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Original Article

The link between social anxiety disorder, treatment outcome, and sleep difficulties among patients receiving cognitive behavioral group therapy

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ABSTRACT

Objective: The aim of our study was to examine the association between sleep disturbances and social anxiety disorder (SAD). Another aim was to explore the impact of cognitive behavioral group therapy (CBGT) for SAD on co-occurring sleep difficulties.
Methods: Data were obtained retrospectively from patient files receiving CBGT for SAD. The sample included 63 patients with SAD (mean age, 30.42 years [standard deviation, 6.92 years]). There were 41 men and 22 women, of whom 41 participants completed the treatment protocol. Before treatment onset participants completed the Liebowitz Social Anxiety Scale (LSAS), the Beck Depression Inventory (BDI), the Pittsburgh Sleep Quality Index, and several sociodemographic questions. On completion of the treatment protocol, the same measures were completed, with the addition of the Sheehan Disabilities Scale (SDS).
Results: The results of our study suggest that: (1) subjective insomnia is associated with SAD severity even after controlling for depression severity and additional variables; (2) participants with SAD with co-occurring clinical levels of subjective insomnia present a more severe clinical picture both at treatment onset and termination; and (3) although CBGT lead to reduction in SAD and depression symptoms severity, it had no significant impact on co-occurring sleep difficulties.
Conclusions: Sleep difficulties predict SAD severity regardless of depressive symptoms and may be linked to a more severe clinical picture. Clinicians should be aware of these sleep difficulties co-occurring with SAD and consider implementing specific sleep interventions. Future studies should incorporate larger samples sizes from clinical populations outside of Israel.

1. Introduction

Social anxiety disorder (SAD) is a common psychiatric disorder, with a 7–13% estimated lifetime prevalence rate in western countries [1]. The results of a previous study [2] assessing the prevalence of SAD in an Israeli sample of 850 participants corroborated those from other studies in western countries, both regarding the high prevalence of SAD symptoms and its demographic and clinical correlates. The comorbidity of SAD and other anxiety and mood disorders is high [3,4] and has significant adverse effects on functioning and well-being [5]. The disorder usually starts in childhood or adolescence and if untreated can be a chronic lifelong disorder with a low spontaneous remission course [6].

Sleep disturbances (e.g., extended sleep onset latency, frequent night awakenings, reduced sleep efficiency) are also a common clinical concern. In the general population, more than 33% of individuals experience sleep difficulties, and between 8% and 27% experience severe sleep problems [7–9]. Sleep disturbances may have considerable negative impact on health, general and emotional functioning, and quality of life [10,11]. In addition, it has been suggested that sleep disturbance can be a risk factor for the development and maintenance of mood and anxiety disorders (AD) [12–14]. Moreover, up to 50% of individuals who report sleep problems have comorbid psychiatric disorders [12,15].

Sleep difficulties are common in patients with AD [16–19]. Previous studies examined both reports of sleep problems in those with AD, and anxiety symptoms in those with sleep disturbances [19]. One study revealed that sleep difficulties increased the likelihood to have clinically significant anxiety by 17 times [20]. In a more recent study [19] examining the relationship between

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self-reported sleep disturbance and AD, 74% of patients with AD reported that they had some kind of sleep disturbance (e.g., difficulty falling asleep, waking during the night, restless sleep).

Sleep disturbances are listed as specific diagnostic criteria in several AD [21]. For example, nightmares and insomnia symptoms (e.g., difficulty in falling or resuming sleep) are included in the posttraumatic stress disorder (PTSD) diagnostic criteria [19,21]. Between 20% and 70% of patients diagnosed with PTSD report symptoms of frequent nightmares [17,22], and approximately 70% report on difficulty falling or staying asleep [17]. Additionally 60–70% of individuals diagnosed with generalized AD (GAD) reported sleep disturbances [18,23], which are also listed as part of GAD *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5) diagnostic criteria [21]. Although not listed in the DSM-5 diagnostic criteria, sleep disturbances are also associated with other AD, such as obsessive–compulsive disorder, panic disorder with or without agoraphobia, and specific phobia [24–27].

In this context, the findings of previous studies assessing the links between sleep disturbances and SAD have been limited and inconsistent [19,28–31]. One study revealed no differences in polysomnography data when patients with SAD were compared to healthy controls [31]. However, when sleep quality and sleep disturbances were assessed in individuals with SAD compared to controls using the Pittsburgh Sleep Quality Index (PSQI), the results demonstrated that the majority of individuals with SAD reported impairments in sleep quality, longer latency to fall asleep, more nocturnal disturbance, and more daytime dysfunction than controls [28]. In another study [30], individuals with SAD frequently reported insomnia when directly questioned about sleep problems. In a more recent study [19], SAD was not associated with self-reported sleep disturbances.

Although the efficacy of cognitive behavioral therapy (CBT) for AD has been widely documented, few of these studies reported CBT effects on co-occurring sleep problems [16]. Moreover, despite the wide body of evidence linking various AD and sleep disturbances, many CBT protocols for AD do not include strategies specifically targeting sleep disturbances. In addition, although several researchers have suggested that successful treatment for AD has a positive impact on sleep disturbances [30], this notion is not well supported [16].

A recent meta-analysis indicated a moderate effect of anxiety treatment on co-occurring sleep difficulties, and it was suggested that "the current state of knowledge does not permit definitive conclusions and future research is needed" [16]. Along these lines, it has been suggested that sleep difficulties might produce or exacerbate anxiety symptoms. Previous studies linked sleep deprivation and insomnia with heightened levels of anxiety in healthy individuals [32,33]. Among patients with panic disorder, sleep deprivation and insomnia symptoms were associated with heightened anxious reactivity [34] and panic attacks [35] the following day. Moreover, sleep problems often continue without improvement after successful treatment for anxiety [36,37]. Thus it might be speculated that the impact of CBT on anxiety symptoms may not be optimal without specifically targeting sleep difficulties.

2. Methods

2.1. Participants

The aims of our study were to examine the associations between sleep disturbances, and SAD and to explore the impact of cognitive behavioral group therapy (CBGT) for SAD on co-occurring sleep difficulties. Based on previous findings, we hypothesized that sleep disturbances would be linked to higher levels of SAD both at pre- and post-treatment time points, and that minimal or no improvement of sleep disturbances would concurrently occur with reduction in anxiety level following CBGT for SAD. To the best of our knowledge, our study is the first to assess the link between sleep disturbances and SAD in the context of CBGT. Data were obtained retrospectively from patient files receiving CBGT for SAD. These data were routinely gathered as part of clinical process. The participants received treatment for SAD at an AD clinic of a regional mental health center in the greater Tel-Aviv area. The outpatient clinic provides treatment services free of charge. Participants were either self-referred or referred by a medical doctor or mental health professional. Inclusion criteria for study participation were (1) a current diagnosis of SAD according to the DSM fourth edition; (2) a minimum of a 1-year duration of SAD; (3) a primary diagnosis of SAD, that is, in cases with comorbidity SAD was deemed as the most distressing and clinically significant condition among the comorbid disorders; (4) a stable pharmacotherapy, that is, participants receiving a pharmacologic treatment who were taking a stable medication for at least 3 months before the beginning of CBGT; and (5) age between 18 and 60 years. Exclusion criteria were past or present diagnosis of psychotic state and schizophrenia, another psychotherapeutic treatment during CBGT, and change in medication status during the CBGT.

At the onset of treatment, the sample included 63 participants (mean age, 30.42 years [standard deviation, 6.92 years]). There were 41 men and 22 women who were diagnosed with SAD. Diagnoses were determined by the Mini-International Neuropsychiatric Interview and were conducted by doctorate-level clinical psychologists or experienced graduated psychology students trained in their administration. Out of the 63 participants, 41 completed the treatment, 18 participants dropped out in the process, and four additional participants were excluded from the study because they did not meet the criteria above (Fig. 1). Dropouts were defined as premature cessation of participating in CBGT (i.e., nonparticipation in the last session), or absence from six or more CBGT sessions.

2.2. Measures

2.2.1. Liebowitz Social Anxiety Scale

Social Anxiety severity was assessed using the self-report version of the Liebowitz Social Anxiety Scale (LSAS) [38], which has been shown to have high internal consistency; strong convergent, and discriminant validity; and high test–retest reliability [39,40]. The LSAS comprises 24 social situations that are each rated for level of fear (0 = none to 3 = severe) and avoidance (0 = none to 3 = usually) for the past week. The Hebrew version of the self-report LSAS has been validated in previous research [41].

2.2.2. Beck Depression Inventory

The Beck Depression Inventory (BDI) is a 21-item self-report measure that assesses depressive symptoms in the past 7 days [42,43]. It covers cognitive (e.g., thoughts about past failure), emotional (e.g., sadness), and somatic/vegetative (e.g., tiredness or fatigue) symptoms. Each item is scored from 0 to 3 with a maximum score of 63. The BDI has shown high validity and reliability scores and high internal consistency ($\alpha = 0.81–0.86$) [42].

2.2.3. Sheehan Disabilities Scale

The Sheehan Disabilities Scale (SDS) assesses the degree of impairment in work, social, and family areas [44]. The SDS evaluates the degree of impairment in work, social, and family areas on a 10-point Likert scale. The psychometric properties of the SDS are high [31] and previous studies showed that it has strong correlations with symptoms of social anxiety, depressive symptoms, and quality of life among individuals with SAD [45].

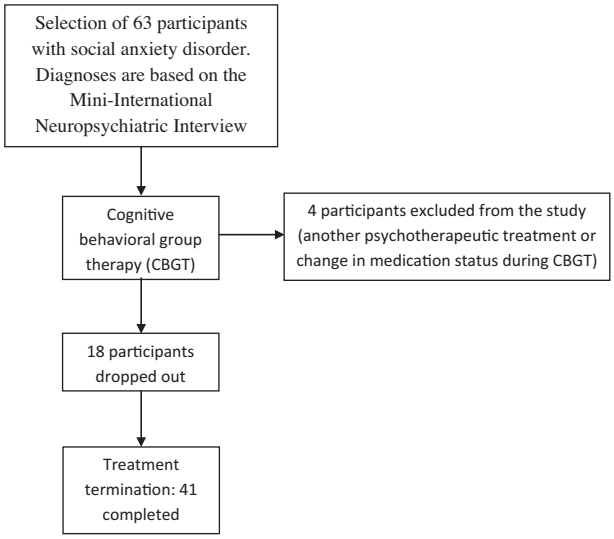

Fig. 1. Flow of participants through each stage of the study.

2.2.4. PSQI

The PSQI is a common self-report measure used in sleep studies to define subjective insomnia [46]. The PSQI consists of 19 questions that evaluate a wide variety of factors related to subjective perception of sleep quality over the past month. The 19 sections contain seven components related to clinical aspects of sleep quality: sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. Each index can be evaluated on a scale of 0–3. The combined seven indices form a total sleep quality value ranging from 0 to 21. A cutoff value of five is used to define subjective insomnia [46,47]. We used a Hebrew validated version of the PSQI [47].

2.3. Procedure

The procedures in our study were approved by the institutional review board of the Geha Mental Health Center, Petah-Tikva, Israel. Patients were interviewed using the Mini-International Neuropsychiatric Interview after the initial contact with the outpatient clinic [48]. Before the treatment onset, patients completed the LSAS, BDI, PSQI, and several sociodemographic questions. On completion of the treatment protocol, the same measures were filled with the addition of the SDS. The participants of our study were included in four treatment groups. The number of participants in each group ranged between 10 and 19. CBGT was conducted by a doctorate-level clinical psychologist and a cotherapist (a psychiatry or psychology resident). The treatment procedure was based on the protocols of Heimberg et al. [49] and was theoretically based on the writings of Clark and Wells [50]. There were 18 weekly sessions of 1.5 h in duration, and in general the protocol included psychoeducation about SAD, exposure to feared social situations, reduction of safety behaviors, cognitive restructuring, instruction on external focus of attention, and social skills training (for a detailed description of the CBGT protocol see [65]).

2.4. Data analysis

Data analysis included the following components, a linear regression to predict LSAS score at the beginning of treatment, between-group comparisons of participants with clinical PSQI score compared to those with nonclinical score, and ANOVA to assess intervention effects. Dependent variables were LSAS, BDI, and PSQI scores. The independent variable was time effect (from treatment onset to termination).

3. Results

3.1. LSAS score prediction at the beginning of treatment

A linear regression analysis was performed to predict LSAS score at the beginning of treatment. We mainly sought to assess the unique contribution of PSQI score to LSAS score before treatment initiation. Table 1 shows the results of the linear regression analyses predicting LSAS score at the beginning of treatment controlling for age, gender, PSQI total score, number of comorbid diagnoses, BDI score, and medication status (treatment or no treatment). Age, PSQI, and BDI scores were found to be significant predictors of the LSAS score. Specifically, age was negatively associated with LSAS score such that younger age predicted higher LSAS score. However, both BDI and PSQI scores were positively associated with LSAS score such that higher BDI and PSQI scores predicted higher LSAS score.

3.2. Clinical vs nonclinical PSQI scores

To assess the impact of having a PSQI clinical score on the overall clinical picture, we conducted between-group comparisons and compared the participants with a clinical PSQI score to those with a nonclinical PSQI score. A PSQI score of >5 was considered a clinical score [46]. In our sample, 30% of the participants had a clinical score of PSQI >5 (2 participants scored 6, three participants scored 7, one participant scored 9, one participant scored 10, one participant scored 13, and 3 participants scored 14).

The clinical PSQI group of participants had higher BDI and LSAS score at treatment onset and termination. The clinical PSQI group also had a higher number of comorbid diagnoses and a higher score on the SDS work subscale on treatment termination. No differences were found in age, years of education, medication status (treated or untreated), LSAS delta score (LSAS score at the beginning if treatment compared to termination), SDS disabilities social and family subscales score, and gender between the clinical PSQI group and the nonclinical group (Table 2).

3.3. Intervention effects on LSAS, PSQI, and BDI scores

Significant correlations were found between BDI score, LSAS score, and PSQI score at the beginning of treatment (BDI and LSAS: $r = 0.61$, $P < 0.0001$; BDI and PSQI: $r = 0.64$, $P < .0001$) and on termination (BDI and LSAS: $r = 0.66$, $P < 0.0001$; BDI and PSQI: $r = 0.74$, $P < 0.0001$). In addition, significant correlations were found between age and LSAS score ($r = -0.35$, $P < 0.01$) and BDI score at

Table 1
Regression analyses: predicting the Liebowitz Social Anxiety Scale score from gender, age, Pittsburgh Sleep Quality Index score, number of comorbid diagnoses, Beck Depression Inventory score, and medication status (medicated/unmedicated).

Predictors	β (SE)	β	t test
Model ($R^2 = 0.67$)			
Gender	−5.98 (7.04)	0.11	−0.85
Age	−1.42 (0.64)	−0.28	−2.20*
PSQI score	3.28 (1.21)	0.47	2.72*
No. of comorbid diagnoses	1.51 (2.84)	0.08	0.53
BDI score	1.06 (0.49)	0.34	2.15*
Medication status (medicated/unmedicated)	−2.94 (7.62)	−0.05	−0.37

Abbreviations: SE, standard error; PSQI, Pittsburgh Sleep Quality Index; No., number; BDI, Beck Depression Inventory.
* $P < .05$.

Table 2
Comparison between participants with clinical vs nonclinical Pittsburgh Sleep Quality Index score.

	Clinical (n = 11)	Nonclinical (n = 22)	t/χ^2 statistic
Age (M/SD)	29.64 (4.93)	29.36 (5.51)	0.14
Men (n/%)	7 (63.6%)	13 (59.1%)	0.06
Medication status: medicated (n/%)	7 (70%)	8 (36.4%)	3.12
LSAS score at onset	90.73 (20)	56.05 (22.37)	−4.34***
LSAS score at termination	73 (19.72)	42.56 (21.46)	−3.25**
LSAS delta score	16.29 (20.06)	12.33 (21.95)	−0.41
BDI score at onset	19.83 (7.65)	9.23 (7.04)	−3.96***
BDI score at termination	18.43 (8.70)	7.28 (5.66)	−3.8**
No. of comorbid diagnoses	2.27 (1.42)	1.36 (1.29)	−1.84*
SDS: work subscale	7 (1.41)	4.72 (3.27)	−1.76*
SDS: social subscale	6.14 (2.67)	6.39 (3.26)	0.18
SDS: family subscale	3.86 (3.72)	1.67 (1.88)	−1.49
Education (years)	14.45 (2.66)	14.36 (2.04)	−0.11

Abbreviations: M/SD, mean/standard deviation; LSAS, Liebowitz Social Anxiety Scale, BDI, Beck Depression Inventory, SDS, Sheehan Disabilities Scale.
* $P < .05$.
** $P < .01$.
*** $P < .0001$.

the beginning of treatment ($r = -0.23$, $P < 0.05$). However, no gender differences were found on the BDI, LSAS, or PSQI score.

To assess the intervention effects, analysis of variance (ANOVA) was performed. Dependent variables were LSAS, BDI, and PSQI scores. The independent variable was time effect from treatment onset to termination.

3.4. LSAS score

Time effect from treatment onset to termination relates to the intervention effect. When gender was entered as between-participants factor, ANOVA revealed a significant time effect ($F[1,43] = 26.15$; $P < .0001$; Table 3) and a significant interaction of time by gender effect ($F[1,43] = 4.47$; $P < 0.05$) on LSAS score, showing a higher reduction in LSAS score following the CBGT in men (baseline: $\chi^2 = 273.15$ [29.17]; termination: $\chi^2 = 48.11$ [22.61]) compared to women (baseline: $\chi^2 = 63.33$ [23.47]; termination: $\chi^2 = 52.94$ [20.18]). No main effect for gender was found ($F[1,43] = 0.14$; nonsignificant [n.s]). However, when analysis of covariance was performed controlling for BDI score at the beginning of treatment, no significant effect was found for time ($F[1,41] = 0.82$; n.s), gender ($F[1,41] = 0.01$; n.s), or time by gender on LSAS score ($F[1,41] = 3.98$; n.s).

3.5. BDI and PSQI scores

ANOVA revealed a significant effect for time on BDI score ($F[1,42] = 8.96$; $P = 0.005$), showing that the BDI score at treatment onset was higher than on termination. No gender ($F[1,42] = 0.41$; n.s) or time by gender ($F[1,42] = 0.14$; n.s) effects on BDI score were found (Table 3). However, ANOVA revealed no significant time ($F[1,23] = 0.02$; n.s), gender ($F[1,23] = 1.2$; n.s), or time by gender ($F[1,23] = 0.42$; n.s) effects on PSQI score (Table 3). When BDI score was entered as a covariate, there also was no significant time ($F[1,22] = 0.03$; n.s), gender ($F[1,22] = 0.7$; n.s), or time by gender ($F[1,22] = 0.34$; n.s) effects on PSQI score.

Table 3
Main effects for session time on the Liebowitz Social Anxiety Scale, Beck Depression Inventory, and Pittsburgh Sleep Quality Index measures (mean ± standard deviation).

Outcome measures	Baseline	Termination	F
LSAS[a]	69.22 ± 27.20	50.04 ± 21.57	26.15***
BDI	12.55 ± 8.43	9.64 ± 7.68	8.96**
PSQI	5.16 ± 3.86	5 ± 3.66	0.42

Abbreviations: LSAS, Liebowitz Social Anxiety Scale, BDI, Beck Depression Inventory, PSQI, Pittsburgh Sleep Quality Index.
[a] When BDI score was entered as a covariate, time effect was nonsignificant.
* $P < .05$.
** $P < .01$.
*** $P < .0001$.

4. Discussion

The aims of our study were to examine the association between sleep disturbances and SAD, and to explore the impact of CBGT for SAD on co-occurring sleep difficulties. The results of our study suggest that subjective insomnia was associated with SAD even after controlling for depression severity and additional variables (e.g., age, gender, number of comorbid diagnoses, medication status). Our results indicated that participants with higher subjective insomnia presented with an elevated SAD level, which adds to previous though limited studies suggesting that there is a link between SAD and sleep difficulties [28,30,51]. Additionally, our results demonstrated that depression severity was positively associated with SAD. This finding is not surprising given that previous studies demonstrated that depression is highly comorbid with AD, including SAD [52–54]. Although the literature consistently shows that depression is linked with increased rates of sleep disturbances [12,55], the majority of previous studies on sleep disturbance and AD have not controlled for co-occurring depression [19]. Thus one possible explanation for the link between sleep problems and AD may be accounted by comorbid depressive symptomology. However, we controlled for co-occurring depressive symptoms to remove any potential effect of depression in our analysis. In this context, one report [51] found that several AD including SAD were associated with poorer sleep and that these associations remained significant after controlling for mood disorders with the exception of obsessive–compulsive disorder.

Recent opinions suggest that anxiety, mood, and sleep difficulties are linked and might even be symptoms of one underlying pathology; however, the exact nature of the relationship remains unclear [13,19,56,57]. Given the high comorbidity rates between AD, sleep, and mood difficulties, it might be speculated that there are common factors that put individuals at a higher risk for developing sleep, mood, and AD, such as genetic (potentially genes involved in the serotonin system), environmental factors, and cognitive biases [13]. This assumption should be further explored.

Another finding of our study was that individuals with SAD and co-occurring clinical levels of subjective insomnia presented with a more severe clinical picture, both at treatment onset and

termination. Specifically, the clinical PSQI group of participants had higher BDI and LSAS score at treatment onset and termination. This group also had higher a number of comorbid diagnoses and a higher score on the SDS work subscale at treatment termination. This result adds to previous studies demonstrating the association of impaired sleep with impaired general and emotional functioning, higher rate of psychiatric disorders, higher use of health care services, as well as the association with a higher risk for morbidity and mortality [10,58–61]. In patients with mood disorders and AD, impaired sleep may be associated with more severe clinical symptoms and may lead to the exacerbation of present clinical symptoms. For example, previous studies showed that impaired sleep quality was associated with more severe posttraumatic symptoms among individuals with PTSD [36] and predicted the development of PTSD after a traumatic event [62]. In depressed patients, insomnia is associated with greater intensity of suicidal ideation, independently of core symptoms of depression such as depressed mood and anhedonia [63]. Other studies suggested that various sleep problems can elevate the risk for daytime anxiety and panic symptoms, even in nonclinical individuals [32–35]. Another study indicated that nondepressed individuals with insomnia compared to individuals with no sleep difficulties were at increased risk for depression [64].

Our results demonstrated that, although CBGT targeted SAD, participants exhibited a significant simultaneous decrease both in SAD level and in depressive symptoms at treatment termination. These results are in line with previous studies demonstrating the effectiveness of CBGT for SAD [65,66]. However, in accordance with our hypothesis, there was no reduction in co-occurring sleep difficulties following CBGT. One rationale for not addressing sleep management in CBT for AD may be the assumption that intervention for AD reduces concomitant sleep disturbances [30]. However, sleep problems often continue without reduction after successful treatment for anxiety [36,37]. Although some level of improvement in sleep can be expected following treatment for several AD (e.g., GAD, PTSD, PD) [16], there currently are no data concerning the impact of CBT for SAD on co-occurring sleep difficulties. To the best of our knowledge, our study is the first to address this issue.

Thus our results provide more support to the notion that clinicians working with anxious patients exhibiting both AD and sleep difficulties should not assume that sleep difficulties will improve in parallel to decrease in anxiety following AD treatment [16]. Therefore, it is suggested to consider adding specific interventions targeting sleep in the treatment of AD [67]. There are several other findings that lend more support to this suggestion. First, there is growing evidence suggesting that interventions that directly target sleep may also have a positive impact on anxiety and mood symptoms in addition to improvement in sleep [68–72]. Second, sleep problems constitute a risk factor for relapse [73]. Third, if sleep difficulties are not addressed in the early stage of anxiety treatment, they might develop into a significant sleep disorder [67]. Fourth, it also has been demonstrated that deprivation of rapid eye movement sleep following a learning task directly interferes with the extinction of both cued [74] and contextually conditioned fears [75] in animals. These data have important clinical significance in suggesting that the effects of comorbid sleep disturbance may interfere with the implementation and effectiveness of various interventions for anxiety and mood disturbances [76].

Given the high prevalence of co-occurring anxiety and sleep problems and findings highlighting the bidirectional relationship between sleep and emotional functioning [77–80], we propose that a vicious cycle is formulated that perpetuates both anxiety and sleep symptoms. From one perspective, cognitive (worry) and physiologic arousal are key features of AD, and can interfere and impair normal sleep patterns [81]. On the other hand, various sleep problems (e.g., sleep loss, sleep deprivation) can magnify the manifestation of daytime anxiety symptoms [34].

Future studies addressing treatments for co-occurring sleep and anxiety should compare interventions targeting overlapping cognitive, somatic, and behavioral disrupted elements of sleep and AD (e.g., heightened arousal, dysfunctional beliefs, cognitive errors, catastrophic thinking, safety behaviors, avoidance) [67] by adding specific sleep interventions (e.g., stimulus control sleep hygiene) to standard CBT treatment for AD on sleep, anxiety symptoms, and general daytime functioning.

Several limitations of our study should be addressed. First, the absence of a control group requires the impact of CBGT on our study outcome measures to be addressed with caution. Future prospective studies should incorporate a control group to strengthen the understanding of intervention impact and possible therapeutic mechanisms. Second, our study only focused on the impact of CBGT for SAD on co-occurring sleep difficulties. Therefore, our findings should not be generalized to other AD without proper research. Third, only a minority of participants ($n = 11$) had a clinical subjective insomnia score. However, the mean baseline score of the PSQI for all the participants did in fact exceed the clinical cutoff score of 5, but only by a small degree (mean, 5.16). Therefore, another possible explanation for the lack of significant change in the PSQI score following treatment is based on this relatively low baseline PSQI score and due to insufficient statistical power. Thus future studies should incorporate larger samples sizes and clinical populations outside of Israel to further validate and generalize our study results and conclusions.

Finally, assessment of sleep disturbances in our study was based on self-report measures. Future studies should incorporate objective sleep measure (e.g., actigraphy), which can provide a more objective picture of sleep patterns. Sleep parameters monitored by objective and subjective measures (e.g., polysomnography assessments, daily sleep diaries, actigraphic recordings) could be included without significant difficulty as an outcome variable in future studies of CBT for AD. The timing of the intervention and the method of delivery (e.g., group vs individualized therapy) should also be assessed in future studies.

5. Conclusion

Sleep difficulties predict SAD severity regardless of depressive symptoms. It seems that individuals with both SAD and insomnia are at higher risk for a severe clinical picture. Clinicians should be aware of these sleep difficulties co-occurring with SAD and consider implementing specific interventions to reduce their impact. Based on findings from our and other studies, it seems that the effect of CBT targeting anxiety symptoms is limited regarding its impact on sleep difficulties, and thus may pose an obstacle for optimal therapeutic gains and further reduction of symptoms. There is a need to further explore the optimal intervention for co-occurring anxiety and sleep difficulties.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.01.012.

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