SOL Sleep Onset Latency; *TST* Total Sleep Time; *SE* Sleep Efficiency Index (Total sleep time/Bedtime*100); *BDI* Beck Depression Inventory (Total score); *STAI-T* Trait score of State-Trait-Anxiety Inventory.
Means and standard deviations (in parentheses)
* = significant changes (Bonferroni corrected p < =0.012)

df = 19, p = 0.000; T3: t = 6.78, df = 19, p = 0.000; T4: t = 4.99, df = 19, p = 0.000; T5: t = 4.35, df = 17, p = 0.000). The decrease in ruminating was smaller than that for focusing and significant at the end of the therapy (t = 3.35, df = 19, p = 0.001) and for the 3-month follow-up (t = 2.33, df = 19, p=0.015), but not for the 12-month (t = 1.41, df = 19, p = 0.086) and the long-term follow-up (t = 1.56, df = 16, p = 0.069). Mean values are shown in Fig. 2. Correlations between focusing and PSQI global score were not significant for all time points.

Although initially not showing clinical depression, the primary insomniacs displayed a significant reduction of BDI scores at post-therapy and 3- and 12-month follow-ups (F = 4.7, df = 3, 57, p = 0.005). In the long-term follow-up the BDI score was still significantly reduced (F = 2.5, df = 4, 68, p = 0.048). After exclusion of sleep-related BDI items (items P and Q) the BDI score was still significantly reduced (F = 2.76, df = 3, 57, p = 0.05). Single comparisons showed significant reduction of the BDI score from baseline (7.0 + 4.8) to the 3-month follow-up (4.2 + 3.7, t = 2.83, df = 19, p = 0.01).

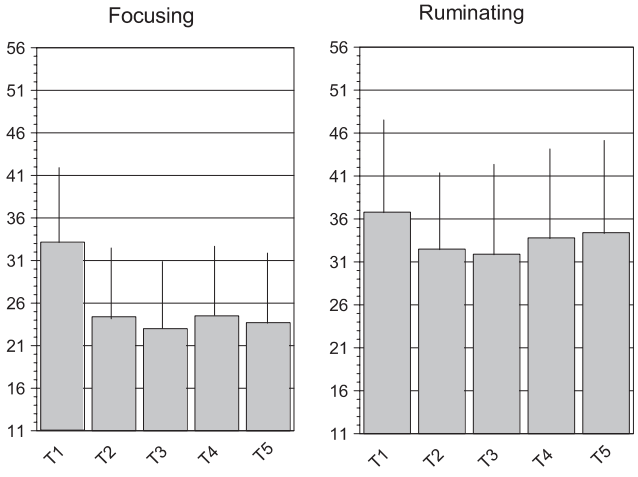

Fig. 2 Questionnaire about insomnia related cognitions (FEPS-II).

Concerning trait anxiety (STAI) significant reductions were observed at the 12-month follow-up (F = 4.1, df = 3, 57, p = 0.036), but not at long-term follow-up (F = 1.9, df = 4, 68, p = 1.18). The results of the BDI and the STAI are given in Table 1.

Intake of hypnotic drugs

Concerning intake of hypnotic drugs, 12 of the 20 primary insomniacs (60 %) were taking sleep medications at the beginning of therapy. Five patients discontinued hypnotic medications until the end of therapy, including four patients who discontinued benzodiazepines or benzodiazepine-like hypnotics. Thus, at the end of therapy, seven patients (35 %) were still using hypnotic medications. At the 3-month follow-up seven patients (35 %) and at the 12-month follow-up six patients (30 %) were using hypnotic medication (three patients used benzodiazepines, one patient herbal medication, one patient a sedative antidepressant and one patient another hypnotic). At the long-term follow-up four of the 18 patients (22.2 %) were taking sleep medication. Only one of the patients who had discontinued hypnotic medication relapsed and used hypnotic medication again, while the other patients abstained after discontinuation over the whole follow-up period.

As an explorative analysis patients who were completely free of hypnotics (n=8) over the whole study were compared to those who took hypnotics (at least at the beginning of therapy) to analyze which group improved the most. No significant differences in age between both groups were found (group without medication: 45.2 years ± 14.4, group with medication: 41.5 years ± 10.9, t-test: t-value: 0.65, df=18, p=0.527) found. The group effect of the 2 (groups) x 4 (time points) ANOVA for the PSQI global score was significant (F=7.1, df=1, 18, p=0.016), as was the time effect (F=20.4, df=1, 18, p=0.000), while the interaction effect was not (F=0.73, df=3, 54, p=0.540). Because of the significant group and

Tab. 2 Differences in therapy outcome between users and non-users of hypnotics using the Pittsburgh Sleep Quality Index (Global Score); means and standard deviations (in parentheses)

	Patients completely free of sleep medication	Patients who took sleep medication at least at T1	t-test between groups		
			t	df	p
T1	12.1 (+3.7)	13.5 (+2.8)	−0.99	18	0.168
T2	6.6 (+1.8)	9.5 (+3.0)	−2.46	18	0.012 *
T3	5.0 (+2.0)	8.9 (+3.8)	−2.59	18	0.009 *
T4	6.2 (+1.6)	9.0 (+4.4)	−1.72	18	0.051
T5	6.5 (+3.7)	8.5 (+6.1)	−0.84	16	0.206

time effects in the ANOVA, explorative single t-tests were carried out, which indicate that the group without medication improved significantly more than the group taking medication. As shown in Table 2, the two groups did not differ in their estimation of subjective global sleep quality (PSQI) at the beginning of therapy, while at the end of therapy they differed significantly: Patients who did not take hypnotic medication improved more than those who did take them. This difference continued to be present at the 3-month follow-up but was no longer present at the 12-month and long-term follow-ups. In addition, the results of the paired t-tests calculated for each group indicated that both improved significantly during therapy. These effects stabilized over all follow-ups (t-tests for both groups between baseline and all other time points all $p < 0.05$).

Discussion

Results from the present study indicate that the described short-term group therapy program is an effective treatment for patients with chronic primary insomnia. The aim of the study was to evaluate this therapy in a natural setting with patients, who were referred by their physicians and not recruited by newspaper advertisements. A methodological limitation of this study is the lack of another treatment or placebo condition. Therefore the waiting period, which all patients underwent prior to treatment, was used as a control condition. During the waiting period there were no changes in the sleep parameters of the patients. After six weekly cognitive-behavioral treatment sessions patients improved their total sleep time and sleep efficiency and reduced their sleep-related negative cognitions. Furthermore, depression scores decreased. Most of the effects stabilized over a long period of 35 (± 6.7) months after the treatment was finished. There are only a few other studies which have evaluated the long-term effectiveness of cognitive-behavioral therapies for primary insomnia over a period of two or more years [8, 23, 32].

The sleep parameters were most improved three months after the end of treatment, when patients had another group meeting as a follow-up. The anticipation of this post-treatment session reinforced their motivation to continue using the cognitive-behavioral techniques after the end of therapy. Therefore, it might be useful to offer some booster sessions after the end of treatment. Because all patients attended this follow-up session, it was not possible to validate this hypothesis with this sample, but subsequent studies could examine the effectiveness of booster sessions.

Patients were treated in groups, a setting that is both cost-effective and encourages patients to motivate each other, particularly for difficult therapy techniques like bedtime restriction. Meta-analyses show that individual therapy has no [26] or only a slightly [24] better outcome than group therapy. Results from the present study support the use of group therapy in the treatment of primary insomnia.

Patients reduced their focusing on sleep. Decreasing sleep-related negative cognitions while staying in bed seems to be an important treatment effect. Studies which analyze the causes and maintaining factors of primary insomnia found that patients stated sleep-related negative cognitions as an important sleep disturbing factor [18, 27]. Because the treatment in this study was a multi-component therapy program, the effectiveness of the cognitive treatment elements as single components could not be explored. Meta-analyses show that with exception of paradoxical intention, other cognitive techniques like thought stopping or cognitive restructuring were usually not used as single therapy techniques, but were combined with techniques for sleep-wake-rhythm structuring so that the effectiveness of these techniques alone is still not proven.

Concerning the intake of hypnotics, the data indicate that patients who took sleep medication during the cognitive-behavioral treatment profited significantly from the treatment, but not to the same extent as patients who abstained from sleep medication. Participation in the treatment program encouraged patients to taper off their sleep medication. One year after the end of therapy six out of twelve patients had discontinued the use of hypnotics, and, with exception of one patient, all these patients continued to abstain after discontinuation over the whole follow-up period. The exclusion of patients from this study, who could not or did not want to stop taking hypnotics, would have increased the internal validity of the study while at the same time external validity would have decreased. In the present study we wanted to evaluate the effectiveness of the treatment program in an unselected clinical population of patients with primary insomnia. Patients with chronic primary insomnia often take hypnotics for years and are not able or not willing to stop taking them on their own. Therefore they would need a therapy to discontinue using hypnotics before starting the treatment program. The results strengthen the idea of integrating behavioral approaches on a much broader scale in the treatment of insomnia, especially with regard to avoiding the risks of benzodiazepine treatment [13–15]. Therefore it would seem useful for physicians, when dealing with insomnia, to add non-pharmacological methods to pharmacological treatment to facilitate withdrawal from hypnotics.

Furthermore, every therapy for withdrawal from hypnotics should include non-pharmacological strategies to deal with insomnia.

Primary insomniacs also showed significant decreases of depression and anxiety, which might reflect their growing ability to apply the methods taught to them and their loss of sleep-related helplessness. This result is important because different studies [5, 7, 9, 13, 34] found that insomnia patients have a significantly higher risk of psychiatric disorders, especially depression, anxiety disorder, or alcoholism. It is possible, but not yet proven, that early and adequate treatment of primary insomnia prevents the development of other psychiatric disorders.

Despite the fact that there is evidence of effective non-pharmacological insomnia treatments, up to now, the majority of insomnia patients have not received such a treatment. Usually, the general practitioner is the first person in the health care system to hear about sleep problems, but insomnia patients normally do not receive systematic non-pharmacological treatment in general practice [2]. Such treatment is usually offered only at a very few specialized sleep disorder clinics. Because of the high insomnia prevalence rate it is unrealistic for all insomnia patients to be treated by a sleep expert. Further research should evaluate the effectiveness of non-pharmacological treatments, particularly self-help manuals, in general practice. Different studies have shown that cognitive-behavioral self-help manuals are effective treatments for insomnia [1, 20, 21, 30]. It might be possible to increase the insomnia treatment rate in general practice by reinforcing the self-management of insomnia patients. Thus general practitioners could supervise the self-management of patients and refer non-responding patients to sleep experts.

Although many insomnia patients have a comorbidity [29], most studies, until now, have analyzed the effectiveness of cognitive-behavioral insomnia treatments for patients with primary insomnia. Therefore, there is need for further research to evaluate the effectiveness of cognitive-behavioral treatments for insomnia patients with a mental and/or somatic comorbidity.

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