Neuropsychiatric Disease and Treatment

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ORIGINAL RESEARCH

CBT-I and HT-I group therapy for adults with insomnia in comparison to those with insomnia and comorbid depression – a pilot study

This article was published in the following Dove Press journal:
Neuropsychiatric Disease and Treatment

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Purpose: The aim of this study was to examine the effectiveness of a combined cognitive-behavioral therapy for insomnia (CBT-I) and hypnotherapy for insomnia (HT-I) program for insomnia patients with or without additional depression regarding depressive symptoms and various sleep parameters.

Patients and methods: A sample of 63 patients suffering from insomnia received a six-session sleep intervention, which combined cognitive-behavioral and hypnotherapeutical elements. Due to violating exclusion criteria, data of 37 patients were analyzed. Ten patients had insomnia comorbid with depression, whereas 27 patients had insomnia only. Sleep diaries were implemented to measure various sleep parameters, whereas depressive symptomatology was assessed with the anxiety and depression scale and Symptom-Checklist-90-R at baseline, before and after the intervention, as well as at 3-months follow-up.

Results: Depressive symptoms decreased from pre to post measurement and follow-up for patients with insomnia comorbid with depression, whereas scores of patients with only insomnia remained relatively on a low level. Both groups showed a significant increase of sleep efficiency and a significant decrease of the duration of wake after sleep onset. However, only patients with insomnia and depression revealed a significant reduction of sleep-onset latency and a higher level of regeneration. Nondepressive insomniacs, on the other hand, showed a significant increase of performance from post measurement to follow-up. For both groups, no change over time was found for number of wake after sleep onset, total sleep time, mood in the morning and evening.

Conclusion: Combining CBT-I and HT-I is effective in reducing depressive symptoms and improving sleep. Therefore, nonresponders to other forms of therapy, eg, pharmacological, interpersonal, or cognitive-behavioral therapy, might benefit from the combined CBT-I/HT-I intervention.

Keywords: CBT-I, hypnotherapy, insomnia, depression, adults, intervention

Introduction

Research showed that insomnia is a widespread disorder among individuals in industrial nations and is associated with various negative consequences, like health problems, increase of medication, job problems, accidents, and a decreased level of quality of life.[7] The lifetime prevalence of insomnia is estimated at 24.6%, and ~40% develop a more chronic form of insomnia.[6,8] While 2.3% of the people aged 20 suffer from insomnia, this number increased to 13.9% at age 35, and dropped again to 5.5% at age 41.[8]

Due to the close bidirectional relationship between insomnia and affective disorders, it is not surprising that in 40% of the cases comorbidities with insomnia occurred regularly.[13,22] Especially, an association between insomnia and affective disorders was

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Dovepress
http://dx.doi.org/10.2147/NDT.S164899

confirmed in various samples.[13] Up to 50% of patients with insomnia developed a major depressive disorder.[8,25] Even after controlling for other symptoms, the odds ratio for a major depression associated with insomnia is 2.1.[6] Hereby, no age effects could be detected.[2] Conclusively, insomnia is one of the most important risk factors for developing a depression and prevents remission.[12,16] Qualitative predictors for recurrence of depression are subjective sleep disturbances and anxiety, though higher levels of sleep disturbances occurred several weeks prior to recurrence.[34,36]

Vice versa, 90% of depressive patients reported complaints about their sleep quality and two-thirds of them will meet the criteria for insomnia.[16,25] Patients with affective disorders showed an increased level of sleep-onset latency (SOL) and percentage of rapid eye movement (REM) sleep, whereas total sleep time (TST), sleep efficiency (SE), slow wave sleep (SWS), and REM latency decreased.[41] Luik et al, however, found that longer REM sleep latency and higher REM density were associated with depressive symptoms.[28] Yet, after excluding patients using medication, only REM density remained related to depressive symptoms.[28]

Cognitive-Behavioral Therapy for Insomnia (CBT-I)

Cognitive-behavioral therapy became a standard treatment for primary insomnia, as well as insomnia comorbid with medical or psychiatric disorders.[37,38,40] Several studies showed that CBT-I influenced various subjective sleep parameters.[19,24,29,44] After CBT-I intervention, often SOL, wake after sleep onset (WASO), insomnia severity, and use of sleep medication declined, whereas SE, TST, and sleep quality increased. Effect sizes fluctuated from small (TST) to large effects (sleep quality) depending on the sleep variable and measurement method.[24] However, younger adults and patients with higher values of sleep self-efficacy showed the greatest improvement of SE at post measurement and follow-up.[27] Besides improving sleep after using a CBT-I intervention, Trauer et al reported improved sleepiness, self-efficacy, motoric vigilance, health status, beliefs and attitudes about sleep, and daytime functioning.[44] These improvements are independent of age, gender, type, or duration of complaints.[46]

Comparing regular CBT-I with other forms of therapy, eg, pharmacotherapy, relaxation training, and sleep hygiene education, showed that CBT-I was superior.[1,3,33,35,43] A meta-analysis by Okajima et al reported that CBT-I was more effective than a control group regarding SOL, WASO, time in bed (TIB), and SE.[35] Effect sizes ranged from small to medium for post measurement and from medium to large for follow-up. Differences between subjective and objective measurements were found for SOL and TST, which only improved in the sleep diaries. A level of good sleep at follow-up was achieved by 38% in the CBT-I group, whereas none achieved a good level of sleep in the control group.[47] Ashworth et al reported a rate of 67% for clinical remission of insomnia (vs 11% for self-help CBT-I), and 78% for those with additional comorbid depression (vs 17% for self-help CBT-I).[1] Furthermore, patients with insomnia and comorbid diseases reported the poorest sleep hygiene practice compared to a group of insomnia only patients, and to good sleepers, who reported the best sleep hygiene.[45] Comparing a CBT-I intervention with a sleep hygiene education revealed a reduction of SOL and WASO, as well as an improvement of SE for the CBT-I group.[14] The same effects account for a relaxation training, whereby sleep quality, early morning awakening, and TST were equal for both groups.[33] Nevertheless, effectiveness of a CBT-I intervention was mediated by the change of depression severity, as well as the content of the intervention rather than the comorbidity.[1,14] Finally, adding CBT-I to a treatment, as usual, leads to higher quality-adjusted life years without a significant increase in cost.[48]

Focusing on insomnia treatments for depressive patients seems to be highly relevant due to the high comorbidity of insomnia with depression and their negative influence on affective symptoms. Koffel et al reported reduced scores for depression from pre to post treatment and to follow-up.[24] In addition, normalized depressive symptoms were shown in 87.5% of treatment completers and normalized sleep patterns in 100% of patients with insomnia and depression.[42] Furthermore, SOL and WASO decreased, while TST, SE, and sleep quality increased with medium to large effect sizes.[5,42] On the other hand, higher scores for depression were associated with shorter sleep duration and shorter TIB.[18] Additionally, comorbidity reduced anxiety and depressive symptoms in patients of the CBT-I group, though this effect could not be found in the only cognitive therapy and the only behavioral therapy groups.[4]

The higher reduction of insomnia symptoms, when using an insomnia treatment instead of a treatment for depression, has proved that insomnia is not just a symptom of depression, but rather needs separate treatment.[5] Blom et al compared patients with insomnia and depression receiving either an insomnia (CBT-I) or depression intervention.[5] Patients from the CBT-I group showed a distinctive increase of SE and decrease of SOL and use of sleep medication with medium to large effects. Depressive symptoms were unaffected by the type of intervention.

Hypnotherapy for insomnia

Adding hypnotherapeutical elements to a CBT-I program might improve sleep parameters even more, because insomniacs often respond to this kind of therapy and relaxation.[32] A meta-analysis by Lam et al found that hypnotherapy increased various subjective sleep parameters from baseline to posttreatment.[26] In comparison to a waitlist-control group, participants receiving hypnotherapy showed decreased SOL. However, the number of nighttime awakenings did not differ between those two groups. Additionally, a hypnotic suggestion led to an extension of SWS in 81% of young females and 57% of older females compared with a control group.[9,10] This enhanced activity led to a better prefrontal cognitive functioning. However, this effect was significant only for high and middle suggestible participants.[10] Besides improving sleep, Holdevici et al investigated the effect of an intervention, which was based on hypnosis and relaxation, on depressive and anxiety symptoms.[21] Both men and women showed a significant reduction of symptoms for anxiety and depression immediately after the intervention and at a 3-months follow-up.[21] Thereby, hypnotherapy seems to be more effective than waitlist-control groups, cerebral electrotherapy, and pharmacological therapy in improving sleep and reducing depressive symptoms.[26]

Research question

The aim of this study was to examine the effectiveness of a combined cognitive-behavioral and hypnotherapeutical program for depressive and nondepressive patients. We assumed that patients suffering from insomnia without depression report less sleep problems after 3-months follow-up to a sleep intervention than patients suffering from insomnia with depression. Furthermore, we assumed that depressive symptoms will decrease for both groups, however, more predominantly in the group of depressive patients.

Patients and methods

Procedure and sample

This longitudinal pilot study included 74 participants between the age of 18 and 65 years suffering from insomnia: difficulties falling or staying asleep for at least 1 month. Patients were informed via our webpage of the outpatient clinic, newspaper, and information sheets in care practices. Prior to participation, information about diagnostic procedure, post-diagnostic measurement, and treatment content was given. Furthermore, they were informed that they could quit at any time without any negative consequences. After information about procedure and treatment, participants gave their written informed consent. In a further step, participants completed questionnaires regarding their demographic background, current life style, medication, sleep problems, as well as somatic and psychological impairments; selected participants were assessed 8 weeks prior to the treatment (baseline, t0). This assessment included a sleep log for 14 days and further questionnaires for mental health. To evaluate effects of waiting time, another assessment with the same instruments was conducted right before the 6-week intervention (t1) and after the intervention (t2), and at 3-months follow-up (t3). This six-session sleep intervention combined cognitive-behavioral (CBT-I), eg, psychoeducation, progressive muscle relaxation, stress management, and problem-solving, and hypnotherapeutical elements for insomnia (HT-I) in the form of different trances for each session. Each group session lasted for 120 minutes and consisted of four to seven patients and two to three trainers in a face-to-face setting. Exclusion criteria for data analysis were additional psychotherapy at the same time (n=11) leading to a participating sample of 63 adults. Furthermore, data sets with >30% of missing data on the post measurement (n=26) were excluded from analysis. Thus, 37 participants with a mean age of 43.86 (SD=13.08; 25 females, 12 males) were included for statistical analyses. Table 1 shows sample description at baseline. The study was approved by the ethical board of the University of Tuebingen and carried out in accordance with national law and the Helsinki Declaration of 1975 (in its current, revised form).

Table 1 Sample description at baseline

Variable	M (SD)	Min	Max
Age (years)	43.86 (13.08)	21	65
Duration of sleep problems (years)	9.47 (9.69)	0.5	41
Variable	**Number**		**Percent**
Gender			
Male	12		32.4
Female	25		67.6
Medication			
Yes	19		51.4
No	18		48.6
Type of medication			
Homeopathic	2		10.5
Antidepressant	7		36.8
Others	10		52.6
Depression			
Yes	10		27.0
No	27		73.0
Severity of depression			
Normal	18		48.6
Mild	9		24.3
Moderate	2		5.4
Severe	8		21.6

Note: n=37.
Abbreviation: M, mean.

Instruments

Sleep log

Sleep logs are an essential tool in the assessment of sleep problems and sleep disorders. Based on the sleep logs of the German association for sleep medicine, each sleep log assesses participants' bedtime, wake-up time, SOL, subjective regeneration, daytime functioning, performance, medication, and substance consumption over a period of 2 weeks.[11] Participants are asked to complete these sleep logs, but only the second week data will be used for statistical analysis to avoid bias of adaption and irritation in the first week. For statistical analysis, the following sleep parameters are used: SOL, number and duration of WASO, TST, SE, mood in the morning and evening, performance, and regeneration ("How restful/tired do you feel at the moment?"). Mood, performance, and regeneration are measured on a six-point Likert scale from 1 ("very good") to 6 ("very bad").

General Depression Scale (ADS)

This 20-item self-report instrument gathers information about impairment regarding depressive affect, somatic complaints, motoric inhibition, and negative thought patterns. Participants rate the frequency of their impairment on a four-point scale referring to the last week. It can be used for adults between the age of 14 and 80 years in clinical and nonclinical samples. Sum score above 23 is an indicator of a depressive disorder. With an internal consistency of α=0.89 and a split-half reliability of r=0.81. This instrument shows adequate psychometric properties.[20]

Symptom-Checklist-90-R (SCL-90-R)

Subjective impairment of somatic and psychological symptoms was assessed with the SCL-90-R, which gathers information retrospectively over a period of 1 week and consists of 90 items, which are rated on a four-point scale from "not at all" to "very intense". Adolescents' (from the age of 12) and adults' psychological distress is classified on nine subscales: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. Three global indices give information about participant's response: Global Severity Index, Positive Symptom Distress Index, and Positive Symptom Total. Internal consistencies for each subscale are between α=0.74 and α=0.97 for clinical and nonclinical samples. The instrument has demonstrated acceptable retest reliabilities between 0.69 and 0.92.[15]

Data analysis

Statistical analyses were carried out with IBM SPSS Statistics 23. The P-value was set to $P<0.05$. For testing the longitudinal effects of this intervention, analyses of variances with repeated measures were used. In case of significance of the Mauchly test of sphericity, data were corrected using the Greenhouse–Geisser correction. Post hoc analyses of significant main and interactions effects included Bonferroni corrected pairwise comparisons for normally distributed data and Wilcoxon tests for non-normally distributed data. The effect size Cohen's d will be reported for group comparisons and partial eta squared (η_p^2) for the analyses of variance. An effect size of d=0.2 and η_p^2=0.01 shows a small effect, d=0.5 and η_p^2=0.06 a medium effect, and d=0.8 and η_p^2=0.14 a large effect.

Results

Group comparisons at baseline

Analyzing the effect of an intervention in depressive and nondepressive patients on various variables required a comparison at baseline. Thus, t-tests for independent samples were conducted which showed that nondepressive patients reported a significant better mood in the morning and evening, felt more regenerated, more efficient, and less psychologically stressed prior to treatment (Table 2). Age, duration of sleep problems, SOL, WASO, TST, and SE did not differ significantly between the two groups. However, depressive patients exceeded the cutoff scores for SOL with 36 minutes (cutoff 30 minutes), duration of WASO of 38 minutes (cutoff 30 minutes), and TST with 357 minutes (cutoff 390 minutes) according to literature.

A chi-squared test showed no significant difference between depressive and nondepressive patients regarding gender, χ^2 (1, N=37)=1.93, P=0.16, and medication, χ^2 (1, N=37)=0.01, P=0.92. Patients who completed did not differ regarding age, sleep problems, or depression in comparison to those who did not complete (all $P>0.05$).

Sleep parameters

According to our hypothesis, it was assumed that various sleep parameters from the sleep diary would improve over time for both groups; however, these improvements will be less distinctive for insomnia patients with comorbid depression.

SOL of depressive patients decreased significantly from 36 minutes at baseline to 21 minutes at post measurement, which is under the clinical cutoff, but increased again to

Table 2 Group comparisons of depressive vs nondepressive participants at baseline

Variable	Depressive	Nondepressive	*t*(35)	*P*-value	Cohen's *d*
	M (SD)	M (SD)			
Age (years)	41.30 (13.45)	44.81 (13.07)	0.72	0.476	–
Duration of sleep problems (years)	9.75 (9.62)	9.36 (9.89)	0.11	0.916	–
Sleep-onset latency	36.33 (20.61)	27.91 (29.19)	0.84	0.409	–
Number of WASO	1.44 (1.20)	1.57 (1.14)	0.30	0.859	–
Duration of WASO	38.36 (53.16)	26.30 (25.93)	1.04	0.359	–
Total sleep time (minutes)	357.17 (81.52)	390.36 (58.36)	1.38	0.177	–
Sleep efficiency	75.09 (16.83)	78.13 (11.30)	0.63	0.530	–
Mood in the morning	3.27 (0.80)	2.70 (0.71)	**2.10***	**0.042***	**0.78**
Mood in the evening	3.23 (0.80)	2.53 (0.71)	**2.58***	**0.014***	**0.96**
Regeneration	3.78 (0.83)	3.06 (0.81)	**2.39***	**0.021***	**0.89**
Performance	3.38 (0.97)	2.64 (0.66)	**2.66***	**0.012***	**0.99**
Psychological distress (SCL-90-R)	69.70 (9.44)	56.76 (9.80)	**3.60****	**0.001****	**1.33**

Notes: *t* = statistics for group comparisons; *P* = probability, *$P<0.05$, **$P<0.01$; Cohen's *d* = effect size. The significant results are shown in bold.
Abbreviations: M, mean; WASO, wake after sleep onset.

30 minutes at follow-up, whereas no significant change occurred for nondepressive patients. No significant interaction was found, F (3, 105)=1.818, P=0.168. Also, the number of WASO showed no significant main or interaction effects for neither depressive nor nondepressive patients. On average, patients woke up one to two times per night. However, duration of WASO decreased from 33 minutes before treatment to 17 minutes at follow-up in nondepressive patients. For depressive patients, this main effect was marginally significant with 37 minutes at premeasurement to 17 minutes at follow-up, though no significant interaction occurred, F (3, 105)=1.112, P=0.336; however, the change is clinically significant. SE increased from 76% to 82% for nondepressive patients; similarly from 75% to 84% for depressive patients. Both groups did not differ regarding SE after treatment, F (3, 105)=1.315, P=0.275. Nevertheless, a significant increase of SE could be revealed only from premeasurement to follow-up in nondepressed, P=0.04.

Regeneration was significantly influenced by the presence of a depression, meaning that depressive patients improved their scores for regeneration from premeasurement to follow-up, P=0.008, whereas no change occurred for nondepressed insomnia patients. The interaction of time and group was marginally significant, F (3, 105)=2.281, P=0.084, η_p^2=0.061. Regarding performance, nondepressive patients showed a significant improvement over time, especially from post measurement to follow-up, P=0.036. However, changes over time for depressive patients, and the interaction were not significant, F (3, 105)=0.327, P=0.806.

Concerning TST, no significant main or interaction effects were found, F (3, 105)=1.312, P=0.275, mood in the morning, F (3, 105)=0.377, P=0.77, and in the evening, F (3, 105)=0.474, P=0.701, meaning that depressive and nondepressive patients remained stable over time and did not differ among each other. Table 3 shows the effects over time for depressive and nondepressive patients.

Depressiveness

Analyses of variance revealed a significant decrease of depressive symptoms measured with the anxiety and depression scale (ADS) for depressive, F (3, 27)=9.063, $P<0.001$, η_p^2=0.502, and nondepressive insomnia patients, F (3, 78)=5.208, P=0.002, η_p^2=0.167. The interaction of time and group was significant, F (3, 105)=6.183, P=0.001, η_p^2=0.15, which indicated a significant difference between depressive and nondepressive patients and their depressiveness over time. Remarkably, depressive patients reported an ADS mean score of 31.30 at baseline and 16.84 at follow-up, which is below the cutoff score for indicating a depressive disorder. Post hoc analysis showed a significant decrease from baseline to post measurement, P=0.028, and follow-up, P=0.003, for depressive patients. For nondepressive patients, only marginally significant decreases were revealed between baseline and follow-up, P=0.052, and premeasurement to post measurement, P=0.083, and follow-up, P=0.071 (Figure 1).

Severity of depression measured with the SCL-90-R showed a main effect of time for depressive, F (3, 27)=5.912, P=0.003, η_p^2=0.369, and nondepressive patients, F (3, 78)=2.428, P=0.072, η_p^2=0.085, as well as a significant interaction, F (3, 105)=6.183, P=0.001, η_p^2=0.15. Post hoc analyses revealed a significant decrease of depressive symptoms between baseline and follow-up, P=0.029, for depressive and

Table 3 Descriptives and analysis of variance for depressive and nondepressive patients

Parameter	Time	Depressive[a]					Nondepressive[b]				
		M	SD	*F* (3/78)	*P*-value	η_p^2	M	SD	*F* (3/78)	*P*-value	η_p^2
Sleep-onset latency	t0	36.33	6.52	3.789	**0.022***	0.296	27.91	5.62	0.044	0.945	0.002
	t1	27.03	2.40				27.88	5.69			
	t2	20.82	4.14				29.01	8.72			
	t3	30.29	4.99				27.69	8.15			
Number of WASO	t0	1.44	0.38	2.605	0.109	0.224	1.57	0.22	2.142	0.123	0.076
	t1	1.26	0.35				1.76	0.29			
	t2	0.93	0.37				1.48	0.30			
	t3	0.86	0.37				1.36	0.25			
Duration of WASO	t0	38.36	16.81	3.593	**0.061#**	0.285	26.30	5.00	4.221	**0.017***	0.140
	t1	37.78	16.52				33.13	5.13			
	t2	21.31	9.78				24.33	4.29			
	t3	17.36	8.29				17.42	3.52			
Total sleep time	t0	357.17	25.78	1.610	0.210	0.152	390.36	11.23	0.840	0.476	0.031
	t1	377.98	20.22				382.81	12.63			
	t2	383.59	19.05				391.94	12.55			
	t3	405.51	15.75				398.32	10.04			
Sleep efficiency	t0	75.09	5.32	3.987	**0.059#**	0.307	78.13	2.18	4.153	**0.017***	0.138
	t1	75.15	5.34				76.30	2.34			
	t2	83.01	3.41				80.02	2.49			
	t3	84.27	2.29				82.02	2.09			
Mood in the morning	t0	3.27	0.25	1.544	0.226	0.146	2.70	0.14	2.021	0.118	0.072
	t1	3.20	0.22				2.75	0.14			
	t2	3.07	0.25				2.67	0.14			
	t3	2.89	0.26				2.52	0.14			
Mood in the evening	t0	3.23	0.25	1.689	0.193	0.158	2.53	0.14	0.655	0.583	0.025
	t1	3.06	0.25				2.58	0.14			
	t2	3.07	0.18				2.51	0.16			
	t3	2.86	0.30				2.41	0.13			
Regeneration[c]	t0	3.78	0.26	5.043	**0.018***	0.375	3.06	0.16	0.542	0.655	0.020
	t1	3.60	0.23				3.12	0.15			
	t2	3.34	0.26				3.00	0.15			
	t3	3.10	0.24				2.97	0.15			
Performance[c]	t0	3.38	0.31	0.345	0.793	0.037	2.64	0.13	2.743	**0.049***	0.095
	t1	3.33	0.15				2.76	0.12			
	t2	3.29	0.27				2.72	0.13			
	t3	3.18	0.29				2.45	0.13			

Notes: t0 = Baseline, t1 = premeasurement, t2 = post measurement, t3 = 3-months follow-up; [a]n=10, [b]n=27; [c]Six-point Likert scale 1 =very good to 6 =very bad; #$P<0.10$, *$P<0.05$. The significant results are shown in bold.

Abbreviations: M, mean; SD, standard deviation; WASO, wake after sleep onset; η_p^2, partial squared eta.

a marginally significant decrease for nondepressive patients, $P=0.076$ (Figure 2).

Discussion

The main goal of this study was to examine the effects of a sleep intervention, which combines CBT-I and HT-I techniques, to treat insomnia patients with and without depression. Data of 37 patients were implemented for statistical analyses, in which 10 patients suffered additionally from depression. Sleep parameters, depression, and psychological distress were assessed at four measurement times: 8 weeks before intervention (baseline), right before (premeasurement) and after the intervention (post measurement), and 3 months later (follow-up).

Patients suffering from insomnia and comorbid depression responded differently to this intervention regarding several sleep parameters than insomnia patients without depressive symptoms. Whereas the latter group's SOL remained firm under the clinical cutoff of 30 minutes for all four measurement points, depressive patients showed a decrease in SOL from baseline to post measurement and a slightly elevated score at follow-up. These results are in line with previous studies such as Taylor et al and Trauer et al, who also reported a reduction in SOL.[42,44] Although both groups did not significantly differ at baseline, patients with insomnia and comorbid depression exceeded the clinical cutoff, supposedly due to their comorbidity, which might lead to more thoughts that are negative and rumination,

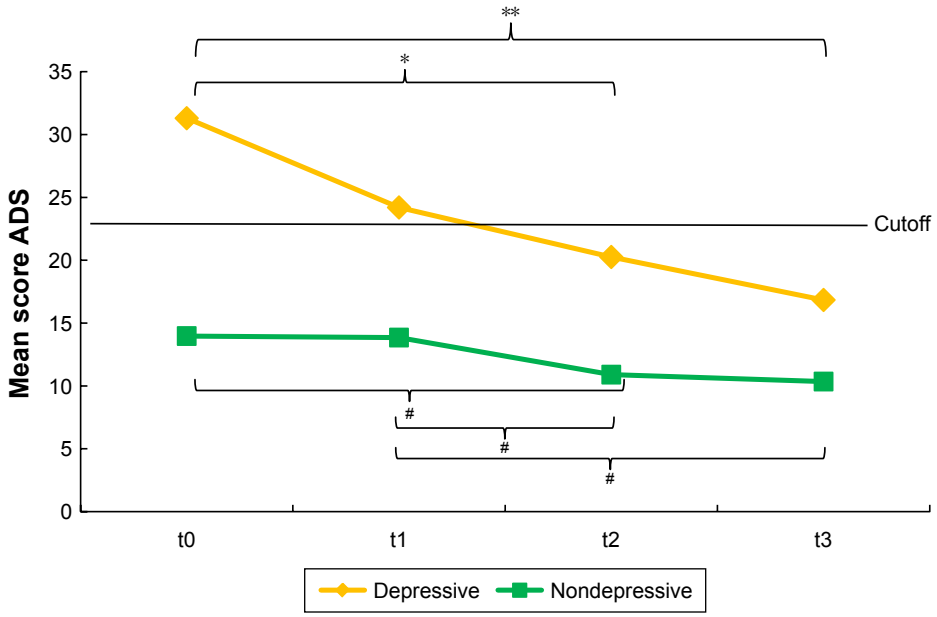

Figure 1 Mean scores of ADS scale for depressive and nondepressive patients over time.
Notes: t0 = Baseline, t1 = premeasurement, t2 = post measurement, t3 = 3-months follow-up; #$P<0.10$, *$P<0.05$ **$P<0.01$.
Abbreviation: ADS, anxiety and depression scale.

as well as a higher presleep arousal.[23] In the study by Hsu et al, an intervention contributed to the reduction of patients' presleep arousal and an improvement of sleep parameters.[23] Furthermore, anticipation and hope for therapy seemed to play a major role for these changes in depressive patients, due to a tremendous decline from baseline to pre and post measurement.[30] Nondepressed patients did not show these effects of anticipation; however, a higher level of psychological distress in the group of depressives might explain the clinical difference at baseline. Patients with insomnia and depression might be more pleased because of the opportunity of a therapy and the attention regarding their impairment.

For both groups, duration of WASO decreased and SE increased significantly, which is in line with results of Blom et al, Koffel et al, and Trauer et al;[5,24,44] SE raised up to >80% at posttreatment and follow-up. Even duration of WASO decreased by almost 50%, which then influences mood, SE, and regeneration. For depressive patients, regeneration enhanced significantly from baseline to follow-up, whereas this was not found for nondepressives. One explanation can

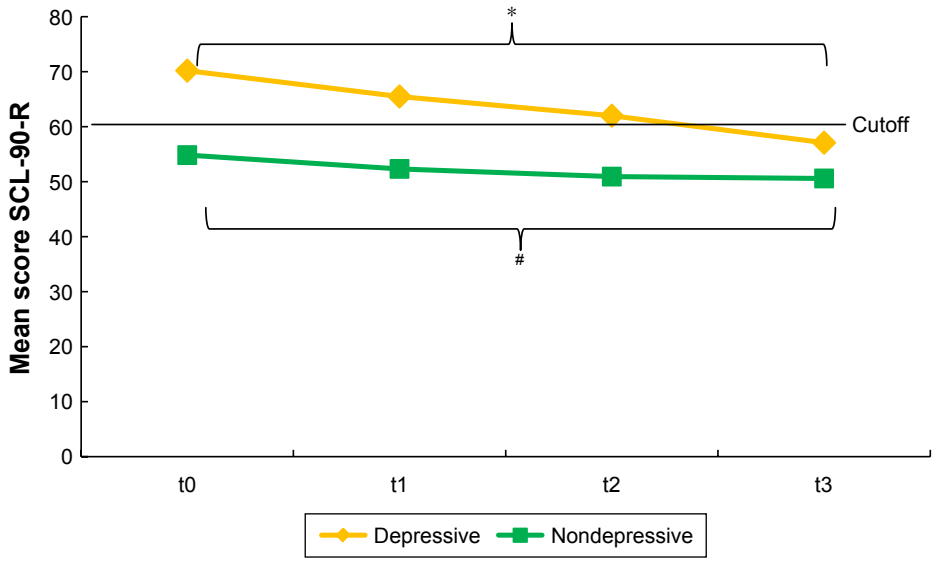

Figure 2 Mean scores of SCL-90-R subscale depression for depressive and nondepressive patients over time.
Notes: t0 = Baseline, t1 = premeasurement, t2 = post measurement, t3 = 3-months follow-up; #$P<0.10$, *$P<0.05$.

be the significant difference between these two groups at baseline and the comorbidity of insomnia and depression. Finally, mood in the morning and evening, as well as TST remained stable over time for both groups. Koffel et al found elevated TST after treatment, though, only small effects were reported.[24] For depressive patients, TST increased from 357 minutes at baseline to 405 minutes at follow-up. Due to the high level of variance, this difference was not significant.

Furthermore, depressive symptomatology over time differed for depressive patients in contrast to the nondepressive group. Depressive insomnia patients decreased their ADS score from baseline to post and follow-up significantly, whereas nondepressed patients showed no such changes. This result is in line with results from Bélanger et al, Holdevici, and Koffel et al.[4,21,24] Importantly, after CBT-I treatment, depressive patients scored below the cutoff score, indicating no clinical abnormality. This result is very important, meaning that such a sleep intervention reduces sleep parameters as well as depressive symptoms in patients suffering from both disorders.

Limitations

However, several limitations have to be listed. For example, the implementation of further insomnia-specific questionnaires as the Insomnia Severity Index would be helpful to evaluate more precisely insomnia-specific outcomes. Besides, more detailed dropout analysis might enhance the quality of the results. However, most participants reported at the end of the treatment an enhancement regarding their sleep quality and mental health, but not all filled in the full set of questionnaires and sleep logs for a further measurement. Therefore, future research should add either treatment costs or award systems for every completed data set.

Besides, future research could investigate the issue that different parts of an intervention have different effects on sleep behavior, especially focusing on patients suffering from insomnia and comorbidities.[31] On the one hand, components like stimulus control and sleep deprivation appeared to be more effective, whereas sleep hygiene alone showed no effect on sleep. Furthermore, the use of cognitive strategies rather than focusing on the physiological arousal seemed superior.[31] A meta-analysis by Friedrich and Schlarb about psychological sleep interventions for college students with sleep problems revealed that CBT elements were most effective, whereas relaxation, which includes hypnotherapy, showed a high level of variance regarding effect sizes.[17] Furthermore, the course of insomnia and depression might be mediated by various other variables, eg, presleep arousal, self-efficacy, and sleep-related beliefs.[39] Gałuszko-Węgielnik et al reported a decrease in psychophysiological arousal after CBT-I, but did not investigate the impact of arousal on treatment success regarding sleep parameters.[19] The level of stress vulnerability, though, did not influence this effect.[19] All these factors should be taken into account in further studies addressing insomnia patients with comorbid disorders or symptoms.

Conclusion

An intervention combining CBT-I and HT-I is effective in reducing depressive symptoms and improving various sleep parameters in adults with insomnia comorbid with depression, whereas patients with isolated insomnia showed these enhancements on less sleep parameters. Therefore, nonresponders to other forms of therapy, eg, pharmacological, interpersonal, or cognitive-behavioral therapy, could benefit from a combined therapy including hypnotherapy. The additional effect of hypnotherapy itself should be part of future research. Mediator variables influencing the course of insomnia should be considered.

Acknowledgments

We acknowledge support for the Article Processing Charge by the Deutsche Forschungsgemeinschaft and the Open Access Publication Fund of Bielefeld University.

Disclosure

The authors report no conflicts of interest in this work.

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