



EMPIRICALLY GROUNDED CLINICAL INTERVENTIONS

Treating co-morbid insomnia and social anxiety disorder with sequential CBT protocols: a single-case experimental study

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Abstract

Background: Although insomnia disorder and social anxiety disorder are among the most prevalent psychiatric disorders, no studies have yet evaluated the use of sequential evidence-based treatment protocols in the population with co-morbid social anxiety disorder and insomnia disorder.

Aims: This study aimed to investigate the effects of sequential treatments on co-morbid insomnia disorder and social anxiety disorder. As depression is a common co-morbid syndrome for both insomnia and social anxiety, a secondary aim was to examine depressive symptoms.

Method: A single-case repeated crossover AB design was used. Ten participants between 18 and 59 years of age with co-morbid *DSM-5* diagnoses of insomnia disorder and social anxiety disorder received sequential treatments with cognitive behavioural therapy (CBT). Seven participants completed the treatment course. The primary outcomes were symptoms of insomnia and social anxiety, and the secondary outcome was symptoms of depression.

Results: The effects of CBT on people with co-morbid social anxiety disorder and insomnia disorder were mixed. The majority of participants improved their sleep quality and lessened symptoms of social anxiety and depression. However, participants differed in their degree of improvement concerning all three disorders.

Conclusions: Sequential CBT treatments are potentially effective at decreasing symptoms of social anxiety and insomnia for people with co-morbid social anxiety disorder and insomnia disorder. The variation in outcome across participants makes firm conclusions about the treatment efficacy difficult to draw.

Keywords: cognitive behavioural therapy; co-morbidity; depression; insomnia; single case experimental design; social anxiety

Introduction

Co-morbidity of psychiatric disorders, as defined by *the Diagnostic and Statistical Manual of Mental Disorders (DSM, 5th edition; American Psychiatric Association, 2013)* are very common (Kessler *et al.*, 2005; Roca *et al.*, 2009). Nevertheless, knowledge on the most effective treatments for certain combinations of disorders is lacking, as reflected by the scarcity of publications. Co-morbid insomnia disorder and social anxiety disorder is one such

understudied psychiatric combination, with only one identified case study ($n=1$) investigating treatments for this co-morbidity (Tang, 2010).

Insomnia is characterized by sleep onset latency, undesired periods of wakefulness during the night, or early awakenings combined with impaired functioning during daytime. Approximately 25–30% of the general population reports at least one criterion for insomnia disorder, while 6–10.5% meet the diagnostic criteria (American Psychiatric Association, 2013; Mallon *et al.*, 2014; Ohayon, 2002).

Social anxiety disorder is one of the most common anxiety disorders across western populations, with a lifetime prevalence of approximately 13% (Bandelow and Michaelis, 2015). Social anxiety disorder is characterized by persistent fear of underperforming in social situations, being negatively judged by others, and impairment in everyday functioning through avoidance (American Psychiatric Association, 2013). Insomnia has historically been considered symptomatic of other psychiatric disorders, as reflected by earlier versions of the *DSM*. However, more recent research indicates a complex and bi-directional relationship between insomnia and anxiety disorders (Blake *et al.*, 2018; Krystal, 2012).

First, the presence of either insomnia or an anxiety disorder seems to predict an increased incidence of the other disorder (LeBlanc *et al.*, 2009; Morphy *et al.*, 2007; Neckelmann *et al.*, 2007). People suffering from insomnia disorder are twice as likely to develop anxiety disorders relative to healthy controls (Krystal, 2012) and disturbed sleep has been proposed to increase fear responses and negatively affect emotion regulation, thereby worsening symptoms of anxiety (Babson *et al.*, 2009; Walker and van der Helm, 2009). Second, research on the relationship between insomnia disorder and social anxiety disorder indicates that the presence of social anxiety predicts greater insomnia disorder severity (Buckner *et al.*, 2008). However, a review found no significant differences in sleep parameters between people with social phobia and healthy controls (Papadimitriou and Linkowski, 2005). Furthermore, Uhde *et al.* (2009) described this combination as ‘much less frequent’ than co-morbidity between insomnia and other anxiety disorders. Finally, Buckner *et al.* (2008) proposed that the relationship between insomnia disorder and symptoms of social anxiety is mediated by major depressive disorder. To summarize, the relationship between insomnia and social anxiety disorder seems to be somewhat more nuanced than the relationships between insomnia and other anxiety disorders; nevertheless, further research is warranted.

Cognitive behavioural therapy (CBT) has been found to be a relatively effective treatment for both social anxiety and insomnia disorder (Rodebaugh *et al.*, 2004; van Straten *et al.*, 2018). A meta-analysis found that 36% of patients with insomnia and co-morbid psychiatric or medical disorders receiving CBT for insomnia (CBT-I) reached remission, and the effect sizes for most symptoms and sleep parameters varied between medium and large (Wu *et al.*, 2015). Similarly, van Straten *et al.* (2018) reported robust and similar results for CBT-I on symptoms of insomnia disorder with and without co-morbidity. However, social anxiety disorder was not included among other co-morbid anxiety disorders, and it remains unknown whether or how symptoms of insomnia are affected by CBT for social anxiety disorder (CBTS; Belleville *et al.*, 2010). One study suggested that treating social anxiety does not affect sleep problems (Kushnir *et al.*, 2014). Similarly, treatment of insomnia disorder seems to affect symptoms of anxiety only moderately (Belleville *et al.*, 2011; Tang, 2010). Poorer sleep quality at baseline, however, predicts a longer recovery period and poorer treatment outcomes of CBTS on social anxiety disorder (Zalta *et al.*, 2013). In a systematic review, Eskildsen *et al.* (2010) concluded that data on co-morbidity as a predictor for drop-out and outcome when treating social anxiety disorder were somewhat inconclusive for major depressive disorder but not for anxiety disorders, where no such relationship was found. No studies exploring insomnia as a predictor for outcome or treatment of social anxiety disorder were identified.

In conclusion, insomnia disorder and social anxiety disorder are common psychiatric disorders whose relationship requires further clarification. The robust results for CBT-I and CBT-S and the

unclear relationship between the disorders suggests that the use of sequential protocols in accordance with the current evidence base may be a feasible strategy to treat co-morbidity of insomnia disorder and social anxiety disorder. No studies have yet investigated the use of both CBT-I and CBT-S on patients with this combination of disorders. This study was designed to explore the effects of sequential CBT treatments on these disorders through the following questions: (1) does sequential treatment based on CBT-S and CBT-I improve symptoms of social anxiety disorder for people with co-morbid insomnia disorder?; (2) does sequential treatment based on CBT-S and CBT-I improve symptoms of insomnia disorder for people with co-morbid insomnia disorder?; and (3) does sequential treatment based on CBT-S and CBT-I decrease depressive symptoms?

Method

Design

A single-case repeated crossover AB design was used in which the baseline measurement phase (A) preceded the treatment phase (B). The repeated measurements during phase A allows for predictions to be made about current symptoms in the absence of treatment, and can be compared with the measurements during treatment in phase B. The combination of repeated measurements in both phases, randomization to the different sequences, and the replication across participants allow for exploration of order effects, testing effects, and regression towards the mean (Kazdin, 2011). Participants were randomized into two different treatment sequences for phase B: CBT-S followed by CBT-I (sequence 1) and CBT-I followed by CBT-S (sequence 2). Participants completed daily and weekly self-rating scales during phase A, phase B, at post-treatment (sequence 1, one week after treatment, and sequence 2 four weeks after treatment due to practical constraints), and at the 4-month follow-up.

Participants and procedure

Participants were recruited through advertisements placed in local media, social media, through email lists to university students, and through counsellors at primary healthcare centres. The included participants ($n=10$; Fig. 1) met the *DSM-5* criteria for social anxiety disorder and insomnia disorder but not for other primary sleep disorders such as sleep apnea (American Psychiatric Association, 2013). All participants were ≥ 18 years of age, not at high risk of suicide [as indicated by ≥ 4 on the Montgomery-Åsberg Depression Rating Scale-Self-Rated (MADRS-S)] and did not suffer from more severe psychiatric disorders like severe depression (total score of ≥ 30 on MADRS-S) or manic states [indicated by Mini International Neuropsychiatric Interview (MINI)]. The participants did not receive any other concurrent psychological treatment and had not changed their medication regimen within the past three months (Table 1).

Measures

All questionnaires and structured interviews were found to have satisfactory psychometric properties (Amorim *et al.*, 1998; Baker *et al.*, 2002; Bastien *et al.*, 2001; Carney *et al.*, 2009; Fantino and Moore, 2009; Fresco *et al.*, 2001; Furmark *et al.*, 1999; Lecrubier *et al.*, 1997; Morin *et al.*, 2011; Sheehan *et al.*, 1997; Sheehan *et al.*, 1998; Svanborg and Asberg, 1994).

Interviews

The MINI (Sheehan *et al.*, 1998) with modified criteria for social anxiety disorder according to *DSM-5* was used to assess psychiatric disorders during screening (American Psychiatric

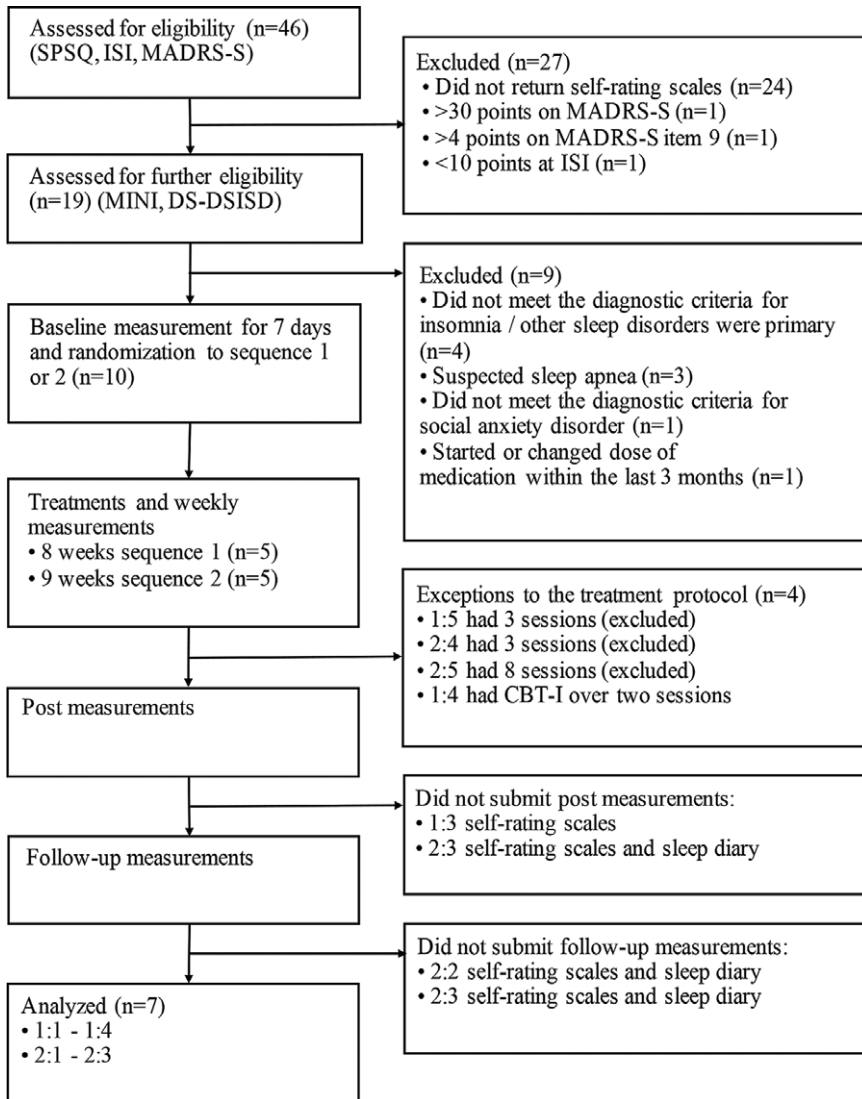


Figure 1. Flowchart of participants.

Association, 2013). The Duke Structured Interview for Sleeping Disorders (DSISD; Carney *et al.*, 2009) was used to assess clinical levels of current sleep disorders according to the *DSM-IV* and the *ICSD-2*, with updated *DSM-5* criteria for insomnia disorder (American Psychiatric Association, 2013). Both interviews were conducted over the telephone.

Self-rating scales

The Social Phobia Screening Questionnaire (SPSQ; Furmark *et al.*, 1999) was administered during the screening procedure and was used alongside the MINI to assess social anxiety disorder. The scale includes 14 items consisting of situations related to social interaction or social performance that are graded from 0 (not at all distressing) to 4 (extremely distressing). The total score ranges from 0 to 56. The assessment of clinical levels was performed according to the instrument's

Table 1. Demographics

Participant	Sex	Age range	Relationship status	Highest education	Employment	Pharmacological treatment	Other psychiatric conditions diagnosed with MINI	Completed treatment
1:1	Female	18–29	Single	High school	Yes	No	Recurrent major depressive disorder, agoraphobia	Yes
1:2	Male	50–59	Partner	High school	Yes	No	—	Yes
1:3	Female	40–49	Single	High school	Yes	No	—	Yes
1:4	Female	18–29	Partner	High school	Yes	No	Generalized anxiety disorder	Yes
1:5	Female	18–29	Partner	High school	Yes	No	Agoraphobia, generalized anxiety disorder	No
2:1	Female	18–29	Partner	High school	Yes	No	Generalized anxiety disorder, primary agoraphobia	Yes
2:2	Female	18–29	Partner	University	Yes	No	Major depressive disorder	Yes
2:3	Female	18–29	Single	High school	Yes	No	Major depressive disorder, primary agoraphobia	Yes
2:4	Male	30–39	Single	High school	Yes	No	Major depressive disorder, generalized anxiety disorder	No
2:5	Male	30–39	Partner	University	Yes	No	—	No

Age ranges were used to maintain participant anonymity.

guidelines, namely, 0–16: probably no social phobia, 17–21: risk zone, >21: probably social phobia, >29: probably generalized social phobia.

The Liebowitz Social Anxiety Scale-Self Report (LSAS-SR; Baker *et al.*, 2002) was used to assess symptoms of social anxiety. This report consists of 24 different social interaction and performance situations that are graded according to the level of induced anxiety and avoidance, respectively, from 0 (no fear/anxiety and no avoidance) to 3 (severe fear/anxiety and severe avoidance). The total score ranges from 0 to 144. Assessment of patient estimates followed the instrument's guidelines, where scores >30 indicate probable social phobia, while scores >60 indicate probable generalized social phobia. The LSAS-SR was administered three times over 7 days during baseline (A), weekly during treatment (B), once post-treatment, and once at the 4-month follow-up.

A modified short version of the Social Phobia Rating Scale (SPRS; Wells and Leahy, 1998) was used to collect daily data on social anxiety. The first three of the five original items were used, which assessed the past 24 hours instead of the past week. The three items consisted of questions regarding how distressing or disabling social anxiety has been, how much social situations have been avoided due to anxiety, and degree of self-consciousness in difficult social situations. The items are rated on an 8-point scale, where low numbers indicate lower extents of distress, avoidance and self-consciousness. The modified version of the SPRS was administered daily during baseline (A) and treatment (B), once daily for a 1-week period post-treatment and once daily for a 1-week period at the 4-month follow-up. A short scale with a relatively wide range of answer options was considered feasible for daily measurements. The modified version of the SPRS was used as the primary measure of social anxiety disorder.

The Insomnia Severity Index (ISI; Bastien *et al.*, 2001) was used to assess symptoms of insomnia disorder. Its seven items are each answered on a 5-point scale, where higher points indicate more severe difficulties. The total score ranges from 0 to 28 (Bastien *et al.*, 2001; Morin *et al.*, 2011). Assessment of participant estimates followed the guidelines of Morin *et al.* (2011), which suggested that a total score ≥ 10 is indicative of clinical insomnia. ISI was administered three times over 7 days during baseline (A), once weekly throughout the treatment period (B), once at post-treatment, and once at the 4-month follow-up.

The Montgomery-Åsberg Depression Rating Scale-Self-Rated (MADRS-S; Svanborg and Åsberg, 1994) was used to assess depressive symptoms. Nine items are answered on a 7-point scale, with a total possible score of 54. The clinical categories used were: 0–12: normal/symptoms absent, 13–19: mild depression, 20–34: moderate depression, and >34: severe depression (≥ 30 was mistakenly used in line with the clinician-administered version to indicate severe depression during screening). The MADRS-S was administered three times over 7 days during baseline (A), once weekly throughout the treatment period (B), once at post-treatment, and once at the 4-month follow-up.

A sleep diary was used to assess sleep patterns. Data consisted of time points for bedtime, last awakening, and time out of bed, but also sleep onset latency (SOL; minutes between going to bed and first sleep onset), wake after sleep onset (WASO; minutes awake after first sleep onset), early morning awakening (EMW; minutes in bed after last awakening), and number of awakenings. Undesired time awake (SOL+WASO) was calculated and used as a primary measure. The sleep diary was administered daily during baseline (A) and treatment (B), once daily for a 1-week period post-treatment and once daily for a 1-week period at the 4-month follow-up.

Treatment

Each participant received 10 treatment sessions: four CBT-I sessions and six CBT-S sessions. Sequence 1 (CBT-S before CBT-I) ran over 9 weeks, and sequence 2 (CBT-I before CBT-S) ran over 8 weeks. The discrepancy in treatment duration was a result of practical constraints.

The CBT-I was a modified version of the Edinger and Carney (2014) protocol. The Swedish translation was made by A. Norell-Clarke (unpublished) and has been used in other CBT-I studies (Norell-Clarke *et al.*, 2015). The protocol consists of two blocks over four sessions. The first block touched on behavioural aspects, such as sleep restriction and stimulus control, while the second block addressed cognitive aspects, such as constructive worry, thought registration and cognitive distortions. As CBT-S included tracking and self-registration of negative automatic thoughts, it was removed from the second block of CBT-I to avoid overlap. Sleep efficiency (effective sleep time divided by total time in bed) was calculated from the participant's sleep diary. Sleep efficiency between 80 and 85% was used as a threshold during sleep restriction (Edinger and Carney, 2014).

The CBT-S was a protocol based on a modified version of a treatment administered online (Furmark *et al.*, 1999; Tillfors *et al.*, 2008). The original protocol was modified to accommodate six individual sessions and included psychoeducation, cognitive restructuring, including tracking and self-registration of negative automatic thoughts (sessions 1–3), behavioural experiments with and without safety behaviours (session 3), exposure (sessions 4–6), and relapse prevention (session 6). The participants completed related assignments at home between sessions in both treatment groups.

The therapists were two Master's students in clinical psychology in their final year of education (D.G. and H.E.). They received supervision from a clinical psychologist and professor in clinical psychology (M.T., an expert on social anxiety disorder) and a clinical psychologist with a PhD in psychology (A.N.-C., an expert on insomnia).

Data analysis

Daily measures

Social anxiety and undesired time awake were analysed primarily visually (see Figs 2 and 3), which is considered the standard procedure for analysing data in a single case design (Kazdin, 2011). Median values, rather than means, were calculated for the baseline phase (phase A), treatment phase (phase B), post-treatment phase, and follow-up phase in order to handle fluctuations with extreme outliers.

Non-overlap of all pairs (NAP) was used to calculate phase differences and effect sizes for the daily measures of social anxiety (modified version of SPRS) and undesired time awake (SOL+WASO; Manolov *et al.*, 2016; Parker and Vannest, 2009; Parker *et al.*, 2011). All data points in phase A were individually compared with all individual data points in phase B; thus, NAP shows the percentage of data that improve across phases. The NAP values were transformed to adjust for chance level ($1 - \text{NAP}/0.5$), with negative values indicating deterioration. The proportion of non-overlaps can be interpreted as follows: weak effects: 0–.31; medium effects: .32–.84; large or strong effects: .85–1.0 (Parker and Vannest, 2009).

For weekly measures, the mean scores for each phase and scale, as well as the differences between baseline means and post-treatment scores, were calculated. The baseline phase (A) consisted of three measures over 7 days, and the treatment phase (B), comprising CBT-I and CBT-S, consisted of weekly measures. The scales used for weekly measures were administered once during post-treatment data collection and once during the 4-month follow-up. Data from missing single items (10 data points across all subjects) were replaced with the mean for the previous and the following weekly measure of that same item, in order to not lose all data from that measurement. More advanced imputation methods were considered superfluous given the few missing data points for single items. Imputation was not conducted when data from more than single items were missing. The data from participants 1:1–4 and 2:1–3 were analysed. Due to missing data and low adherence, data from participants 1:5, 2:4, and 2:5 were excluded from the analysis (Fig. 1).

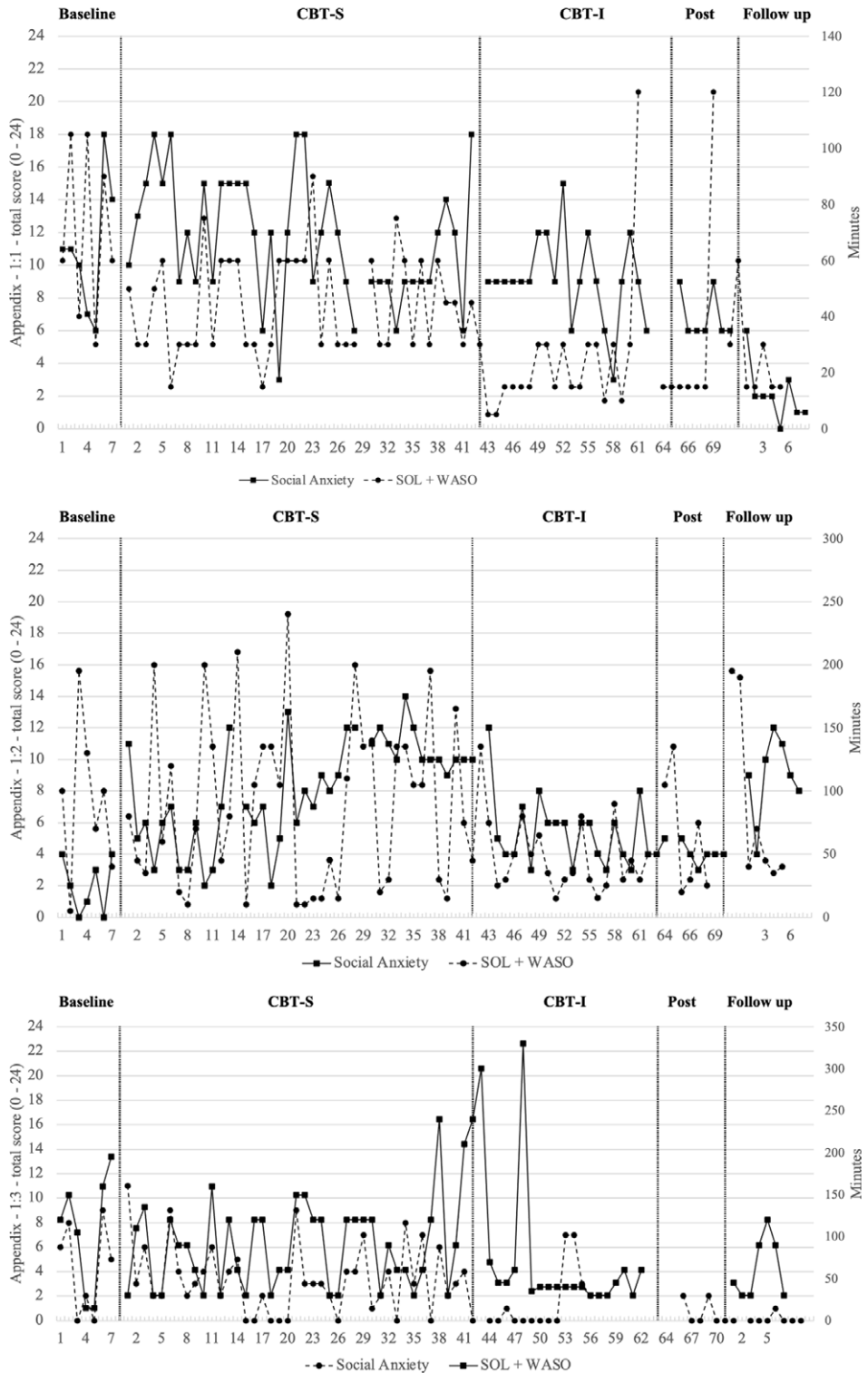


Figure 2. Social anxiety and insomnia symptoms over time for treatment sequence 1.

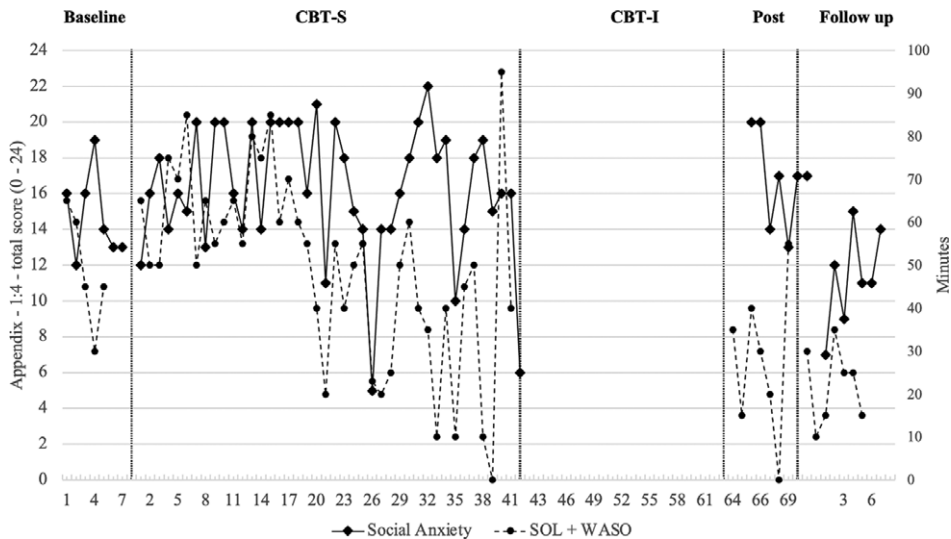


Figure 2. (Continued).

Results

Does sequential treatment based on CBT-S and CBT-I improve symptoms of social anxiety disorder for people with co-morbid insomnia disorder?

Visual analyses of data from the daily social anxiety measures (with a modified version of SPRS) from participants 1:1, 1:3, 2:1 and 2:2 indicated a decrease in reported symptoms between the baseline phase (A) and treatment phase (B). The reported symptoms were either stable or had improved from post-treatment to the 4-month follow-up. Follow-up data for participant 2:2 were not obtained (see Figs 2 and 3). Calculations with NAP corroborated the medium-to-large effect sizes seen in the visual analysis (see Table 2). Further, the weekly social anxiety measures (LSAS-SR) showed symptom reductions for participants 1:1, 1:3, 2:1 and 2:2; however, for participants 2:1 and 2:2, the symptom reductions did not reach non-clinical levels (i.e. <30), which was the case for participants 1:1 and 1:3 at follow-up (see Table 3).

The daily social anxiety data from participants 1:2, 1:4 and 2:3 indicated an increase in reported symptoms between the baseline (A) and treatment (B) phases. However, the reported symptoms for participants 1:4 improved from post-treatment to the 4-month follow-up (see Figs 2 and 3). Neither the post-treatment data nor the follow-up data were obtained for participant 2:3. The NAP scores for these three participants corroborated the visual analysis (see Table 2). The weekly social anxiety measures (LSAS-SR) for participant 2:3 showed similar patterns with increased reported symptoms. For participants 1:2 and 1:4, however, the weekly social anxiety measures instead indicated slight improvements (see Table 3).

Does sequential treatment based on CBT-S and CBT-I improve symptoms of insomnia disorder for people with co-morbid social anxiety disorder?

Visual analyses of data from the daily insomnia measures (SOL+WASO; Figs 2 and 3) from participants 1:1, 1:2, 1:3, 1:4, 2:1 and 2:3 indicated a reduction in undesired time spent awake between baseline (A) and treatment phase (B). The reported undesired time awake was either stable or had improved from post-treatment to the 4-month follow-up. Follow-up data for participants 2:2 and 2:3 were not obtained. The data from participant 2:2 indicated an increase in reported undesired time awake between baseline (A) and treatment phase (B) (see Figs 2 and 3). The same pattern was observed in NAP scores and weekly insomnia measures

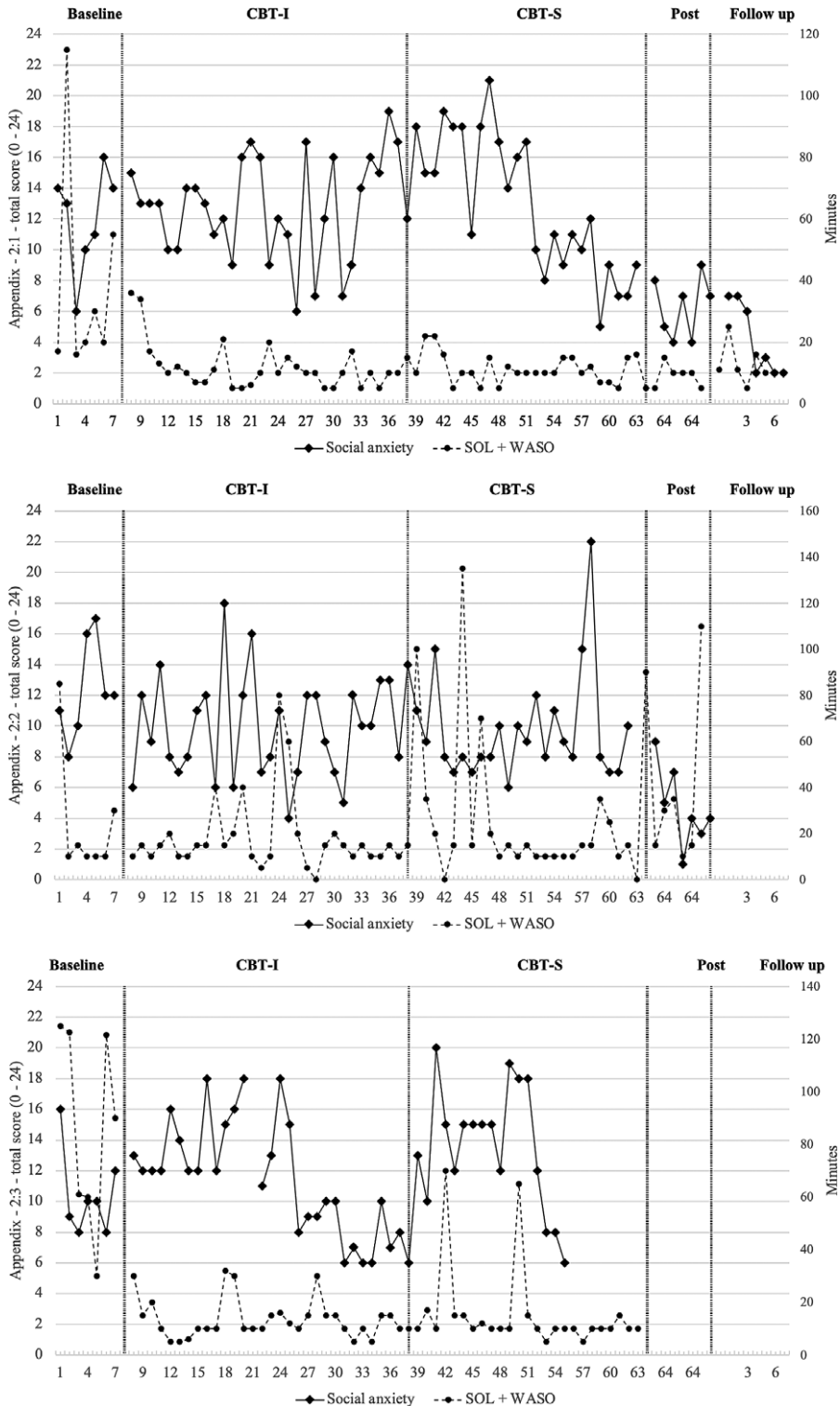


Figure 3. Social anxiety and insomnia symptoms over time for treatment sequence 2.

Table 2. Effect sizes (non-overlap of all pairs) of daily measures

		Participants sequence 1				Participants sequence 2			
SPRS		1:1	1:2	1:3	1:4		2:1	2:2	2:3
	CBT-S	-.11	-.88	.15	-.37	CBT-I	-.01	.45	-.44
	CBT-I	.29	-.79	.44		CBT-S	-.2	.45	-.09
	CBT-S+CBT-I	.02	-.85	.22	-.37	CBT-S+CBT-I	-.12	.45	-.26
	Post	.73	-.69	.59	-.53	Post	.84	.96	
	Follow-up	.98	-.96	.67	.67	Follow-up	.90		
SOL+WASO									
	CBT-S	.51	-.05	.14	.12	CBT-I	.78	-.04	.97
	CBT-I	.84	.47	.29		CBT-S	.91	-.04	.95
	CBT-S+CBT-I	.62	.12	.18	.12	CBT-S+CBT-I	.85	-.04	.96
	Post	.71	.19		.74	Post	1	-.49	
	Follow-up	.84	.14	.37	.93	Follow-up	.82		

Small effect size

Medium effect size

Large effect size.

Non-overlap of all pairs (NAP) on daily measures of social anxiety (modified short version of the Social Phobia Rating Scale [SPRS]) and undesired time awake (sleep onset latency [SOL]+wake after sleep onset [WASO]) between baseline and following sub-phases, including CBT-S+CBT-I. Negative values indicate deterioration. Measurements are presented in chronological order for each sequence.

with ISI (see Tables 2 and 3). Participant 2:2 showed improvement on ISI (14 to 3) but a deterioration in daily measures at post-treatment. A deviation in the opposite direction was noted for participants 1:3 and 1:4, whose ISI scores deteriorated between post-treatment and follow-up (2 to 16 and 11 to 12, respectively; Table 3), whereas the NAP scores of daily measures improved (Table 2). However, post-treatment data from daily measures were missing for participant 1:3. A comparison was therefore made between baseline (A) and the daily measures from the CBT-I treatment).

Does sequential treatment based on CBT-S and CBT-I decrease depressive symptoms?

Weekly measurements with the MADRS-S showed reduced scores (indicating reduced self-reported depressive symptoms) for participants 1:1, 1:2, 1:3, 2:1 and 2:2 between baseline (A) and post-treatment. Post-treatment data from participant 2:3 was missing. Participants 1:1, 1:2 and 2:1 were either stable or had improved between post-treatment and the 4-month follow-up. Follow-up data for participants 2:2 and 2:3 were not obtained (see Table 3). The opposite pattern was observed for participants 1:2 and 1:4, with increased MADRS scores between post-treatment and the 4-month follow-up. Neither the post-treatment data nor the follow-up data were obtained for participant 2:3. However, decreased MADRS scores between baseline (A) and the last sequence for participant 2:3 was noted.

Table 3. Clinical outcome measures

		Participants sequence 1				Participants sequence 2			
		1:1	1:2	1:3	1:4	2:1	2:2	2:3	
LSAS-SR	Baseline	100	57	61	67	Baseline	92	71	40
	CBT-S	98	62	56	68	CBT-I	94	71	43
	CBT-I	69	56	42	—	CBT-S	95	76	60
	Post	45	48	47	44	Post	74	59	—
	Follow-up	29	51	28	35	Follow-up	42	—	—
	Dif	-55	-9	-14	-23	Dif	-18	-12	20
	Dif %	-55%	-16%	-23%	-34%	Dif %	-20%	-17%	50%
ISI	Baseline	20	15	16	18	Baseline	18	14	21
	CBT-S	20	17	14	19	CBT-I	13	10	7
	CBT-I	12	12	15	—	CBT-S	9	8	4
	Post	10	11	2	11	Post	5	3	—
	Follow-up	6	8	16	21	Follow-up	9	—	—
	Dif	-10	-4	-14	-7	Dif	-13	-11	-17
	Dif %	-50%	-27%	-88%	-39%	Dif %	-72%	-78%	-80%
MADRS-S	Baseline	24	11	12	26	Baseline	22	14	15
	CBT-S	19	12	15	25	CBT-I	19	13	11
	CBT-I	9	11	17	—	CBT-S	12	10	12
	Post	7	9	8	21	Post	8	5	—
	Follow-up	7	13	7	29	Follow-up	4	—	—
	Dif	-17	-2	-4	-5	Dif	-14	-9	-3
	Dif %	-71%	-16%	-35%	-19%	Dif %	-64%	-65%	-20%

Mean scores of weekly measurements for each phase and instrument, difference (Dif) between baseline mean and post (or last available) measurement in absolute score and percentage. a–c marks severity through clinical categories: The LSAS; 30 = probably social phobia (a), >60 = probably generalized social phobia (b). ISI; 0–7 = absence of insomnia, 8–14 = subthreshold insomnia (a), 15–21 = moderate insomnia (b), 22–28 = severe insomnia (c).

Discussion

The aim of the current study was to explore the effects of sequential CBT-treatments (CBT-I and CBT-S), primarily on symptoms of social anxiety disorder and insomnia disorder, and secondarily on symptoms of depression. In summary, the majority of participants improved in sleep, social anxiety and depressive symptoms. However, the treatment effects were mixed and participants exhibited varying levels of improvement (and deterioration) for all three disorders. Hence, sequential CBT-treatments are potentially effective in decreasing symptoms of social anxiety and insomnia for people with this understudied co-morbidity, and symptoms of depression do potentially decrease along with the primary symptoms.

The post-treatment measurements indicate that the sequential treatments are more effective for insomnia than for social anxiety, with six of the seven participants showing improvements. However, some reported deviations and fluctuations over time, and there were contradictory data between daily and weekly measures for one participant. One explanation could be that the sleep diary captures time-related insomnia symptoms night by night, whereas the weekly measure with ISI captures symptoms and difficulties related to insomnia more broadly. Taken together, these results are in line with previous studies demonstrating that CBT-I has an effect on insomnia disorder with co-morbid psychiatric or medical conditions (Wu *et al.*, 2015) as well as with co-morbid social anxiety disorder (Tang, 2010). The changes in undesired time awake in this study seem to co-occur with the CBT-I phase rather than during the CBT-S phase, which suggests that CBT-I in itself is effective at reducing symptoms of insomnia disorder in people with co-morbid social anxiety disorder.

The results concerning the treatment effects on social anxiety are more difficult to interpret. Fewer participants reported improvements in social anxiety symptoms than in insomnia symptoms. Participants 1:1, 1:3, 2:1 and 2:2 responded well or relatively well to the treatment, as

demonstrated by both daily and weekly measures of social anxiety. However, the data from the remaining three participants (1:2, 1:4 and 2:3) did not demonstrate any clear improvements except for participant 1:4, who demonstrated improvements in both daily and weekly measures at the 4-month follow-up. From the four participants that displayed improvements, the change seems to be gradual but not within CBT-S. This suggests that the improvement of social anxiety disorder could be attributed to CBT-S with a delayed effect. As the exposure module is found in the latter part of the CBT-S protocol it is not far-fetched to imagine that participants who experienced some initial success would keep approaching previously aversive stimuli in a generalized manner. Another explanation might be that both treatments are needed when treating co-morbid social anxiety disorder and insomnia. It is possible to retrospectively imagine pathways such as a decrease in rumination or worry at night-time, or that one would simply be more well-rested. The results from Tang (2010), however, indicated no decrease in social anxiety from CBT-I in the same population. It is interesting to note that the four participants who improved, together with participant 1:4, reported severe levels of social anxiety before treatment. This suggests that sequential CBT treatments may be more effective for severe social anxiety than for mild or moderate social anxiety. Although comparisons between single-case experimental designs and group designs should be done with great caution, the distribution of the results nevertheless align with those of previous studies on CBT-S whose main components were exposure and cognitive restructuring (Loerinc *et al.*, 2015; Rodebaugh *et al.*, 2004). It is important to note that CBT-S was compressed into six sessions instead of nine in the current study, and that several of the participants (1:1, 1:4, 2:1, 2:2 and 2:3) had additional co-morbid psychiatric diagnoses.

Concerning depression, these results are also inconclusive and with the current design, inferences about treatment effects are therefore difficult to make. All participants who reported data at post-treatment displayed a decrease in MADRS-S scores that ranged between 16 and 71%. However, at the 4-month follow-up, two out of five participants deteriorated (1:2 and 1:4) from the baseline and two participants did not report any follow-up data (2:2 and 2:3). It is interesting to note, however, that the participants with the largest relative decrease in reported depressive symptoms between baseline and post-treatment measurements (1:1, 1:3, 2:1 and 2:2) were the same participants with the highest decrease in social anxiety symptoms between baseline and post-treatment measurements through visual inspection of daily measures from SPRS. This is partly supported by the previous findings proposing depression as a mediator between insomnia disorder and social anxiety disorder (Buckner *et al.*, 2008). As no treatment for depression was administered in the current study, this suggests the possibility of a more complex bidirectional – or just coincidental – relationship between these disorders.

In the current study, only three out of 10 participants completed all of the daily measure entries during baseline, treatment, post-treatment, and follow-up. Furthermore, only half of the participants completed the treatment without modifications. However, drop-out rates are similar to those reported previously (Fernandez *et al.*, 2015), although proportions must, once again, be interpreted carefully when comparisons are made between group studies and single-case experimental studies. The current study was primarily focused on internal validity, but questions of ecological validity in clinical studies with a heavy load of weekly and daily measurements are warranted in future research, especially as the idiographic scientist/practitioner perspective attracts more interest (Hayes *et al.*, 2019).

Some limitations with the current study must be considered. First, relatively large fluctuations were observed during the baseline (A) and treatment (B) phases, which lower the internal validity and render causal conclusions based on visual analysis more difficult to draw. Furthermore, most fluctuations did not stabilize during baseline, which made it more challenging to assess trends and effect sizes. Statistical analyses of effect sizes with NAP were used to address the uncertainty introduced by these fluctuations. Future studies are encouraged to use sufficiently long baseline periods to stabilize fluctuations and to randomize the duration of baseline periods in order to minimize further threats to internal validity, such as history, which cannot be

excluded with the current design. However, no participants in this study reported any major events during the baseline or treatment phases.

Second, the daily social anxiety measure consisted of a modified version of SPRS (in which three of the original five items were used), which increases the uncertainty of some of its psychometric properties. SPRS is developed for group studies wherein the purpose is to measure differences between people over more than 7 days, rather than measuring changes within a person over 1 day. However, the selected items are theoretically relevant to people with social anxiety, namely, distress, avoidance and self-focus. However, it remains unknown to what extent these three items correspond to the problems reported by each participant.

Third, the missing data and drop-out rates are problematic in that they render accurate interpretations of the results more difficult. Future studies should aim to systematically collect more qualitative data to better assess treatment feasibility, especially when scarcely investigated populations and treatments, or combinations of treatments, are investigated with small sample sizes. Fourth, no measures of adherence besides attendance were used. Future studies should track participant adherence to assignments and therapist adherence to protocols. However, treatment manuals were used and reading assignments were given to increase internal validity. Fifth, the two treatments (CBT-I and CBT-S) were given one after the other, which means that delayed effects cannot be ruled out, hence diffusion of treatments. However, an attempt to mitigate this was made by randomizing participants to the different sequences. No clear differences in outcome attributed to the order of treatments were detected even though the ideographic design does not really allow such comparisons.

Finally, some strengths with the current study should also be highlighted. First, participants in single-case experiments double as their own control. By the repeated measurements in our design, threats to internal validity, such as testing and statistical regression, could be explored. Second, additional support for internal validity and to some extent also external validity is that the initial case could be said to have been replicated six times. Third, the participants were randomized to each sequence to control for order effects. Fourth, by using both visual analysis and statistical analysis (NAP), we have a relatively high analytical validity. It is especially important to use a statistical analysis method in addition to the visual analysis when the daily measures show a great deal of fluctuations, as in our case. Fifth, the daily measures are supplemented with weekly measures with questionnaires with sufficient psychometric properties as well as norm data, which allow for somewhat more safe conclusions concerning symptoms of insomnia disorder and social anxiety disorder. Sixth, we tried to reduce effects of social desirability by letting the participants submit their completed questionnaires in sealed envelopes. Last, in order to strengthen internal validity, treatment protocols for both treatments were used and the participants were thoroughly screened with MINI.

In conclusion, the sequential treatments with CBT-I and CBT-S seem to decrease symptoms of insomnia and social anxiety in people with co-morbid insomnia disorder and social anxiety disorder. This study takes a first step to explore one passable treatment strategy for co-morbid insomnia and social anxiety. Future research should strive to increase experimental control and to further assess the relationship between insomnia and social anxiety disorder. In relation to the latter, it would be of interest for future studies to investigate treatment strategies that address the processes of change through which social anxiety and insomnia symptoms could be related. A mediational study on standard CBT and acceptance and commitment therapy (ACT) for social anxiety disorder found that an initial decrease in negative cognitions predicts better outcomes in both CBT and ACT, whereas an initial decrease in experiential avoidance predicts better outcomes in ACT (Niles *et al.*, 2014). Another study found that changes in self-focused attention was associated with improvements in social anxiety in both individual cognitive therapy (ICT) and cognitive behavioural group therapy (CBGT), whereas improvements in social anxiety in CBGT were also associated with changes in anticipatory and post-event processing (Hedman *et al.*, 2013). Both ACT and unified protocol (UP) addresses emotional/experiential avoidance and recent

reviews found that both UP and ACT are effective for insomnia disorder (Cassello-Robbins *et al.*, 2020; Salari *et al.*, 2020) and promising for social anxiety disorder (Cassello-Robbins *et al.*, 2020; Norton *et al.*, 2015). These results suggest that emotional/experiential avoidance, negative cognitions and self-focused attention may be interesting candidates to examine further as processes of change in psychological treatments for people with co-morbid social anxiety disorder and insomnia disorder.

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Author contributions. M.T. and A.N.-C. designed the study. M.T., A.N.-C., D.G. and H.E. contributed to the data collection. M.T., D.G. and H.E. conducted preliminary analyses of the data and D.W., S.A., M.T. and A.N.-C. conducted the final analyses. The first draft of the manuscript was created by D.W. All authors critically reviewed the manuscript and approved the final version.

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Ethical statements. Approval was obtained from the regional ethics review board in Uppsala, Sweden (Dnr: 2016/302). All participants received written information concerning the study and their participation, and they completed an informed consent form. All data were coded and an age range was presented to protect participant anonymity. The clinical trial was registered retrospectively at: =<https://www.anzctr.org.au/Trial/Registration/TrialReview.aspx?ACTRN=12617001165370>. (clinical registration number: ACTRN12617001165370).

Data availability statement. The data that support the findings of this study are available on reasonable request from the corresponding author, D.W. The data are not publicly available due to information that could compromise research participant privacy/consent.

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