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BRIEF CLINICAL REPORT

Anxiolytic impact of cognitive behavioural therapy for insomnia in patients with co-morbid insomnia and generalized anxiety disorder

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Abstract

Background: Cognitive behavioural therapy for insomnia (CBT-I) is an effective treatment for chronic insomnia that also improves non-sleep symptoms, such as mood and anxiety. Identifying sleep-specific variables that predict anxiety change after CBT-I treatment may support alternative strategies when people with generalized anxiety disorder (GAD) do not improve from standard GAD treatment.
Aims: To investigate CBT-I on changes in anxiety and evaluate whether changes in sleep-specific variables predict anxiety outcomes.
Methods: Seventy-two participants presenting with insomnia and GAD (GAD-I) completed four sessions of CBT-I. Participants completed daily diaries and self-report measures at baseline and post-treatment.
Results: CBT-I in a co-morbid GAD-I sample was associated with medium reductions in anxiety, and large reductions in insomnia severity. Subjective insomnia severity and tendencies to ruminate in response to fatigue predicted post-treatment anxiety change, in addition to younger age and lower baseline anxiety.
Conclusions: The findings suggest that younger GAD-I participants with moderate anxiety symptoms may benefit most from the anxiety-relieving impact of CBT-I. Reducing perceived insomnia severity and the tendency to ruminate in response to fatigue may support reductions in anxiety in those with GAD-I.

Keywords: Anxiety; CBT; Insomnia; Predictors of recovery

Introduction

Chronic insomnia is a costly and debilitating disorder characterized by a subjective complaint of difficulty falling asleep, staying asleep, and/or waking up too early, occurring at least three times a week for a period of three months or longer. These night-time complications are paired with perceived sleep deficits and subsequent daytime impairments, such as fatigue and concentration difficulties.

Although insomnia is pernicious and disruptive as a stand-alone disorder, chronic sleep disturbances are also highly co-morbid across numerous medical and psychiatric conditions. One area that has received relatively less scrutiny is generalized anxiety disorder (GAD). This paucity is surprising given that chronic insomnia and GAD are frequently co-morbid and mutually influence each other's course and outcome (Hertenstein *et al.*, 2019).

Given this relationship, one clinical implication that naturally follows is that treatment of one disorder may naturally lead to alleviation of the co-morbid disorder. One recent study evaluated the efficacy of CBT-I on a larger sample of patients with insomnia disorder co-morbid with GAD

(Jansson-Fröjmark and Jacobson, 2021). Results indicated a moderate to large effect size in GAD symptom reduction after 10 weekly individual face-to-face CBT-I treatment sessions. The researchers attributed these pronounced improvements to the intervention's emphasis on cognitive therapy and the use of behavioural experiments. However, more research is needed to identify sleep-specific predictors that may contribute to reductions in GAD-related worries and anxiety.

There are several conceptual mechanisms that are likely to contribute to the bi-directional relationship between insomnia and GAD. For instance, elevated levels of worry and arousal in individuals with GAD at night may significantly disrupt sleep architecture, impacting sleep initiation and continuity. In the daytime, GAD is characterized by repetitive negative thinking, which is a cognitive process that involves repetitive and negative thoughts about oneself and the world, which are often abstract, intrusive, and difficult to control. This sustained cognitive activation may contribute to subjective fatigue, which may pull an individual towards reduced activity and rest. The resulting compensatory behaviours may subsequently impact sleep–wake homeostasis and reduce build-up for deep sleep, which in turn further drives the cycle for poorer sleep quality at night-time, contributing to greater levels of anxiety.

Overall, the research, although nascent, appears promising with regard to the utility of CBT-I in alleviating anxiety in individuals presenting with co-morbid insomnia and GAD. Moreover, there is strong theoretical reasoning that CBT-I and its treatment components are well-equipped to disrupt the potential mechanisms that perpetuate anxiety from a sleep perspective.

The present study further contributes to the empirical foundations laid out by Jansson-Fröjmark and Jacobson (2021) by investigating the anxiolytic effects of CBT-I on individuals with co-morbid insomnia and GAD at a relatively lower dose of treatment (i.e. four sessions). Moreover, this study evaluates possible sleep-specific predictors of anxiety change that are directly targeted in a typical CBT-I protocol.

Method

Participants

Participants included patients aged 19–79 years with a *DSM-5* diagnosis of insomnia and GAD based on semi-structured interviews and self-report measures. Exclusion criteria were individuals that needed immediate psychiatric or medical care, psychiatric co-morbidities that prevented therapy adherence (e.g. addictions, bipolar and psychotic disorders), and sleep challenges (e.g. sleep apnoea, restless legs syndrome, circadian rhythm disorders, or irregular work schedules).

Intervention

Received four bi-weekly 60-minute sessions of CBT-I with a graduate student therapist. The first session focused on psychoeducation about sleep (e.g. sleep needs) and sleep systems (e.g. sleep drive, circadian rhythm). Treatment recommendations (e.g. sleep restriction therapy, stimulus control, consistent rise times, etc.) were provided based on personalized sleep data collected from daily diaries. The second session consisted of cognitive therapy (e.g. use of a thought record) and discussed the role of thoughts in maintaining sleep anxiety. The final two sessions were based on a case conceptualization approach (see Manber and Carney, 2015) and the content of the sessions varied depending on hypothesized mechanisms maintaining each individual participant's sleep problems.

Measures

Assessments were completed at baseline and post-treatment. Outcomes included the Insomnia Severity Index, Depression Anxiety Stress Scale, Daytime Insomnia Symptom Rumination Scale,

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Glasgow Sleep Effort Scale, and Consensus Sleep Diary (CSD). The CSD is a standardized, patient-informed sleep diary used to assess sleep on a night-by-night basis. For the sleep diaries, sleep variables, including total wake time, time in bed, and rise time were obtained.

Statistical analyses

IBM SPSS Statistics v26 was used to conduct the exploratory statistical analyses. Paired *t*-tests were employed to determine improvements in insomnia severity and anxiety from pre-treatment. Remission rates based on definitions in the literature were also calculated. Afterwards, a simultaneous multiple regression was conducted with subjective insomnia severity, age, sex, total wake time, total time in bed, rise time variability, sleep effort, and insomnia rumination as predictor variables and anxiety severity as the outcome variable.

Results

Baseline characteristics and completion rates

The sample ($n = 99$) was mainly single, middle-aged adults (65% female) of European descent (mean age = 41.1 years, $SD = 13.3$). For the purpose of this study, completion was defined as completing both baseline and post-treatment assessments. A total of 27 (27.3%) participants completed the baseline assessment only (non-completers) and 72 (72.7%) participants completed both the baseline questionnaires and post-treatment assessment (completers).

Preliminary exploratory analyses: changes in symptoms of insomnia and anxiety

A paired-samples *t*-test found that subjective insomnia severity scores reduced from pre- ($M = 20.40$, SD = 3.13) to post-treatment (M = 9.75, $SD = 5.01$), a statistically significant mean-difference of –10.65 (95% CI, –11.91 to –9.39), $t_{71} = -16.85$, $p < .001$, $d = 1.99$. Anxiety scores also lowered from pre- ($M = 10.64$, $SD = 8.82$) to post-treatment ($M = 5.83$, $SD = 6.88$), a statistically significant mean-difference of –4.81 (95% CI, –6.78 to –2.83), $t_{71} = -4.85$, $p < .001$, $d = .67$. Using a literature-based definition of insomnia remission (i.e. ISI < 10; Morin *et al.*, 2011), 55.6% of participants remitted from insomnia. Furthermore, based on a score of <10 on the DASS-21 anxiety subscale (Lovibond and Lovibond, 1995), 76.4% of participants reported normal or mild anxiety severity post-treatment.

Primary exploratory analyses: predictors of anxiety outcome

The full model of baseline anxiety, age, sex, insomnia change, total time in bed change, total wake time change, insomnia rumination change, rise time variability change, and sleep effort change to predict anxiety change scores was statistically significant, $R^2 = .712$, $F_{9,56} = 15.38$, $p < .001$, adjusted $R^2 = .666$. In terms of sleep-specific variables, changes in subjective insomnia severity ($\beta = .25$, $p = .02$) and symptom-focused rumination ($\beta = .22$, $p = .015$) were found to be significant. Specifically, a one-unit reduction in the ISI and DISRS predicted a .44 and .14 reduction in DASS-21 anxiety subscale, respectively. Younger age ($\beta = -.21$, $p = .007$) and lower baseline anxiety scores ($\beta = -.69$, $p < .001$) also predicted greater anxiety change at post-treatment. See Table 1 for a detailed depiction of relevant statistics.

Discussion

The present study contributes to the relative paucity of empirical evidence evaluating the role of CBT-I in a co-morbid GAD-I sample. Specifically, the results of the study demonstrated that a low dose of CBT-I led to a moderate reduction ($d = -.67$) in anxiety symptoms.

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Table 1. Multiple regression predicting anxiety change

Variable	*B*	95% CI for *B*		*SE B*	β	*R*	R^2
		LL	UL			.844***	.712***
Age	−.127**	−.218	−.037	.045	−.207**		
Sex	−1.212	−3.905	1.481	1.344	−.067		
BL Anxiety	−.659***	−.806	−.513	.073	−.689***		
ΔISI	.401*	.065	.738	.168	.249*		
ΔTWT	−.990	−2.401	.420	.704	−.142		
ΔTIB	.958	−.684	2.600	.820	.120		
ΔDISRS	.144*	.029	.259	.057	.223*		
ΔRTvar	−.410	−1.152	.332	.370	−.088		
ΔGSES	.175	−.304	.655	.239	.069		

*$p < .05$, **$p < .01$, ***$p < .001$. BL Anxiety, baseline anxiety (DASS-21 anxiety subscale); DISRS, Daytime Insomnia Symptom Rumination Scale; GSES, Glasgow Sleep Effort Scale; ISI, Insomnia Severity Index; RTvar, rise-time variability; TIB, time in bed; TWT, total wake time.

One novel aspect of this study was its examination of insomnia-related constructs targeted in CBT-I as predictors of post-treatment anxiety outcome. Results found that reductions in perceived insomnia severity and symptom-focused rumination predicted improvements in anxiety symptoms. There are a few possible reasons to explain why feeling better about sleep and spending less time thinking about how sleep problems will negatively affect daytime performance leads to less anxiety in this sample. For instance, in this study, the sample actively sought treatment for their insomnia; therefore, one parsimonious reason for insomnia severity being a predictor is that alleviation of their insomnia problems led to sleep being less of a pertinent concern (i.e. a key 'worry').

The finding that reductions in symptom-focused rumination (i.e. worries about how perceived sleep deficits, such as fatigue, will impact performance) predicts alleviation of anxiety symptoms can be explained in a couple of ways. First, rumination can be conceptualized as part of a broader construct of repetitive negative thinking (RNT) that captures ruminative and worry behaviours. Consequently, cognitive strategies that target ruminative behaviours about perceived sleep deficits and subsequent daytime functioning may contribute to this transdiagnostic process and relieve anxiety. Second, based on a cognitive model of insomnia, reductions in ruminative tendencies may implicate attentional biases being less likely to be deployed towards signs of sleep disruptions and negative interpretations of daytime symptoms that maintain and exacerbate distress (Harvey, 2002). Shifts in attentional biases towards less anxiety-provoking sleep stimuli may be an insomnia-specific pathway towards reduced anxiety.

Beyond insomnia-specific variables, younger adults with less severe anxiety symptoms also benefited more in terms of anxiety outcomes. The finding that older populations presenting with GAD tend to receive less benefits after psychological treatment is consistent with other research (Covin *et al.*, 2008). The results also implicate that when anxiety is significantly elevated, stand-alone sleep treatments may be insufficient. Consequently, GAD-specific treatments or taking a combination/sequential approach targeting both sleep and anxiety may be preferable in this case.

Future directions

There are components of the current exploratory study that would benefit from additional empirical rigour with larger sample sizes to further the research in this area. Although an open trial reflects real-world conditions well, ruling out competing explanations can be difficult. As such, a randomized controlled trial with treatment arms that offer alternative (e.g. CBT for GAD), combination, or sequential treatment would be helpful to identify who, in what context, and by which treatment, an individual with GAD-I most benefits from intervention.

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Methodologically, there is also utility in replicating this research using different empirically validated measurements. Although the DASS-21 anxiety subscale has been found to correlate highly with other common measures of GAD (e.g. GAD-7; Peters *et al*., 2021), there may be utility in employing other measures, such as the Penn State Worry Questionnaire. Finally, in terms of the specific study sample, participants intentionally sought out insomnia treatment; therefore, we cannot make inferences about those who preferred to receive treatment for GAD symptoms or concurrent treatment.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S1352465823000656

Data availability statement. The data that support the findings of this study are available on request from the corresponding author, P.L. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

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Author contributions. **Parky Lau:** Conceptualization (lead), Formal analysis (lead), Writing – original draft (lead), Writing – review & editing (equal); **Elisha Starick:** Project administration (equal), Writing – review & editing (equal); **Colleen Carney:** Conceptualization (equal), Funding acquisition (lead), Methodology (lead), Supervision (lead).

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Competing interests. The authors report there are no competing interests to declare.

Ethical standards. The research herein conforms to the Declaration of Helsinki with respect to ethical principles. This study has received ethics approval from Toronto Metropolitan University (REB 2019-294). All participants provided informed consent to take part in the study.

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