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Self-efficacy in insomnia symptom management after digital CBT-I mediates insomnia severity during the COVID-19 pandemic

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Abstract

Study Objectives: Digital cognitive behavioral therapy for insomnia (dCBT-I) can reduce acute insomnia and depressive symptoms and prevent symptom recurrence. The current study evaluated self-efficacy to manage insomnia symptoms as a potential mediator of the relationship between prior dCBT-I and subsequent insomnia and depressive symptoms assessed during the coronavirus 2019 (COVID-19) pandemic.

Method: Participants were 208 adults who completed a randomized controlled trial of dCBT-I versus sleep education in 2016–2017 and also completed self-report assessments of insomnia, depression, and self-efficacy to manage insomnia symptoms in May 2020, five weeks into state-wide COVID-19 stay-at-home orders. Regression and mediation analyses were used to evaluate the extent to which self-efficacy accounted for the relationship between treatment condition and improvement in insomnia and depressive symptoms from pre-treatment to COVID-19 follow-up.

Results: Prior dCBT-I predicted improved self-efficacy in managing insomnia symptoms, and self-efficacy accounted for 49% of treatment-related improvement in COVID-era insomnia symptoms and 67% of treatment-related improvement in COVID-era depressive symptoms.

Conclusions: This study affirms the importance of self-efficacy as a key intervention outcome and potential mechanism by which dCBT-I predicts future sleep and mental health. Future studies that evaluate the role of self-efficacy in treatment effectiveness and resilience can provide additional clues about how to optimize dCBT-I for maximum benefit to public health.

Keywords

cognitive behavioral therapy for insomnia (CBT-I); randomized controlled trial (RCT); treatment mechanisms; self-efficacy; insomnia; depression; coronavirus (COVID-19)

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Disclosures

The authors have no conflicts of interest to disclose.

Introduction

The health consequences of the 2019 coronavirus disease (COVID-19) pandemic have been pervasive, reaching far beyond the direct effects of viral infection. Given that stress is a robust precipitant of insomnia (Bastien et al., 2004; LeBlanc et al., 2009; Singareddy et al., 2012), it is unsurprising that insomnia has been rampant during this global pandemic (Kokou-Kpolou et al., 2020; Voitsidis et al., 2020; Zitting et al., 2021), along with general declines in health and social functioning (Pierce et al., 2020; Xiong et al., 2020; Zhang et al., 2020).

Despite the seriousness and pervasiveness of the COVID-19 pandemic, its impact has not been experienced uniformly across individuals. We recently demonstrated that those who previously received treatment for insomnia (digital Cognitive Behavioral Therapy for Insomnia; dCBT-I) pre-COVID-19 were half as likely to exhibit a resurgence of clinically significant insomnia compared to those who only received sleep education (Cheng, Casement, et al., 2020). Additionally, those who received dCBT-I pre-COVID-19 also reported lower COVID-19 related stress, less depression, and better health during the pandemic. In short, these data provided evidence that insomnia treatment not only improves health and functioning acutely but may also bolster longer-term resilience. This is consistent with studies that have shown that insomnia treatment can prevent future incidence of depression (Batterham et al., 2017; Cheng et al., 2019).

One important line of inquiry in this area of research is establishing the mechanisms by which insomnia treatment protects against future adverse outcomes. A promising candidate is self-efficacy in managing insomnia symptoms. Self-efficacy refers to confidence in ability to navigate a challenge to produce a desired outcome (Bandura, 1977, 1986). Indeed, self-efficacy is a critical factor in motivating behavior change; it predicts behavior initiation, the amount of effort expended, and behavioral persistence in the face of adverse circumstances. In a clinical context, self-efficacy predicts treatment outcomes across domains (O'Leary, 1985; Schwarzer & Fuchs, 1996), including all-cause mortality (Assari, 2017). This has been replicated with CBT-I research indicating that self-efficacy predicts adherence to CBT-I (Bouchard et al., 2003; Hebert et al., 2010; Horsch et al., 2015; Ruiter Petrov et al., 2014), which in turn enhances treatment gains. Additionally, studies have also have shown that insomnia treatment increases self-efficacy (Harris et al., 2007; Lovato et al., 2014), including when the treatment is delivered digitally (Siengsukon et al., 2020). Indeed, CBT-I specifically targets self-efficacy via a focus on relapse prevention. Together, these data suggest that self-efficacy plays an important role in the success of CBT-I; however, no studies have examined if self-efficacy in managing insomnia symptoms following CBT-I is a protective mechanism against future adverse outcomes.

In this study, we followed individuals who received either dCBT-I or sleep education three to four years prior to the COVID-19 pandemic. We evaluated self-efficacy in managing insomnia symptoms as a candidate mechanism for protecting against insomnia and depression during COVID-19. We hypothesized that those who completed dCBT-I would have higher self-efficacy in managing insomnia symptoms than those who completed sleep education. We further hypothesized that self-efficacy would partially account for the

effects of dCBT-I on later insomnia and depressive symptoms during the early months of the COVID-19 pandemic.

Method

Participants

Participants for this study were recruited from a previous randomized controlled trial (NCT02988375) testing the efficacy of self-guided dCBT-I compared to a sleep education control in treating insomnia (Cheng et al., 2018) and preventing incident depression (Cheng et al., 2019). Participants in the SPREAD trial were enrolled between 2016 and 2017, with a final sample of 358 in the dCBT-I condition and 300 in the control condition who completed post-treatment assessments. Those in the dCBT-I condition completed 6 sessions of CBT-I through the Sleepio platform (Espie, Kyle, Williams, et al., 2012). Sessions were directed by an animated "virtual therapist" who reviews and guides progress with the participant. Individuals randomized to the online sleep education condition received six weekly e-mails based on the NIH guide to healthy sleep (National Institutes of Health, 2011). Eligibility for the SPREAD trial was assessed via an online screener. This approach has been validated against clinician-administered diagnostic interviews (Espie, Kyle, Hames, et al., 2012; Kessler et al., 2010). Eligible participants met criteria for insomnia disorder based on the DSM-5: insomnia symptoms present on 3 or more days per week, with significant distress or impairment, and of at least 3 months duration. Participants were excluded from the SPREAD trial if they reported a diagnosis of any untreated sleep disorders other than insomnia (e.g., obstructive sleep apnea, restless legs, narcolepsy, etc.), and bipolar or seizure disorders. Because the SPREAD trial included a depression prevention aim, individuals with high depression chronicity (self-reported daily or near daily depressed mood and anhedonia) were excluded (for additional details, see Cheng et al., 2018).

All 658 participants in the SPREAD trial were eligible for this follow-up study. The recruitment plan targeted enrollment at 200 participants, which would achieve sufficient power (0.8) to detect a moderate effect size for each hypothesis test. Email invitations were sent during the last week of April 2020, five weeks into the Michigan state-wide stay-at-home order, with approximately 40,000 cases and 3800 deaths across the state. Enrollment was closed in the first week of May when the targeted sample size was achieved. The final sample included 208 participants (dCBT-I: $n = 102$; control: $n = 106$). Study procedures were approved by the Henry Ford Health System Institutional Review Board (#13784), and all participants provided informed consent prior to participating in the study.

Measures

Insomnia and depression symptom severity during COVID-19.—Symptoms of insomnia were assessed using the 7-item Insomnia Severity Index (ISI; Bastien et al., 2001), with higher scores indicating increased insomnia severity (range 0 – 28). Scores of 15 or higher on the ISI indicate moderate to severe insomnia symptoms. Depression was assessed using the 16-item self-report Quick Inventory of Depressive Symptomatology (QIDS-SR_{16}; Rush et al., 2003), a reliable and validated instrument for measuring depression symptoms that is commonly used in clinical trials. To capture the unique impact of COVID-19

beyond baseline risk of insomnia and depression, the direct effect in the mediation analyses utilized a difference score where pre-treatment ISI and QIDS-SR_{16} scores were subtracted from scores during COVID-19. We opted to account for pre-treatment scores instead of post-treatment scores to avoid redundancy between the outcome variable and the predictor (i.e. treatment condition). This approach also allowed temporal precedence of variables to support inferences via mediation.

Self-efficacy in managing insomnia symptoms.—Self-efficacy in management of insomnia symptoms following prior treatment was assessed in accordance with the confidence ruler that is used in Motivational Interviewing (Miller & Rollnick, 2012). The prompt was, "*On a scale of 1 to 10, how confident are you about being able to manage insomnia symptoms?*" The lowest response (1) was anchored with "Not at all confident" and the highest response (10) was anchored with "Most confident".

Additional descriptive measures.—The impact of COVID-19 on daily life was assessed with the Coronovirus Impact Scale (Kaufman & Stoddard, 2020), a research tool assembled by the Office of Behavioral and Social Sciences Research at the National Institutes of Health. The impact of COVID-19 is rated for seven dimensions of daily life (i.e., routines; income/employment; access to food, medical care, and mental health care; social support; pandemic-related stress; familial stress and discord; COVID diagnosis) on four-point Likert scales (0 = no change, 3 = severe). The scale has adequate internal consistency (α=.64–.75) and validity, with mean scores ranging from 9.2–12.0 across five validation samples that consisted of primary caregivers (https://psyarxiv.com/kz4pg/).

Analytical approach

First, descriptive statistics were used to characterize the demographic and symptom characteristics of the sample. Potential group differences were evaluated using chi-square analysis for sex, and independent sample t-tests for age, pre-treatment ISI and QIDS scores, the self-efficacy score, and the CIS score. Based on reviewer suggestions, analyses were also performed to evaluate potential group differences in treatment compliance, moderate-to-severe insomnia at post-treatment, insomnia remission, and access to medical and mental health care during COVID-19. The null hypothesis of no group difference for demographic and pre-treatment symptom characteristics was accepted at a conservative threshold of $p > .10$ to minimize possible type II error (i.e., false negative).

The hypotheses were tested using mediation analyses conducted in accordance with procedures outlined by Fairchild and MacKinnon (2009). Specifically, three regression models were conducted for mediation analyses with insomnia symptoms (see Figure 1): 1) the direct effect of the predictor (treatment condition) on the outcome variable (difference in pre-treatment insomnia and insomnia during COVID-19); 2) the effect of the predictor (treatment condition) on the proposed mediator (confidence in managing insomnia symptoms); and finally 3) the effect of the mediator on the outcome variable. The indirect (i.e., mediated) effect of the predictor on the outcome variable was tested using the product of the α and β parameter estimates. Three parallel regression models were conducted for mediation analyses with depressive symptoms.

Significance testing of mediation analyses was conducted using confidence intervals estimated using the PRODCLIN method implemented in R (Tofighi & MacKinnon, 2011). This method can be more accurate than traditional significance tests because it does not assume a normal distribution, which allows for asymmetric confidence intervals (MacKinnon et al., 2002, 2004). Statistical significance was determined if the 95% CI for the indirect effect did not include zero. The proportion of the mediated effect was calculated using a ratio of the indirect effect to the total effect.

Given that research conducted during a global pandemic may be vulnerable to selection bias (Sullivan, 2020; Q. Zhao et al., 2020), we utilized sampling weights for all analyses to mitigate differences in the probability of selection into the study relative to the original population of SPREAD trial participants. Sampling weights equal to the reciprocal of the selection probability in each condition were utilized to balance the probability of selection based on insomnia severity following treatment in the SPREAD trial. The final weighted mean (9.8 ± 5.7 SD) did not differ significantly from the population mean (10.4 ± 5.8 SD), suggesting that selection bias was likely minimal.

Results

Descriptive results

The final sample included 208 participants (dCBT-I: $n = 102$; control: $n = 106$; see Table 1 for a summary of sample characteristics by group). Comparison of the final sample in this study to those in the SPREAD trial revealed no differences in treatment compliance [$t(840) = 1.104$, *ns*], moderate-to-severe insomnia at post-treatment [$\chi^2(1) = 0.581$, *ns*], insomnia remission at post-treatment [$\chi^2(1) = 2.27$, *ns*], or access to medical and mental health care during COVID-19 [respectively, $t(204.16)=0.316$, *ns; t*$(205.62)=0.374$, *ns*]. In this sample, Both the control and dCBT-I conditions were similarly impacted by the COVID-19 pandemic, although people in the dCBT-I condition reported higher self-efficacy in managing insomnia symptoms during the pandemic compared to those in the control group (pathway α: B = 0.43 ± 0.14 SE, $p < .01$).

A previously published analysis demonstrated that insomnia symptoms during the pandemic were on average 2.7 points lower in those who previously received dCBT-I compared to the SE group (Cheng, Casement, et al., 2020). The odds of moderate to severe insomnia during COVID-19 was approximately 50% lower in those who received dCBT-I (probability = 23.9%) relative to SE (probability = 38.0%) (Cheng, Casement, et al., 2020).

Self-efficacy as a mediator of insomnia severity during COVID-19

The first regression in the mediation analysis showed a significant direct effect wherein those who received dCBT-I before COVID-19 exhibited less severe insomnia symptoms during the COVID-19 pandemic compared to those who received SE (pathway c: B = −0.51 ± 0.14 SE, $p < .001$). Prior experience with dCBT-I was also associated with greater self-efficacy in managing insomnia symptoms during COVID-19 compared to the SE group (pathway α: B = 0.43 ± 0.14 SE, $p < .01$). The third regression indicated that self-efficacy in managing insomnia remained a significant protective factor against more severe insomnia

(pathway β: B = −0.58 ± 0.06 SE, $p < .001$), while controlling for treatment condition (pathway c': B = −0.26 ± 0.12 SE, $p < .05$). The indirect effect (α × β) of self-efficacy was estimated to be −0.25, 95% CI [−0.40, −0.10]. As the CI did not overlap with zero, these results indicated a significant indirect effect in which self-efficacy mediated the effect of dCBT-I on insomnia severity during COVID-19. Together, these results indicate that just under half (49%) of the protective impact of dCBT-I can be explained by self-efficacy in managing insomnia symptoms.

Self-efficacy as a mediator of depression severity during COVID-19

Analyses indicated that the direct effect of treatment condition on QIDS-SR_{16} did not reach statistical significance (pathway c: B = −0.21 ± 0.14 SE, $p = .13$); however, as testing the significance of an indirect effect does not require a direct effect (Hayes, 2009; MacKinnon et al., 2000; Shrout & Bolger, 2002; X. Zhao et al., 2010), we continued to test for a significant indirect pathway from treatment condition through self-efficacy. As demonstrated above, prior experience with dCBT-I was associated with greater self-efficacy in managing insomnia symptoms (pathway α: B = 0.43 ± 0.14 SE, $p < .01$). Finally, self-efficacy in managing insomnia remained a significant protective factor against depressive symptoms during the pandemic (pathway β: B = −0.33 ± 0.07 SE, $p < .001$), while controlling for treatment condition (pathway c': B = −0.07 ± 0.13 SE, $p = .60$). The indirect effect (α × β) of self-efficacy was estimated to be −0.14, 95% CI [−0.24, −0.05]. As the CI did not overlap with zero, these results indicated a significant indirect effect in which change in confidence mediated the treatment effect of dCBT-I on insomnia severity. These results indicated that 67% of the protective impact of dCBT-I on COVID-era depressive symptoms can be explained by self-efficacy in managing insomnia symptoms.

Discussion

This aim of this study was to examine self-efficacy in managing insomnia symptoms as a candidate mechanism in the protective effect of prior dCBT-I on insomnia and depression during the COVID-19 pandemic. Results from this study provide the first evidence that improved self-efficacy in managing insomnia symptoms associated with dCBT-I is a potential mechanism in protecting against insomnia and depression during COVID-19. Specifically, improved self-efficacy from dCBT-I accounted for approximately half of the protection from COVID-era insomnia symptoms, and two-thirds of the relationship between treatment condition and COVID-era depressive symptoms. The implications of these results are that dCBT-I may have long-term protective effects against insomnia and depression because it is associated with increases in perceived control over insomnia.

Models of health behavior — including the Unifying Theory of Behavioral Change (Bandura, 1977), Transtheoretical Model of Behavior Change (Prochaska & DiClemente, 1983), and Theory of Planned Behavior (Ajzen, 1985) — posit that improved self-efficacy is a key mechanism of effective behavioral intervention (Mead & Irish, 2020). Furthermore, self-efficacy predicts behavior initiation and persistence in the face of adverse circumstances. In the context of COVID-19, increased self-efficacy in managing insomnia may enhance initiation of strategies taught in CBT-I (e.g., sleep restriction and stimulus

control) in response to sleep disruption due to COVID-19. When implemented, these strategies would serve as guardrails preventing the freefall of worsening insomnia symptoms and the eventual relapse of insomnia disorder.

These results are also significant given that insomnia is generally a persistent and recurrent disorder (Buysse et al., 2008; Morin et al., 2020; Morphy et al., 2007). Of those who complete CBT-I, which is the gold-standard treatment for insomnia (Qaseem et al., 2016; Riemann et al., 2017), approximately 60% will experience disorder recurrence within 6 months (Morin et al., 2009). The onset and recurrence of insomnia has previously been related to physiological and cognitive hyperarousal, which may be exacerbated by stressors such as the COVID-19 pandemic (Bonnet & Arand, 2010; Fernández-Mendoza et al., 2010; Perlis et al., 1997; Riemann et al., 2010). Though it is unclear what impact CBT-I has on hyperarousal as a predispositional factor for insomnia, these results suggest that increasing self-efficacy in managing insomnia may be an important mechanism in counteracting or protecting against resurgent insomnia in the face of future stressors. Future studies should examine if treatment-related increases in self-efficacy precede and predict decreases in hyperarousal, and/or result from reductions in arousal related to behavioral changes in CBT-I (e.g., stimulus control, sleep opportunity restriction). Although the measurement of self-efficacy at a single time-point in this study does not allow for predictive modeling of the longitudinal relationships between self-efficacy and hyperarousal, understanding the relationships between these proposed mechanisms could help optimize CBT-I efficacy.

Insomnia also more than doubles the risk of later depression incidence (Baglioni et al., 2011; Hertenstein et al., 2019; Li et al., 2016). There are a range of potential mechanisms by which insomnia may contribute to depression risk, including the effect of sleep and circadian disruption on reward processing (Boland et al., 2020; Casement et al., 2016), increasing rumination (Ballesio et al., 2019; Cheng, Kalmbach, et al., 2020) and other maladaptive emotion regulation (Baglioni et al., 2010; Schmidt et al., 2011). These mechanisms may be closely related to factors that promote self-efficacy, including anticipation of successful goal achievement, memory of past success and competence, and perceived ability to manage stressors. Likewise, enhanced anticipation of goal achievement, memory of past accomplishments, and perceived ability to manage stressors may reduce insomnia symptoms. Furthermore, increased self-efficacy may reduce distress and rumination, which would protect against depression. For example, self-efficacy has been established as a strong predictor of improved mental health and achievement outcomes in college students (Varghese et al., 2015) who are commonly at risk for depression (Furr et al., 2001). A treatment study also showed that higher self-efficacy in managing negative automatic cognitions following CBT was predictive of lower relapse in depressive episodes (Kavanagh & Wilson, 1989), and a study of high-school students found that self-efficacy distinguished between resilient and maladapted adolescents (Hamill, 2003). The results in this study are consistent with this literature: self-efficacy is an important predictor of functioning and may protect individuals against insomnia and depression during COVID-19.

The primary strength of this study is the examination of long-term resilience following a randomized controlled trial of dCBT-I. Opportunities to evaluate long-term protective effects of treatment in the context of stressors are very limited, and such studies are non-existent for

trials of dCBT-I. The inclusion of pre-treatment insomnia symptoms in our statistical models is an additional strength, making it unlikely that insomnia severity prior to dCBT-I can account for the mediating effect of self-efficacy on later insomnia severity. This study also included a representative sample of participants from the original trial (Cheng, Casement, et al., 2020) with a sufficient sample size for reliable hypothesis tests.

The primary limitations of the present study are the measurement of self-efficacy at a single time-point, and the potential that unmeasured factors (e.g., use of behavioral strategies to manage insomnia) can account for the relationship between treatment condition and symptoms of insomnia and depression during COVID-19. We were unable to adjust for self-efficacy prior to treatment, or evaluate the degree of change in self-efficacy during or after the initial dCBT-I trial. Notably, participant debriefing following the initial dCBT-I trial did not identify dCBT-I as the "active" treatment or sleep education as the control condition, which reduces the likelihood that expectancy bias in favor of dCBT-I could account for higher self-efficacy to manage insomnia in this COVID-19 follow-up study. A second limitation is the reliance on a single-item measure of self-efficacy. Previous studies indicate that single-item measures of self-efficacy predict CBT-I adherence (Bouchard et al., 2003; Ruiter Petrov et al., 2014) and track closely with multi-item scales of task-related and self-regulation self-efficacy ($r = 0.72$ and 0.73, respectively; [Bouchard et al., 2003]). Our measure of self-efficacy (i.e., "On a scale of 1 to 10, how confident are you about being able to manage insomnia symptoms?") is a more global and temporally non-specific measure than the single-item measures used in previous CBT-I studies (e.g., "Judge how confident you are, right now, that you can perform all the tasks that the treatment involves for the coming week?"[Bouchard et al., 2003]; "If I wanted to follow treatment recommendations, it would be easy for me to do so tonight." [Ruiter Petrov et al., 2014]), but it is based on similar models of health behavior that emphasize self-efficacy as a key mechanism of behavior change. Although the results in this study suggest that a 1-point change in self-efficacy (on a 10-point scale) may be clinically meaningful, further research is needed to establish general thresholds for clinically meaningful improvement when self-efficacy is assessed with single- or multi-item scales. A third and final limitation of the present study is that self-selected participation may have resulted in sampling bias, particularly during the COVID-19 pandemic. The sample here is representative of the sample in the original SPREAD trial, and generalization should be tempered prior to further research.

Conclusion

Our previous report demonstrated that the benefits of dCBT-I are robust to time and stressor exposure (Cheng, Casement, et al., 2020), and the present study indicates that these benefits can be accounted for by improved self-efficacy to manage insomnia symptoms.

Behavioral change, including sleep opportunity restriction and stimulus control, are often considered the "active" components of CBT-I (Edinger et al., 2020), but perceived efficacy is foundational to these and other planned behavioral changes. This study affirms the importance of self-efficacy as a key intervention outcome and potential mechanism by which dCBT-I predicts future sleep and mental health. Future studies that evaluate the role of

self-efficacy in treatment effectiveness and resilience can provide additional clues about how to optimize dCBT-I for maximum benefit to public health.

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Data availability statement

The data that support the findings of this study are available from the corresponding author, PC, upon reasonable request.

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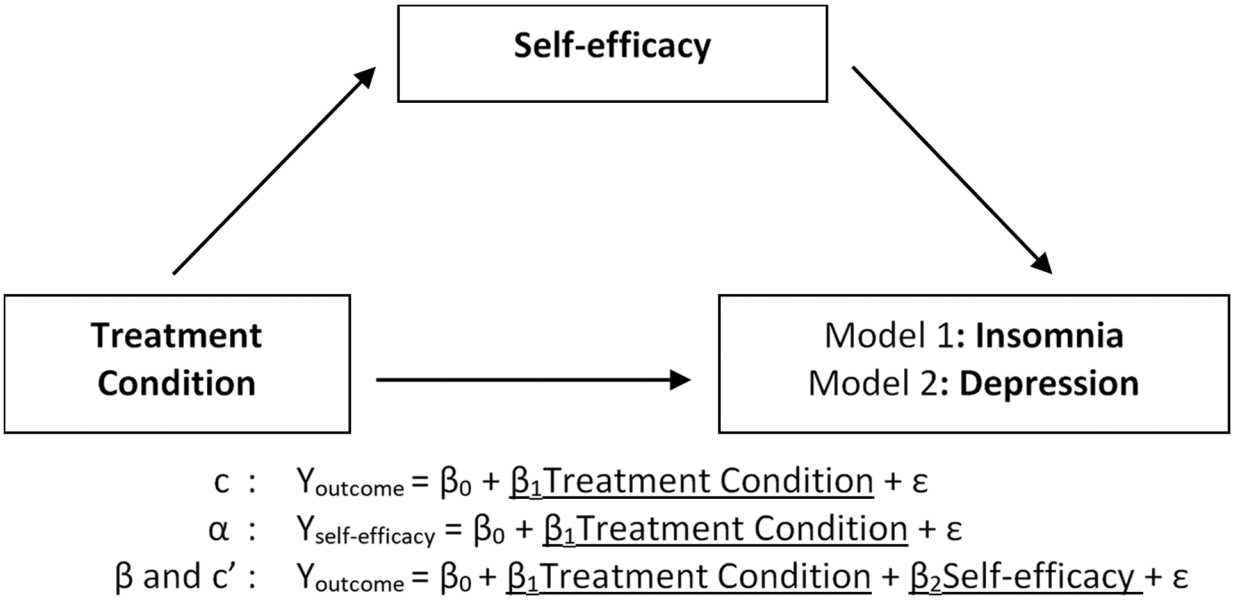

Figure 1.
Mediation model

Table 1.

Baseline sample characteristics by group.

	Control (*n*=106)	**dCBT-I (*n*=102)**
Age (M ± SD)	44.7 ± 14.2	44.6 ± 14.1
Sex (Female)	84.0%	72.5%
Pre-treatment ISI (M ± SD)	17.0 ± 4.1	18.0 ± 3.8
Pre-treatment QIDS-SR$_{16}$ (M ± SD)	11.1 ± 4.1	10.2 ± 4.6
Self-efficacy in managing insomnia	5.5 ± 2.1	6.5 ± 2.4
CIS (M ± SD)	12.1 ± 5.3	11.4 ± 4.3

dCBT-I = digital Cognitive Behavioral Therapy for Insomnia; CIS = Coronavirus Impact Scale. No group differences in demographic or pre-treatment characteristics were detected at $p < .10$.