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Sleep quality and outcome of exposure therapy in adults with Social Anxiety Disorder

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Abstract

Introduction: Poor sleep is prevalent among individuals with social anxiety disorder (SAD) and may negatively affect exposure therapy outcomes. Poor sleep may impair memory and learning, and thus compromise fear extinction learning thought to take place in exposure therapy. We examined poor sleep as a predictor of exposure therapy outcomes for SAD and the moderating role of D-cycloserine (DCS) on this relationship.

Methods: Participants were 152 individuals with a primary diagnosis of SAD. As part of a randomized clinical trial evaluating the efficacy of DCS for enhancing the effects of exposure therapy, they completed self-report baseline measure of sleep quality, and self-report sleep diaries assessing sleep duration (total sleep time [TST]) and sleep quality the nights prior to and after treatment sessions.

Results: Poorer baseline sleep quality was significantly associated with slower improvement over time and worse symptom outcomes at the end of treatment and follow-up after controlling for baseline symptoms of depression and social anxiety. Greater TST the night prior to treatment

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Study concept and design: Dutcher, Smits, Rosenfield, Taylor, Dowd, Zalta

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predicted lower SAD symptoms at the next session, after controlling for symptoms at the previous session. There was no relation between prior or subsequent night sleep quality on symptoms at the next session. No associations were moderated by DCS.

Conclusions: We replicated and extended findings indicating that poor sleep quality is associated with poorer exposure therapy outcomes for SAD. Assessing for sleep difficulties prior to treatment initiation and incorporating sleep interventions into treatment may enhance exposure therapy outcomes for SAD.

Keywords

social anxiety disorder; cognitive behavioral therapy; exposure therapy; treatment outcomes; sleep quality; sleep difficulties; d-cycloserine

INTRODUCTION

Social anxiety disorder (SAD), a prevalent psychiatric diagnosis (Kessler et al., 2005), is often associated with sleep disturbance (Buckner et al., 2008; Ramsawh et al., 2009). One study found that individuals with SAD report poorer sleep quality, longer sleep latency, more frequent sleep disturbance, and more severe daytime dysfunction (Stein et al., 1993). However, the literature on sleep disturbance in SAD is limited and inconsistent compared to literature on sleep and other anxiety disorders (Mellman, 2008; Roth & Stang, 2007; Taylor et al., 2020). One study revealed no associations between SAD and self-reported sleep disturbances (Marcks et al., 2010) and another reported sleep disturbance did not differ from that of healthy controls (Brown et al., 1994). Pace-Schott et al. (2018) found sleep disturbance was only moderately elevated among individuals with SAD. Other work, however, suggests SAD and sleep are closely related, such that 50–66% of individuals with SAD report sleep disturbances (Horenstein et al., in press; Stein et al., 1993; Zalta et al., 2013).

Sleep difficulties may impede learning and memory, both of which play a crucial role in cognitive behavioral therapy (CBT; Harvey et al., 2014). Sleep promotes cognitive flexibility in problem solving, aids integration of new information, and helps stimulate memory associations (Stickgold & Walker, 2013). Impairments in sleep contribute to reduced ability to reason logically and sustain attention (Blagrove et al., 1995; Gobin et al., 2015). Because sleep before learning facilitates new learning (i.e., encoding information; Harvey et al., 2014; Kredlow et al., 2018), poor sleep prior to therapy may lessen acquisition of new learning during the session. Specifically, sleep loss compromises the ability to commit new experiences to memory (Yoo et al., 2007) and sleep deprivation impedes forming memories of emotional content (Walker & Stickgold, 2006). Furthermore, sleep after learning promotes key neural processes required for memory consolidation (Abel et al., 2013; Diekelmann & Born, 2010; Rasch & Born, 2013). Difficulties with sleep can disrupt memory consolidation processes (Krause et al., 2017), possibly impairing fear extinction learning, a putative mechanism of action of exposure-based treatments (Craske et al., 2008; Kredlow et al., 2018). Indeed, results from experimental fear conditioning have shown that sleep deprivation impairs extinction learning, poorer extinction retention, and reduced ability

to generalize extinction learning (Pace-Schott et al., 2009, 2015; Spoormaker et al., 2010; Spoormaker et al., 2012; Straus et al., 2017).

Studies manipulating sleep after exposure therapy have attempted to translate the findings relating sleep to fear extinction. One study showed that a 90-minute nap after a one-session virtual reality exposure improved the effectiveness of exposure therapy for spider phobia (Kleim et al., 2014). Another study found that overnight sleep after exposure therapy for spider phobia, relative to daytime wakefulness, resulted in greater retention and generalization of extinction learning to novel stimuli (Pace-Schott et al., 2012). Pace-Schott et al. (2018) also examined the impact of post-exposure naps compared to wakefulness after two exposure sessions of a five-session protocol for individuals with SAD. Although sleep did not change clinical outcomes, participants who napped showed a trend toward lower psychophysiological and cortisol responses to a social stressor task post treatment, which suggests sleep might reduce stress reactivity.

To date, few studies have examined self-reported sleep disturbances in relation to exposure-based CBT outcomes for SAD. One study found that poorer baseline self-reported sleep quality was associated with slower improvement over time and higher symptom severity at the end of treatment (Zalta et al., 2013). Similarly, another study found that individuals with co-occurring SAD and subjective sleep disturbances, compared to those without sleep disturbance, evidenced a more severe clinical presentation both at treatment onset and termination (Kushnir et al., 2014). Results from a recent study, however, found that baseline sleep quality did not moderate CBT treatment outcomes for SAD (Horenstein et al., 2019). Only one study to date has examined the within-treatment relation of sleep, in addition to baseline sleep, and exposure therapy. Zalta et al. (2013) found that greater self-reported "restedness" *after* exposure-based therapy sessions uniquely predicted subsequent symptom reduction. To the best of our knowledge, no studies have examined whether sleep quality and duration the night *prior* to sessions is related to symptom change.

If sleep disturbances negatively impact extinction learning, it is also possible that medications known to facilitate consolidation of extinction learning mitigate the effects of sleep disturbances. D-cycloserine (DCS), a partial NMDA receptor agonist, has been shown to augment fear learning and enhances exposure therapy outcomes for SAD (Guastella et al., 2008; Hofmann et al., 2006). Work from animal literature examining DCS and sleep found that administration of DCS to sleep deprived rats partially reversed the negative impact of sleep deprivation on extinction learning (Silvestri & Root, 2008). Furthermore, recent work with healthy adults found that a single dose of DCS administered to a sleep condition and a wakefulness condition prior to a declarative learning task enhanced new learning (Alizadeh Asfestani et al., 2018). In clinical samples, however, DCS did not mitigate the negative effects of sleep disturbance on exposure therapy outcomes for SAD (Zalta et al., 2013).

Building upon the aforementioned work, this study aimed to test the relation between exposure therapy outcome and sleep disturbances, measured both at baseline, and before and after exposure-based therapy sessions. Based on the previous research, we hypothesized that (1) lower self-reported sleep quality at baseline would be associated with less treatment gains and higher symptom severity both at end of treatment and at follow up; and (2) lower

self-reported sleep quality and duration, both before and after a designated therapy session (session X), would be associated with worse symptoms at the subsequent session (session X + 1) when controlling for symptoms at the designated session (session X). Lastly, we investigated whether DCS would moderate the relationship between both sleep quality and sleep duration with SAD treat outcome in hypotheses 1 and 2.

METHODS

Participants

Participants (N=152) were adults with a diagnosis of SAD who participated in a clinical trial testing the efficacy of D-cycloserine augmentation of exposure therapy (Hofmann et al., 2015; Smits et al., 2020). The full sample of 152 participants, recruited across three study sites (Boston University [BU, N=52], Rush University Medical Center [Rush, N=49], University of Texas at Austin [UT, N=51]), was used to analyze perceived baseline sleep. A truncated sample (N=105) was used to analyze sleep before and after therapy sessions due to incomplete sleep diary data from the Rush site. To be eligible for the study trial, participants had to score ≥60 on the Liebowitz Social Anxiety Scale (LSAS). Exclusion criteria included: (1) lifetime history of bipolar, psychotic, or obsessive-compulsive disorder; (2) eating disorder, posttraumatic stress disorder, or substance use disorder in the past 6 months; (3) cognitive dysfunction; (4) significant suicidal ideation or suicidal behaviors in the past 6 months; (5) history of seizures; (6) pregnant, lactating or of childbearing potential and not using contraception; or (7) concurrent treatment or prior non-response to exposure therapy.

Procedures

Detailed information about the methods of the parent trial has been described elsewhere (Hofmann et al., 2015; Smits et al., 2020). The sites used identical study protocols and received IRB approval. After completing a basic online eligibility screen, potential participants provided written informed consent and completed an in-person eligibility session (e.g., diagnostic clinical interview, administration of symptom severity measures, medical evaluation). Eligible participants completed a baseline assessment with an independent evaluator and underwent a 5-week, 90-minute session/week group-exposure therapy protocol. The treatment protocol included one psychoeducation session followed by exposure practice sessions combined with pill administration. At session 2, participants were randomly assigned to one of four pill conditions: placebo (PBO) before the session, and either DCS after a successful session or PBO after a non-successful session (Tailored); (2) DCS before the session and PBO after the session (Pre-session); (3) PBO before the session and DCS after the session (Post-session); or (4) PBO before and after the session (Placebo). Pill administration occurred on sessions 2–5. For the purposes of this study, the three DCS conditions were collapsed into one DCS group, as no prior work suggests dose-timing impacts sleep.

Measures

Retrospective Baseline Self-Report Sleep Quality.—The Pittsburgh Sleep Quality Index (PSQI; (Buysse et al., 1989) comprises seven component items rated on 0–3 scale,

yielding a total score between 0–21. A score of 5 differentiates “good” sleep quality (lower scores) from “poor” sleep quality (higher scores).

Prospective Self-Report Duration and Quality.—The Consensus Sleep Diary (Carney et al., 2012) is a self-monitoring tool to record sleep on a night-by-night basis, collecting information such as nightly sleep-onset latency, wakefulness after initial sleep-onset, total sleep time, total time spent in bed, sleep efficiency, and sleep quality (e.g., “How would you rate the quality of your sleep?” on a 5-point Likert scale ranging from 1 (very poor) to 5 (very good). The current study specifically investigated only perceived sleep quality and duration (i.e., total sleep time [in minutes]; TST) the nights prior to and after all therapy sessions.

Treatment Outcome.—The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) and the Social Phobic Disorders-Severity Form (SPD-S; (M. R. Liebowitz et al., 1992) were administered by trained independent evaluators blind to study condition. Symptom severity was assessed at baseline, across the course of the intervention, and at a 1-week, 1-month, and 3-month follow-up.

Covariate.—The Montgomery-Asberg Depression Rating Scale (MADRS; (Montgomery & Asberg, 1979) assesses 10 symptoms of depression rated on a scale from 0 to 6, with anchor points specific to each symptom item.

Statistical Analysis

Multilevel modeling (MLM) was used to analyze data from the multiple assessments of LSAS and SPD-S, and the sleep predictor variables. MLM is intent-to-treat and is the recommended method for analyzing longitudinal psychiatric data (Hamer & Simpson, 2009), as it accommodates missing data without requiring imputation. Because there were two primary outcome variables (LSAS and SPD-S) we performed a multivariate MLM (MMLM) as it reduces Type I error, increases power (Hox et al., 2017), and avoids inconsistent findings. The 4-level MMLM analysis consisted of the 2 primary outcomes nested within repeated assessments over time, which were nested within individuals, which were nested within treatment cohort. As described in the main outcome paper, we modeled the growth curve of the multivariate outcome from the time of randomization through the 3-month follow-up (Smits et al., 2020). Time was modeled as the LN of weeks from randomization because improvement levelled off over time.

For hypothesis 1, we examined baseline PSQI-assessed sleep quality as a predictor and moderator in our growth curve model. Baseline LSAS and SPD-S, as well as baseline MADRS, were included in the model as covariates. For hypothesis 2, we conducted two sets of analyses. Our first set of analyses for hypothesis 2 examined the relation between both sleep diary-assessed sleep quality the night prior to exposure therapy sessions (i.e., sessions 2–5), and sleep quality the night after exposure sessions, with next session symptom severity (in separate analyses). In these MMLM analyses, self-reported sleep quality prior to (or following) each weekly exposure session was modeled to predict symptom severity at the beginning of the next therapy session. In the second set of analyses for hypothesis

2, we examined sleep diary-assessed TST the night prior to (and the night after) all exposure therapy sessions as a predictor of symptom severity at the next session (in separate analyses). In both sets of analyses for hypothesis 2 (see Figure 1), sleep quality or TST was added as a time-varying predictor (TVP) of SAD symptoms at the next assessment, after controlling for the outcome at the previous session. Because TVPs can confound the between-subjects and within-subjects relations among the TVP and outcome, we disaggregated sleep quality and TST at each timepoint for each subject into their between-subjects average level and their within-subjects deviations from their average level (Hedeker & Gibbons, 2006; Wang & Maxwell, 2015). When TVPs are disaggregated in this way and used as cross-lag predictors of outcome while controlling for earlier level of outcome, the within-subjects relations can reflect quasi-causal relations between predictors and outcome (Hamaker et al., 2015). Since Wang and Maxwell (2016) have shown that these within-person effects (the association of deviations in sleep with symptom severity across time-points) are uncorrelated with between-person effects (which include the associations of sleep with symptom severity at baseline), these within-person effects are unchanged when controlling baseline associations between sleep and symptoms severity.

Finally, we assessed whether the relations between sleep quality and/or TST and symptom severity (hypotheses 1 and 2) were moderated by DCS treatment. We added the interaction between DCS condition and sleep quality (or TST) as a predictor of symptom severity at the next therapy session. Since this analysis involved many analyses, we used the Benjamini-Hochberg method to correct for inflation of Type 1 error due to the multiple tests (Benjamini & Yekutieli, 2001).

RESULTS

Means and standard deviations of baseline study variables are reported in Table 1. Participants (mean [SD] age, 29.24 [10.16] years; 84 men [55.26%]) reported an average baseline global PSQI score of 6.40 (SD=3.08, range 0–16), with 56% of the sample identified as “poor” sleepers. Means and standard deviations of symptoms (LSAS and SPD-S) at each timepoint are reported in Table 2. Means and standard deviations of sleep diary variables prior to and following exposure therapy sessions (sessions 2–5) are reported in Table 3.

Hypothesis 1: Relation between baseline sleep quality and treatment outcome

Baseline PSQI sleep quality predicted improvement over time, such that poorer baseline sleep quality was associated with slower symptom improvement over time, b=.03, t(821)=3.71, p<.001, for the sleep quality x time interaction. Similarly, poorer baseline sleep quality predicted worse outcomes at the final follow-up, b=.08, t(285)=4.51, p<.001. Figure 2 displays the change in outcomes over time for participants with low baseline PSQI scores (1 SD below the mean; PSQI=3.32, reflecting better sleep quality) and high baseline PSQI scores (1 SD above the mean; PSQI=9.48, reflecting poorer sleep quality).

Hypothesis 2: Relation of pre- and post-session sleep quality and sleep time with next session outcome.

Relation between sleep diary perceived sleep quality and next session outcome.—No significant effects of pre-session sleep quality were detected. Analyses also showed there were no effects of post-session sleep quality on next session symptom outcome.

Relation between sleep diary perceived total sleep time and next session outcome.—Participants who had higher mean levels of TST before the exposure sessions had lower symptoms at the next session, b=−.09, t(73)=−2.35, p=.021. Also, deviations from their average level of pre-session TST were related to symptoms at the next session. Specifically, there was a significant time x pre-session TST deviations interaction, b= −.001, t(322)=−2.07, p=.039, indicating that the relation between pre-session TST deviations and outcome varied over time. To understand the nature of this interaction, we examined the relation between pre-session TST deviations and outcome at the next session, at each time point (each session). Whereas deviations from their average level of pre-session TST were not related to next session symptom severity for the initial exposure sessions, higher deviations of pre-session TST at the final exposure session were significantly related to symptoms at the 1-week follow-up assessment, b= −.001, t(304)=−2.00, p=.046.

Post-session TST was also related to outcome, but there was a significant time x mean levels of post-session TST interaction, b= −.11, t(261)=−2.30, p=.022. Higher mean levels of post-session TST were not related to symptoms severity at the next session at the first few exposure sessions, but greater mean level of TST after exposure sessions was related to lower symptom severity at the last exposure session, b= −.12, t(110)=−2.59, p=.011.

DCS Interaction Analyses

DCS as a moderator of the effects of sleep quality and TST in Hypotheses 1 and 2.—None of the interactions or main effects involving DCS were significant (ps = .06 - .90, after the Benjamini-Hochberg correction for multiple tests. Effect sizes (uncorrected for multiple tests): Cohen’s ds = .026 - .295, mean effect size = .126. Thus, even the largest uncorrected effect size was only slightly greater than a “small” effect size, which is a Cohen’s d = .20).

DISCUSSION

Our results suggest poorer baseline self-reported sleep quality was related to significantly slower improvement over time and worse symptom outcomes at the end of treatment and at the 3-month follow-up, after controlling for baseline symptoms of depression and social anxiety. We also found that TST the night prior to therapy sessions was related to subsequent symptom outcomes after controlling for prior symptom severity levels, a finding consistent with the notion that pre-therapy sleep has an important role in next-day therapeutic learning (Harvey et al., 2014). Additionally, individuals who reported greater sleep duration (i.e., TST) than their own average the night after therapy had lower symptoms at the next assessment. This finding, however, was only evident at the last therapy session, which may

be spurious or may be evidence of an additive effect. Our findings that sleep duration prior to therapy are related to symptoms at the next assessment are in line with findings from studies employing fear conditioning and extinction paradigms. Indeed, recent work suggests sleep deprivation prior to extinction learning impaired subsequent memory of extinction learning during extinction recall (Straus et al., 2017). Reduced sleep duration (i.e., sleep deprivation) impairs acquisition of new information and the ability to recall previously stored information (Harvey et al., 2014; Kredlow et al., 2018; Stickgold & Walker, 2013), thus it is possible new learning in session was impaired for individuals with reduced sleep duration prior to treatment sessions.

Contrary to our prediction, neither sleep diary-assessed sleep quality the night prior to or after therapy sessions was significantly associated with next session symptom outcome. The finding that post-session sleep quality was not significant was particularly surprising given existing evidence that greater perceived “restedness” the night after a treatment session predicted lower symptoms at the beginning of the next treatment session (Zalta et al., 2013). One possible explanation for these non-significant findings is that participants tended to rate their perceived sleep quality as “fair” and thus did not identify as sleep deprived the nights prior to and after treatment sessions. It is also possible that patient’s heuristic for how they rate their sleep “quality” is not the same as how they rate their “restedness” the next day. It is important to note that underlying problems such as sleep disordered breathing could result in patients feeling unrested the next day, even though they perceived sleeping throughout the night (e.g., quality). Because the TST findings in this study, but not those related to sleep quality, were similar to Zalta et al.’s “restedness” findings, additional work is needed to elucidate which elements of sleep (i.e., sleep quality, sleep duration, sleep efficiency, sleep latency, waking during the night) have the strongest associations with self-reported sleep difficulties in individuals undergoing CBT for SAD.

We also investigated if DCS moderated the negative effects of poor sleep quality and duration on treatment outcomes. Our finding that DCS did not moderate the relationship between prospective self-report sleep disturbances and worse SAD treatment effects were aligned with previous work and replicate Zalta et al., (2013). It is possible that the mechanisms underlying DCS and sleep differentially impact treatment outcomes. Although both sleep and DCS facilitate off-line learning processes (e.g., consolidation of new information), DCS has been found to facilitate off-line learning during wakefulness (Kuriyama et al., 2013). It is also possible that meaningful effects do exist yet detecting those requires a study with a large sample size.

Similar to Zalta et al. (2013) and Horenstein et al. (2019), our study demonstrates relatively high rates of sleep disturbance among treatment-seeking individuals with SAD and provides additional support to the notion that sleep impairments negatively affect exposure-based therapy efficacy for SAD. Hence, clinicians may consider assessing for sleep difficulties at the start of treatment. Our findings point to the critical importance of delineating the distinct roles of sleep before therapy (i.e., learning) and after therapy (i.e., memory consolidation), and their effects on interrupting the efficacy of exposure-based CBT. It is also possible that sleep disrupts exposure therapy through other mechanisms, such as affective functioning processes like emotion regulation (Germain, 2013; Harvey et al., 2014; Watling et al.,

2017). For example, evidence suggests poor sleep quality is predictive of reduced cognitive reappraisal abilities to regulate negative emotion (Mauss et al., 2013; Watling et al., 2017). Nevertheless, implementing interventions that target poor sleep (e.g., stimulus control, relaxation, and cognitive restructuring of sleep-related beliefs) prior to and throughout the course of exposure-based therapy may assist in maximizing therapeutic outcomes for SAD.

Several limitations of our study should be noted. First, participants were not evaluated for insomnia or other sleep disorders. Another limitation was the sole reliance on self-report measures of perceived sleep quality. Because discrepancies between subjective and objective measures of sleep are well documented (Argyropoulos et al., 2003; Baker et al., 1999), the use of both subjective and objective methods are necessary to fully capture and characterize the sleep experience (Dietch & Taylor, 2019; Lauderdale et al., 2008) and their relation with treatment processes and outcome. More work is also needed to examine memory related sleep architecture as predictors of SAD treatment outcomes. Additionally, future work would benefit from account for the timing of treatment sessions to examine circadian effects on exposure therapy outcomes (Meuret et al., 2016). Sleep diary information was only collected on the night before and night after each exposure session. Daily sleep data across the duration of the active treatment period may prove more informative in capturing week-to-week changes in associations of sleep quality and symptom outcome. Furthermore, the current study did not administer the Pittsburgh Sleep Quality Index at end of treatment or follow-up assessments, thus we were unable to examine if perceived sleep quality improved after treatment. Although we controlled for symptoms of depression and social anxiety symptoms at baseline, it is possible that additional variables not accounted for (e.g., hyperarousal, trait anxiety) contributed to sleep quality, symptom severity, and treatment outcome. Lastly, the approach was correlational and therefore limits causal inferences.

CONCLUSIONS

This study provides additional evidence that poor sleep is associated with worse symptom outcome for social anxiety disorder. Furthermore, this is the first study to examine sleep prior to exposure therapy sessions for SAD. Sleep difficulties may prove an obstacle for optimizing therapeutic gains; thus, clinicians should consider assessing for sleep difficulties and incorporate sleep relevant techniques into their treatment plans. Our findings support further clinical investigation into the effects of sleep on exposure-based CBT outcomes for SAD.

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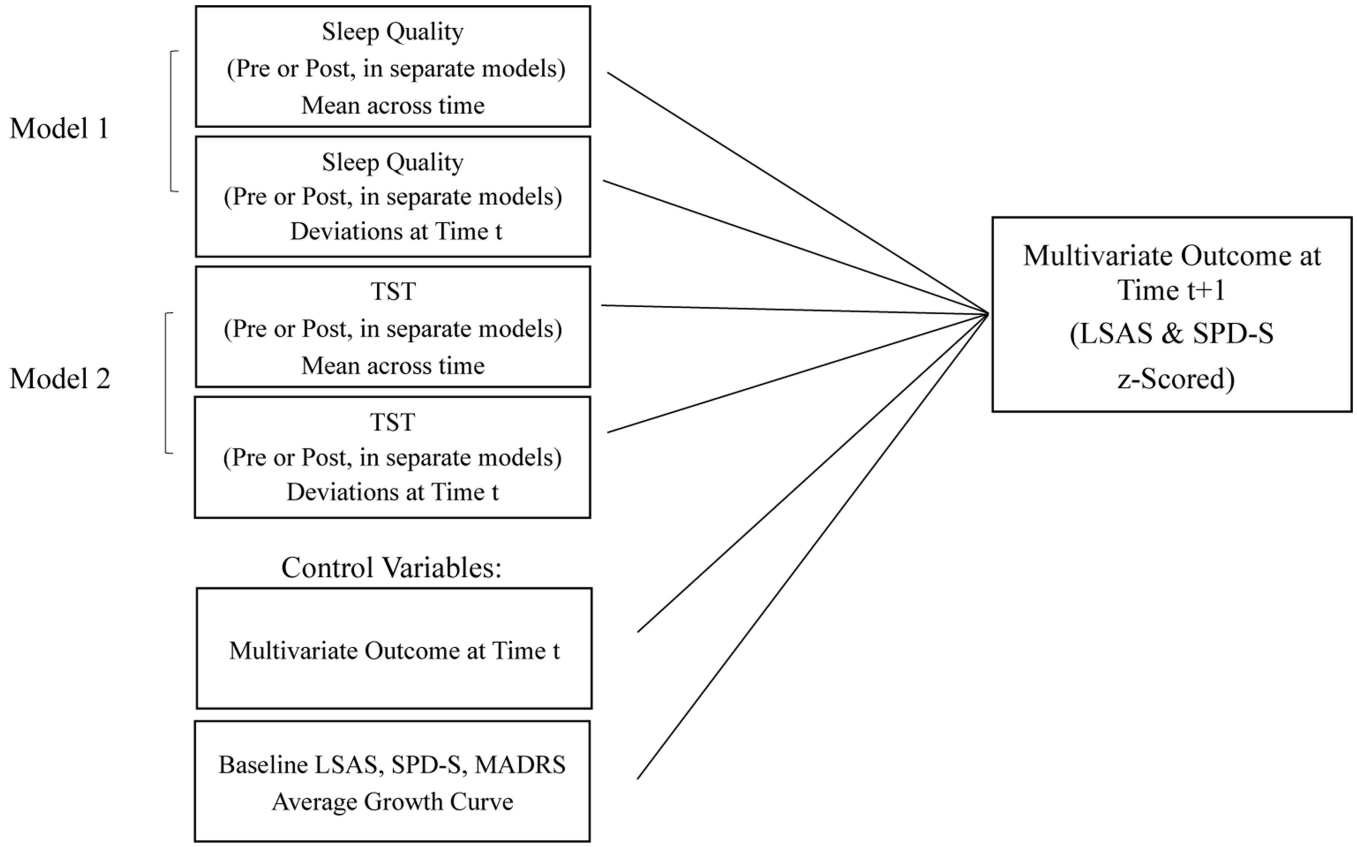

Figure 1.
Cross lag panel model for sleep quality and sleep duration predicting social anxiety symptoms at next session.

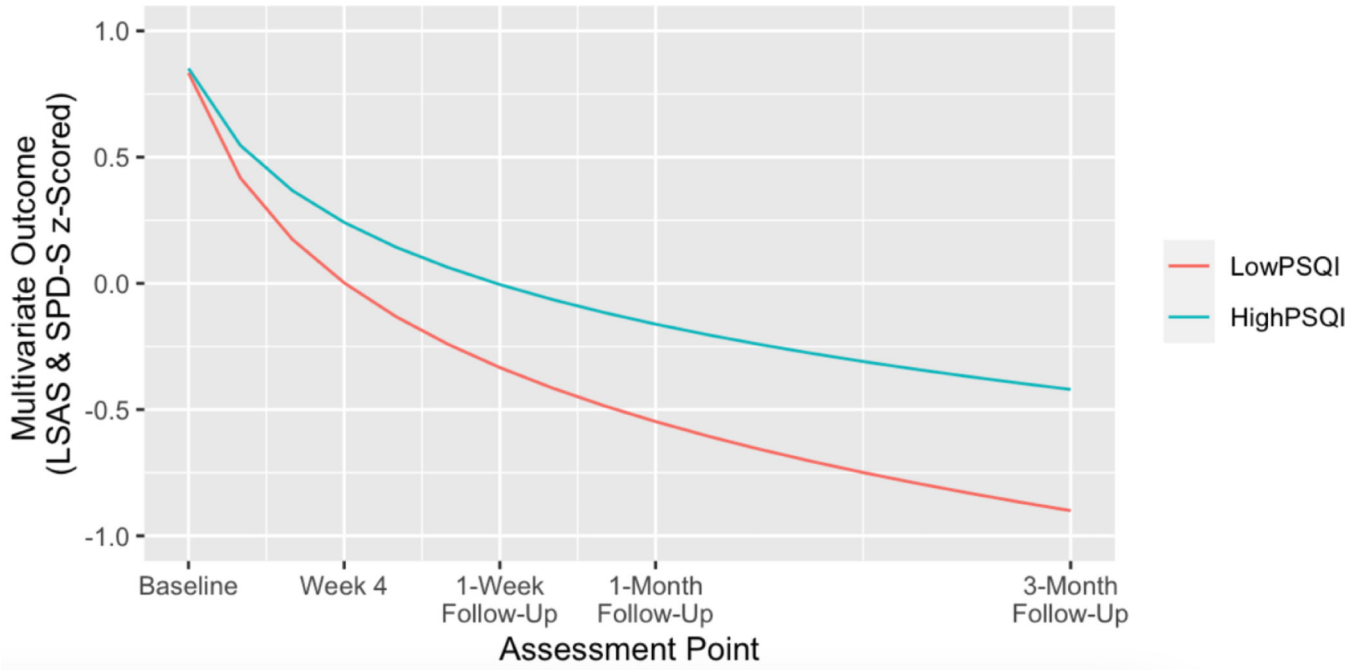

Figure 2.

Slope of symptom change for participants with low or high baseline sleep quality. Low PSQI represents 1 standard deviation below the mean and high PSQI represents 1 standard deviation above the mean.

Table 1.

Baseline study variables.

	Mean N=152	SD N=152
LSAS	82.58	16.90
SPD-S	5.52	0.77
MADRS	13.50	8.68
PSQI	6.40	3.08

Note. LSAS = Liebowitz Social Anxiety Scale, SPD-S = Social Phobic Disorders-Severity Form, MADRS = Montgomery-Asberg Depression Rating Scale, PSQI = Pittsburgh Sleep Quality. Index

Table 2.

Raw means and standard deviations of the symptom outcomes at each timepoint.

Week	LSAS Total (N=152)	SPD-S Total (N=152)
1	82.38 (16.76	5.53 (0.79)
2	80.73 (17.70)	5.44 (0.83)
3	73.49 (18.27)	5.12 (0.97)
4	68.90 (20.89)	4.74 (1.28)
5	64.37 (21.82)	4.44 (1.40)
6	61.80 (23.96)	4.29 (1.55)
10	58.81 (24.60)	4.06 (1.66)
18	55.58 (25.96)	3.80 (1.76)

Note. LSAS = Liebowitz Social Anxiety Scale, SPD-S = Social Phobic Disorders-Severity Form

Table 3.

Raw means and standard deviations of weekly sleep diary variables at each timepoint.

Week		Pre-session (n = 105)	Post-session (n = 105)
Sleep Quality			
	2	3.60 (0.87)	3.49 (0.83)
	3	3.43 (0.88)	3.52 (0.80)
	4	3.44 (0.92)	3.54 (0.78)
	5	3.38 (0.89)	3.48 (0.89)
TST			
	2	457 (114.5)	462 (123.2)
	3	444 (113.0)	441 (109.8)
	4	456 (92.8)	453 (109.0)
	5	440 (114.4)	457 (96.1)

Note. Sleep Quality = perceived sleep quality indexed via Consensus Sleep Diary, TST = total sleep time (duration of minutes slept) indexed via Consensus Sleep Diary.