**Anxiolytic Impact of Cognitive Behavioural Therapy for Insomnia in Patients with Comorbid Insomnia and Generalized Anxiety Disorder**

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**Declaration of Interest**

The authors report there are no competing interests to declare.

**Abstract**

**Background:** Cognitive behavioral 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 GAD do not improve from standard GAD treatment.

**Aims:** To investigate the efficacy of CBT-I on anxiety and evaluate whether changes in sleep-specific variables predict anxiety outcomes.

**Methods:** Seventy-two participants presenting with insomnia and generalized anxiety disorder (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 comorbid 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 effects 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, insomnia, predictors of recovery, CBT

**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 (APA, 2023; Daley et al., 2009; Kyle et al., 2010). These nighttime complications are paired with perceived sleep deficits and subsequent daytime impairments – for example, fatigue, irritability, and difficulty with attention and concentration (Ustinov et al., 2010). Epidemiological research highlights the pervasive nature of chronic sleep disturbances, with approximately one in 10 individuals meeting diagnostic criteria for insomnia disorder (Garland et al., 2018; Roth, 2007) and nearly one out of three adults reporting at least one symptom of insomnia (Morin et al., 2006; Olfson et al., 2018).

Although insomnia is pernicious and disruptive as a standalone disorder, chronic sleep disturbances are also highly comorbid across numerous medical and psychiatric conditions (Sarsour et al., 2010). In fact, sleep disturbance is part of the diagnostic profile for several psychological disorders, including major depression, post-traumatic stress, and generalized anxiety disorder (APA, 2013). Given that sleep disturbances appear to be a transdiagnostic phenomenon, empirical efforts have been levied to evaluate the role of insomnia on the onset and trajectory of other disorders (e.g., depression and anxiety; Alvaro et al., 2013; Riemann & Voderholzer, 2003), and whether treatment of insomnia improves symptoms of the comorbid condition (Hertenstein et al., 2022). For example, research has found that cognitive behavioural therapy for insomnia (CBT-I) – the gold standard frontline treatment for chronic insomnia – produces an antidepressant effect in individuals with insomnia and depression comparable to pharmacotherapy (Carney et al., 2017; Manber et al., 2008). Sleep-specific changes, especially rumination about daytime insomnia symptoms, appear to play an important role in contributing to improvements in mood (Lau et al., 2022). These findings indicate that there is substantial utility in learning more about treating psychological disorders through an insomnia perspective.

Although some psychological afflictions such as depression have received significant empirical attention in the insomnia literature, 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 comorbid and mutually influence each other’s course and outcome (Herteinstein et al., 2019; Shanahan et al., 2014). Epidemiological studies indicate that 85-90% of individuals with GAD report dissatisfaction with their sleep and a majority (52-68%) experience moderate to severe insomnia (Bélanger et al., 2004; Brenes et al., 2009; Ferre Navarrete et al., 2017). Focusing on the inverse relationship, Ohayon et al. (1998) found that GAD was the most common anxiety disorder comorbid with insomnia. This finding is corroborated by other studies employing a structured interview format, which found that 9-13% of those diagnosed with insomnia also meet criteria for GAD (Breslau et al., 1996; Mellinger et al., 1985). Unsurprisingly, the collection of epidemiological literature suggests that sleep and anxiety are inextricably linked.

Given this relationship, one clinical implication that naturally follows is that treatment of one disorder may naturally lead to alleviation of the comorbid disorder. In behavioural sleep medicine, research has explored the role of treating sleep disturbances and its effects on anxiety. In one recent meta–analysis, Lee et al. (2023) investigated the impact of digital CBT-I on anxiety symptoms and found that the insomnia intervention had a moderate effect on anxiety severity. However, the samples included in this study were heterogenous and many of the included samples (16/22) reported subthreshold levels of anxiety. One limitation then is that we cannot extrapolate these effects to a clinical sample reporting more significant anxiety. Previous studies that specifically focused on an insomnia sample comorbid with GAD are relatively sparse and existing studies are limited in sample size (e.g., Belleville et al., 2016). There is one recent study that evaluated the efficacy of CBT-I on a larger sample of patients with insomnia disorder comorbid with GAD (Jansson-Fröjmark & 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 behavioral 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 likely contribute to the bidirectional 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. Indeed, polysomnography research conducted by Fuller et al. (1997) found that individuals with GAD took longer to fall asleep, had a smaller percentage of deep (slow-wave) sleep, and more frequent transitions into light sleep. Moreover, high-anxiety/worry subjects demonstrated a greater percentage of light sleep and more microarousals compared to their low-anxiety counterparts. Meta-analytic findings offer evidence in support of challenges in sleep continuity and depth and implicate the role of arousal system imbalances in anxiety disorders (Baglioni et al., 2016). Specifically, neuroimaging studies suggest that sleep deficits resulting from hyperarousal amplifies activity in brain regions associated with the ‘fear’ network (Simon et al., 2015; Yoo et al., 2007). Along with challenges associated with emotional processing and shared neuromolecular mechanisms (e.g., norepinephrine, adenosine, GABA receptors), these sleep-arousal pathways work in tandem to contribute to the development and maintenance of anxiety-related disorders (for a review, see Chellappa & Aeschbach, 2022).

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 (Ehring & Watkins, 2008). This sustained cognitive activation may contribute to subjective fatigue (Carney et al., 2014; Hare et al., 2019), 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 nighttime, contributing to greater levels of anxiety.

Overall, the research, though nascent, appears promising in regard to the utility of CBT-I in alleviating anxiety in individuals presenting with comorbid 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. For example, sleep restriction and stimulus control (a set of recommendations designed to strengthen the bed as a cue for sleep rather than wake) emphasize the consolidation of sleep through leveraging sleep-wake homeostasis and deconditioning physiological hyperarousal (Bootzin, 1972; Pigeon & Perlis, 2006). Subsequent improvements in sleep depth and quality in addition to reductions in repetitive negative thinking (Ballesio et al., 2021) may potentiate anxiety reductions.

The present study further contributes to the empirical foundations laid out by Jansson-Fröjmark & Jacobson (2022) by investigating the anxiolytic effects of CBT-I on individuals with comorbid 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. Specifically, we examined the role of total wake time, total time in bed, rise time variability, symptom-focused rumination, sleep effort, and subjective insomnia severity in predicting anxiety outcomes after CBT-I, in addition to age, sex, and baseline anxiety.

This unique contribution may be a particularly important avenue of research given that up to half of individuals with GAD do not show a clinically meaningful response to frontline therapeutic strategies. (Fisher, 2006) Consequently, developing an understanding of potential pathways to treatment improvement may prove to be a fruitful endeavor to refine interventions or offer viable alternatives should traditional treatment paths be less effective.

**Methods**

**Participants**

Participants included in this study (N=99) were part of an insomnia study at Toronto Metropolitan University (TMU). This is a report of secondary data of a larger clinical trial evaluating the role of cognitive reactivity and its effect on rates of relapse after treatment completion. The findings of this study are currently in preparation for publication. Those that met DSM-IV-TR (APA, 2000) diagnostic criteria for both current insomnia disorder (ID) and GAD based on a combination of semi-structured interviews and self-report measures were included in the present study. Participants were recruited from the through a combination of advertisement and referrals. All study procedures were approved by the TMU’s research ethics board (2019-294) and all participants provided written consent prior to participating in the study.

Participants who met the following eligibility criteria were included in the present study: (1) met Research Diagnostic Criteria for insomnia (Edinger et al., 2004), assessed using the Duke Structured Interview for Sleep Disorders (Edinger et al., 2009), with the insomnia complaint lasting 3 months or longer; (2) reported a score on the Insomnia Severity Index (Morin, 1993) > 14; (3) demonstrated a mean sleep efficiency (SE = [Total Sleep Time ÷ Time in Bed] x 100%) < 85 % on sleep diaries over a 2-week screening period; and (4) met DSM-IV-TR criteria for current Generalized Anxiety Disorder  (GAD), assessed using the Structured Clinical Interview for DSM-IV Axis I Disorders(SCID-I; First, Spitzer, Gibbon, & Williams, 2002); and (5) reported a score ≥ 10 on the Anxiety subscale of the Depression Anxiety and Stress Scales(DASS-21; Lovibond & Lovibond, 1995), suggestive of mild anxiety severity (Lovibond & Lovibond, 1995).

Individuals were excluded from the study if they met any of the following exclusion criteria: (1) met criteria for bipolar or psychotic disorders; (2) needed immediate psychiatric care (e.g., high risk of suicidality); (3) endorsed a history substance abuse over the past six months (4) had a medical condition which required immediate attention (e.g., acute cardiac symptoms); (5) met criteria for restless leg syndrome, sleep apnea, or a circadian rhythm disorder; or (6) endorsed a lifestyle with an irregular schedule (e.g., night shifts).

**Intervention**

Participants that met inclusion criteria received four biweekly 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, including sleep restriction therapy, stimulus control, and sleep hygiene 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 and the contents of the sessions varied depending on hypothesized mechanisms maintaining each individual participant’s sleep problems (Manber & Carney, 2015). For a full elaboration on the specific standardized treatment protocol, please refer to Edinger and Carney (2014). Graduate level therapists were trained and supervised by the principal investigator (CEC). CEC is an expert in sleep medicine and the delivery of CBT-I.

**Outcome Measures**

***Sleep Parameters***

Mean total wake time (TWT), time in bed (TIB), and risetime variability were obtained using the Consensus Sleep Diary (CSD; Carney et al., 2012). The CSD is a standardized sleep diary used to assess nightly sleep. Participants completed the CSD for a 2-week screening period, throughout treatment, and post-treatment.

***Self-Report Measures***

The Insomnia Severity Index(ISI) (Morin et al., 1993) is a reliable, valid, and recommended self-report measure for assessing perceived sleep difficulties. The ISI is a 7-item scale which assesses self-reported severity, satisfaction, distress, and impairment associated with insomnia symptoms.

The 21-item version of the Depression Anxiety Stress Scale(DASS-21; Lovibond & Lovibond, 1995) is a brief version of the DASS questionnaire. Research has supported the DASS-21 as a valid and reliable measure (Antony et al., 1998; Brown et al., 1997). For the purposes of this study, the anxiety subscale was utilized to measure severity of anxiety symptoms.

The Daytime Insomnia Symptom Rumination Scale (DISRS; Carney et al., 2013) was used to evaluate ruminative tendencies in insomnia populations. The DISRS was adapted from the Symptom-Focused Subscale of the Response Styles Questionnaire (Bagby & Parker, 2001; Nolen-Hoeksema & Morrow, 1991) and includes questions that focus on the frequency and intensity with which an individual repetitively thinks about their symptoms of insomnia when they are tired. The scale has demonstrated excellent internal consistency (α = .93) and construct validity, demonstrating associations with other theoretically linked sleep measures (Carney et al., 2013).

The Glasgow Sleep Effort Scale (GSES; Broomfield & Espie, 2005) is a 7-item self-report measure used to assess one’s direct and indirect efforts to fall asleep, such as expanding the sleep window or engaging in a ritual thought to induce sleep. Previous work supports the psychometric properties of the GSES and has demonstrated adequate internal consistency, convergent validity with measures of similar constructs, and ability to differentiate between good and poor sleepers (Broomfield & Espie, 2005).

**Procedures**

Study candidates were recruited through advertisements and physician referral. The coordinator assessed eligibility through a phone screen. Those who were preliminarily eligible to participate in the study were then scheduled for a screening interview. After providing their consent, participants completed a demographic and medical history form, the DSISD to assess for the presence of ID and other sleep disorders, as well as the SCID to assess for anxiety and comorbid psychiatric conditions.

Following this assessment, participants complete sleep diaries each morning for 14 consecutive days. Participants with a mean sleep efficiency of 84% or less were included in the study. Potential participants who met all entry criteria as described in the *Participants* section went on to complete a baseline measures package, which included the ISI, the DASS-21, and GSES.A flowchart of participants can be found in Figure 1.

**Figure 1**

*Flowchart of Participants*

Enrolled in larger CBT-I Trial (N=524)

Met study criteria for GAD and ID and completed baseline measures (*n*=99)

Enrolled Treatment

Enrollment

Post-Treatment Questionnaires

Did not complete treatment (*n*=27)

Completed post-treatment questionnaires (*n*=72)

Completed treatment (*n*=72)

**Statistical Analyses**

All analyses were conducted using IBM SPSS Statistics v26. Paired t-tests were conducted to examine changes in subjective insomnia severity and anxiety from beginning of treatment. Afterwards, a simultaneous multiple regression was conducted with the following variables: age, sex, subjective insomnia severity, total time in bed, total wake time, sleep effort, rise time variability, and rumination about insomnia symptoms as predictor variables and anxiety severity as the outcome variable. Complete case analysis was used to handle missing data.

**Results**

**Baseline Characteristics and Completion Rates**

The sample was primarily composed of European adults (65% female). See Table 1 for detailed sample descriptive statistics. Study completion was defined as completing assessments at baseline and post-treatment. A total of 27 (27.3%) were non-completers and 72 (72.7%) were completers

**Table 1**

*Demographic Characteristics of Participants*

|  |  |
| --- | --- |
| **Variable** | **Mean (SD) or Count (%)** |
| Age | 41.13 (13.27) |
| Sex |  |
| Male | 34 (34.3%) |
| Female | 64 (64.6%) |
| Ethnicity |  |
| African Canadian | 3 (3.0%) |
| Caribbean Canadian | 1 (1.0%) |
| East/Southeast Canadian | 2 (2.0%) |
| European Canadian | 57 (57.6%) |
| Latin/Central/South Canadian | 5 (5.1%) |
| South Asian Canadian | 10 (10.1%) |
| West Asian/Arab Canadian | 3 (3.0%) |
| Other | 18 (18.2%) |
| Marital Status |  |
| Single | 39 (39.4%) |
| Married/Common-law | 48 (48.5%) |
| Live-in partner (less than 2 years) | 2 (2.0%) |
| Divorced | 7 (7.1%) |
| Separated | 2 (2.0%) |
| Widowed  | 1 (1.0%) |
| Living Arrangement  |  |
| Living alone | 27 (27.3%) |
| Living with spouse/partner and children | 24 (24.2%) |
| Living with friend(s)/roommate(s) | 9 (9.1%) |
| Living with spouse/partner | 25 (25.3%) |
| Living with family member(s) | 13 (13.1%) |
| Employment Status |  |
| Full-time | 62 (62.6%) |
| Part-time | 16 (16.2%) |
| Not working | 20 (20.2%) |

**Preliminary Exploratory Analyses: Changes in Symptoms of Insomnia and Anxiety**

A paired-samples t-test evaluated changes in insomnia severity scores and anxiety symptom scores before and after receiving CBT-I. Subjective insomnia severity scores decreased 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 decreased 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 & Lovibond, 1995), 76.4% of participants reported normal or mild anxiety severity post-treatment. Additional sleep-related changes post-treatment and their effect sizes can be found in Table 2.

**Table 2**

*Paired T-test Results Comparing Pre- and Post-Intervention Means, Standard Deviations, and Cohen’s d Across Sleep Variables*

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Variable** | **Baseline** | **Post-Tx** | ***t*** | ***p*** | **Cohen’s *d*** |
| ***M*** | ***SD*** | ***M*** | ***SD*** |  |  |
| ISI | 20.40 | 3.13 | 9.75 | 5.01 | 16.85 | < .001\*\*\* | 1.99 |
| DISRS | 54.88 | 10.25 | 43.86 | 13.47 | 7.07 | < .001\*\*\* | .83 |
| GSES | 10.61 | 2.60 | 5.97 | 3.28 | 11.96 | < .001\*\*\* | 1.42 |
| FSS | 45.09 | 9.88 | 33.93 | 12.42 | 8.23 | < .001\*\*\* | .97 |
| TWT | 2.88 | 1.31 | 1.13 | .51 | 11.65 | < .001\*\*\* | 1.42 |
| TIB | 8.44 | 1.12 | 7.33 | .86 | 8.38 | < .001\*\*\* | 1.02 |
| RT var | 3.00 | 2.51 | 2.57 | 1.94 | 1.94 | .057 | .24 |

*Note.*

 \*\*\*p < .001

 DISRS = Daytime Insomnia Symptom Rumination Scale, FSS = Fatigue Severity Scale, GSES = Glasgow Sleep Effort Scale, ISI = Insomnia Severity Index, TIB = time in bed, TWT = total wake time, RT var = rise time variability

**Primary Exploratory Analyses: Predictors of Anxiety Outcome**

The full model of age, sex, baseline anxiety, subjective insomnia severity change, total wake time change, total time in bed change, symptom-focused rumination change, rise time variability change, and sleep effort change to predict anxiety change scores was statistically significant, *R2*= .712, *F*(9, 56) = 15.38, *p* < .001, adjusted *R*2 =.666. In terms of sleep-specific variables, changes in subjective insomnia severity, β = .25, *p* = .02, and symptom-focused rumination, β = .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 (β = -.21, *p* = .007) and lower baseline anxiety scores (β  = -.69, *p* <.001) were also predictive of greater anxiety change at post-treatment. See Table 3 for a detailed depiction of relevant statistics.

**Table 3**

*Multiple Regression Predicting Anxiety Change*

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Variable** | ***B*** | **95% CI for *B*** | ***SE B*** | **β** | ***R*** | ***R2*** |
|  |  | ***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 |  |  |

*Note.*

 \**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 = risetime variability, TIB = time in bed, TWT = total wake time.

**Discussion**

The present study contributes to the relative paucity of empirical evidence evaluating the role of CBT-I in a comorbid 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. These results are comparable to meta-analytic studies on effect sizes of existing psychological treatments for GAD (Cuijpers et al., 2014). Moreover, the study also demonstrated significant reductions in subjective insomnia severity (*d* =1.99) in addition to other insomnia-related constructs (*d* = .24 to 1.42) indicating that CBT-I remains a robust sleep intervention in different comorbid populations, consistent with previous research (Wu et al., 2015).

One novel aspect of this study was its examination of insomnia-related constructs targeted in CBT-I as predictors of post-treatment anxiety outcome. This research supports further understanding of the driving factors of anxiety change despite employing an insomnia treatment. 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’). Given that GAD is characterized by excessive worry, reductions in perceived insomnia severity may naturally lead to reduced anxiety.

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 (Ehring & Watkins, 2008). RNT has been empirically regarded as a transdiagnostic construct that contributes to the development and maintenance of various psychological afflictions, such as depression, anxiety, and obsessive-compulsive disorders (e.g., Wahl et al., 2019). 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 (Espie, 2007; 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 researchers hypothesized that more ingrained worry behaviours may be a root cause of this relationship (e.g., Stanley et al., 1996). The results also implicate that when anxiety is significantly elevated, standalone 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.

There are components of the current study that would benefit from additional empirical rigour 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. 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, which is designed to capture the dominant feature of worry in GAD (Meyer et al., 1990). 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. Finally, replications with larger sample sizes would be helpful to further ground the findings in high powered designs.

In sum, the current study supports that CBT-I remains a robust intervention for insomnia in a sample of GAD-I patients, with probable effects on anxiety. Moreover, reductions in perceived insomnia severity and daytime rumination about insomnia symptoms predicted anxiety relief. That is, perceiving sleep to be less of a concern and being less focused on the consequences of sleep deficits appear to be driving reductions in anxiety in this sample. A randomized controlled trial would be helpful to elucidate the relative benefits of different therapy approaches. For example, the finding that older age and more severe anxiety is associated with less of an anxiolytic effect after CBT-I suggests that clinicians may need to target anxiety directly in addition to the insomnia in those who are older or report more severe anxiety symptomatology.

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