

Henry Ford Health

Henry Ford Health Scholarly Commons

Sleep Medicine Articles

Sleep Medicine

11-29-2020

Digital Cognitive Behavioral Therapy for Insomnia Promotes Later Health Resilience During the Coronavirus Disease 19 (COVID-19) Pandemic

Philip Cheng

Henry Ford Health, PCHENG1@hfhs.org

Melynda D. Casement

David A. Kalmbach

Henry Ford Health, DKalmba1@hfhs.org

Andrea Cuamatzi-Castelan

Henry Ford Health, ACuamat1@hfhs.org

Christopher L. Drake

Henry Ford Health, cdrake1@hfhs.org

Follow this and additional works at: https://scholarlycommons.henryford.com/sleepmedicine_articles

Recommended Citation

Cheng P, Casement MD, Kalmbach DA, Castelan AC, and Drake CL. Digital Cognitive Behavioral Therapy for Insomnia Promotes Later Health Resilience During the Coronavirus Disease 19 (COVID-19) Pandemic. Sleep 2020.

This Article is brought to you for free and open access by the Sleep Medicine at Henry Ford Health Scholarly Commons. It has been accepted for inclusion in Sleep Medicine Articles by an authorized administrator of Henry Ford Health Scholarly Commons.

Digital Cognitive Behavioral Therapy for Insomnia Promotes Later Health Resilience During the Coronavirus Disease 19 (COVID-19) Pandemic

Philip Cheng^{1}, Melynda D. Casement², David A. Kalmbach¹, Andrea Cuamatzi Castelan¹, Christopher L. Drake¹*

¹ Sleep Disorders and Research Center, Henry Ford Health System, 2779 West Grant Blvd, Detroit, MI, USA

² Department of Psychology, University of Oregon, 1451 Onyx Street, Eugene, OR 97403 USA

* Corresponding Author: Philip Cheng, PhD

PC had access to all data from the study, and also had complete freedom to direct analyses and reporting of results without influence from funders

Email: pcheng1@hfhs.org

Tel: 248-344-7361

Address: 39450 West 12 Mile Road, Novi, MI 48377

Abstract

Study Objectives: Stressful life events contribute to insomnia, psychosocial functioning, and illness. Though individuals with a history of insomnia may be especially vulnerable during stressful life events, risk may be mitigated by prior intervention. This study evaluated the effect of prior digital cognitive-behavioral therapy for insomnia (dCBT-I) versus sleep education on health resilience during the COVID-19 pandemic.

Methods: COVID impact, insomnia, general- and COVID-related stress, depression, and global health were assessed in April 2020 in adults with a history of insomnia who completed a randomized controlled trial of dCBT-I ($n = 102$) versus sleep education control ($n = 106$) in 2016-2017. Regression analyses were used to evaluate the effect of intervention conditions on subsequent stress and health during the pandemic.

Results: Insomnia symptoms were significantly associated with COVID-19 related disruptions, and those previously received dCBT-I reported less insomnia symptoms, less general stress and COVID-related cognitive intrusions, less depression, and better global health than those who received sleep education. Moreover, the odds for resurgent insomnia was 51% lower in the dCBT-I versus control condition. Similarly, odds of moderate to severe depression during COVID-19 was 57% lower in the dCBT-I condition.

Conclusions: Those who received dCBT-I had increased health resilience during the COVID-19 pandemic in adults with a history of insomnia and ongoing mild to moderate mental health symptoms. These data provide evidence that dCBT-I is a powerful tool to promote mental and physical health during stressors, including the COVID-19 pandemic.

Keywords: stress, COVID-19, insomnia, depression, CBT-I, engagement, prevention, primary care, digital health

Clinical Trial: Sleep to Prevent Evolving Affective Disorders; NCT02988375
(<https://clinicaltrials.gov/ct2/show/NCT02988375>)

Statement of Significance

This study examines the role of insomnia treatment in promoting resilience during the COVID-19 pandemic. Results indicate people who received treatment years prior to the pandemic exhibited better insomnia and stress regulation despite similar exposure to COVID-19. The protective effects were also observed in depression and global health.

Introduction

The 2019 coronavirus disease (COVID-19) pandemic has had health consequences that extend well-beyond symptoms of the virus. Mental health problems, including symptoms of insomnia, posttraumatic stress, and depression are already being observed in the context of COVID-19^{1–5} and have been documented during previous epidemics (e.g., SARS, MERS).⁶ As a consequence, COVID-related disability and mortality will include impairment from mental illness, which is already a leading contributor to global disease burden.⁷ Public health interventions that aim to reduce COVID-related disease burden should include interventions to promote mental resilience.^{8–11}

Insomnia is a common and debilitating consequence of pandemic-related stress and schedule disruption,^{3,9} and thus is of particular relevance during the COVID-19 pandemic. Although sleep disruption can be a symptom of mental disorders, insomnia is increasingly considered a distinct and comorbid disorder that warrants sleep-focused intervention.^{12–14} In addition to the suffering associated with sleeplessness, insomnia triggers and/or exacerbates other mental health problems,^{15–18} adds to distress and impairment when comorbid with mental illness,^{19–24} and frequently persists after other symptoms of mental illness have remitted.^{25–29} This may be because insomnia further sensitizes the stress system,³⁰ leading to increased perceptions of life events as more stressful³¹ and reduced resilience and recovery from stress.³²

Although the evidence clearly supports managing and treating insomnia, it is also important to examine how resilience can be promoted to prevent insomnia in response to new stressors. This aligns with the growing call for a focus on prevention to increase impact for public health.^{33,34} Insomnia is an ideal target for prevention because it is well-defined, highly modifiable, and is a robust risk factor for a range of psychiatric and medical morbidities.^{18,35,36} Moreover, the treatment effects of Cognitive Behavioral Therapy for Insomnia (CBT-I) – the recommended first-line treatment for chronic insomnia^{37,38} – extend beyond insomnia to reduce the incidence, severity, persistence, and recurrence of non-sleep mental and physical health problems. For example, CBT-I has been shown to reduce non-sleep symptoms of depression and anxiety,^{39,40} non-sleep focused rumination,⁴¹ chronic pain,⁴² as well as global health and quality of life.⁴³ Together, these results suggest that the effects of insomnia treatment may strengthen health across multiple domains to promote resilience against future stressors. Indeed, we have shown that CBT-I reduced the one-year incidence of depression by 50%, even when delivered digitally (dCBT-I) for increased accessibility.⁴⁴

The overarching aim of the present study was to examine resilience in the sleep and stress systems during the COVID-19 pandemic. As individuals with a history of insomnia disorder are at higher risk of experiencing stress and insomnia symptoms during the COVID-19 pandemic, we were interested in evaluating if those who had previously received insomnia treatment experienced more resilience. To accomplish this aim, we invited participants from a 2016–2017 dCBT-I intervention trial to complete measures of COVID impact, symptoms of insomnia, general- and COVID-related stress, and depression and physical health. Participants in this trial predominantly resided in the Detroit metropolitan area, which was disproportionately impacted by the COVID-19 pandemic. Our central hypothesis was that individuals who previously received dCBT-I would show more health resilience during the COVID-19 pandemic compared to individuals who received sleep education. Our first specific hypothesis was that insomnia symptoms during the pandemic would be less severe in those who previously received dCBT-I compared to those in the sleep education control condition. Our

second specific hypothesis was that those who previously received dCBT-I would report less stress during the pandemic compared to participants in the control condition. Our third hypothesis was that participants who previously received dCBT-I would have lower depressive symptoms and better global health compared to the control group.

Methods

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 insomnia⁷¹ and preventing incident depression.⁴⁴ 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. Those in the dCBT-I condition completed 6 sessions of self-guided dCBT-I, which 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.^{72,73} 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 (see Cheng et al., 2018⁷¹ for addition details).

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: $n = 106$).

Measures of interest

Once enrolled, participants completed an online survey that assessed for COVID impact, insomnia, stress, and health during the COVID-19 pandemic.

Impact of the COVID-19 pandemic. Direct impact from the COVID-19 was measured using the same framework as the Life Events Checklist.⁷⁴ Three prompts were included: 1) Exposure to the coronavirus, 2) Life-threatening illness or injury related to the coronavirus, 3) Severe human suffering related to the coronavirus. Participants were asked if those items happened to them in six different ways: 1) it happened to them, 2) they witnessed it happening to someone else, 3) they learned about it happening to a close friend or family member, 4) they were exposed to it as part of their job, or if 5) they are unsure or 6) it does not apply to them. Direct impact from the novel coronavirus was operationalized as any endorsement of responses 1 through 4 on at least one of the three items described.

The impact of the COVID-19 pandemic on daily life was measured using the Coronavirus Impact Scale (CIS). The CIS was made available through a collection of COVID-19 Research Tools assembled by the Office of Behavioral and Social Sciences Research at the National Institutes of Health.⁷⁵ The CIS rates the degree of change across multiple domains of daily life on a four-point Likert scale (0 = No change, 1 = Mild, 2 = Moderate, 3 = Severe) across 11 items. Domains assessed included routines; income/employment; access to food, medical care, and mental treatment; access to social support; pandemic related stress; familial stress and discord; and diagnoses of coronavirus. One open-ended item allowed free text responses to capture other ways in which daily life may have been impacted by the COVID-19 pandemic. Given the recency of the pandemic, there is no psychometric data available for the CIS.

Insomnia

. Symptoms of insomnia were assessed using the 7-item Insomnia Severity Index (ISI),⁷⁶ with higher scores indicating increased insomnia severity (range 0 – 28). A score of 15 or greater on the ISI was used as a threshold for moderate to severe insomnia. Because the ISI is not designed to assess insomnia in response to a specific event, another question was included to assess the impact of the COVID-19 pandemic on sleep using a 5-point Likert scale. The prompt was, “*How much impact did the COVID-19 pandemic have on your sleep?*”, and responses ranged from Not at all (0) to Very much (4). Results from this item was examined independently and was not incorporated into the ISI.

Stress. Both general stress and stress specific to the COVID-19 pandemic were assessed. General stress was measured using a validated single-item instrument.^{77,78} The prompt for this instrument was “*Stress means a situation in which a person feels tense, restless, nervous or anxious or is unable to sleep at night because his/her mind is troubled all the time. Do you feel this kind of stress these days?*” Response on this instrument was on a 5-point Likert scale ranging from never (0) to always (4).

Stress and trauma specific to the COVID-19 pandemic was assessed using the 22-item Impact of Events Scale⁷⁹ (IES_{COVID-19}). The IES measures the amount of distress associated with a specific event. Though the IES allows individuals to specify the event in question, we predetermined “the COVID-19 pandemic” in the instructions as we were interested in measuring stress specifically associated with the COVID-19 pandemic. The total score on the IES ranges from 0 to 88, with a score of 24 indicating clinically significant impairment. The IES score comprises three component scores: cognitive intrusion, avoidance, and hyperarousal. Both cognitive intrusion and avoidance describe psychological experiences of stress prior to assimilation of the trauma, the cognitive intrusion

reflecting repeated thoughts about the trauma, and avoidance reflecting effortful avoidance of reminders of the trauma. The third component describes physiological hyperarousal as a cluster of PTSD symptoms.

Health Outcomes. In addition to insomnia symptoms, we also assessed for other health outcomes including depression and global health. Depression was assessed using the 16-item self-report Quick Inventory of Depressive Symptomatology (QIDS-SR₁₆),⁸¹ a reliable and validated instrument for measuring depression symptoms commonly used in clinical trials. Scores on the QIDS-SR₁₆ range from 0 to 27, and a score greater than 10 reflects moderately severe symptoms. Global health was assessed via the Global-10 from the NIH Patient-Reported Outcomes Measurement Information System (PROMIS).⁸² The Global-10 assesses general domains of health and functioning including overall physical health, mental health, and overall perceived quality of life. The Global-10 has two components: Global Physical Health (GPH), and Global Mental Health (GMH). Both components are normed on a T-score distribution, with the population mean at 50 and a standard deviation of 10 points. Higher scores on the Global-10 and its components indicate better health.

Analytical Approach

The hypotheses were tested with ordinary least squares regression models with Condition (dCBT-I, sleep education control [reference group]) as the independent variable. All analyses controlled for pre-treatment insomnia severity as a measure of insomnia risk. The first hypothesis was tested using standardized scores from the ISI as the dependent variable. Because the dCBT-I condition reported less insomnia after treatment compared to those in the control condition, a sensitivity analysis was conducted in a subsample of individuals who reported symptom resolution (ISI < 8) at one-year follow-up. This analysis enabled more robust inference regarding symptom resurgence during the COVID-19 pandemic. To distinguish symptom resurgence from normative variations in sleep (especially given the context of a global pandemic), symptom resurgence was operationalized as moderate to severe symptoms (ISI ≥ 15) in those who previously reported symptom resolution. The second hypothesis was tested using standardized general stress and IES_{COVID-19} total and component scores as the dependent variables via regressions models. The third hypothesis was tested using standardized scores on the QIDS-16_{SR} and the Global-10 component t-scores as the dependent variables in regression models. To examine the robustness of results, effect sizes were also contrasted between those who were and were not directly impact by the coronavirus.

Given that research conducted during a global pandemic may be vulnerable to selection bias,^{83,84} 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. Insomnia severity categories were non-clinically significant ($ISI \leq 7$), subthreshold ($ISI > 7$ and ≤ 14), and clinically significant ($ISI \geq 15$). 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

The final sample included 208 (dCBT-I: $n = 102$; control: $n = 106$; see Table 1 for a summary of sample characteristics by group). During the COVID-19 pandemic, 67.3% of the sample reported direct impact from the coronavirus, and 26.4% reported living alone during the shelter-in-place orders. On average, the sample reported mild disruptions across the domains assessed on the CIS ($M = 1.1 \pm 0.03$ SE). The three domains most impacted by COVID-19 included daily routines ($M = 2.2 \pm 0.06$ SE), stress ($M = 1.7 \pm 0.06$ SE), and social support ($M = 1.6 \pm 0.06$ SE). The control and dCBT-I condition reported similar levels of disruption due to COVID-19. However, despite similar levels of disruption due to COVID-19, those who previously received the sleep education control reported that the pandemic had a larger impact on their sleep ($M = 2.0 \pm 0.12$ SE) compared to those who received dCBT-I ($M = 1.5 \pm 0.11$ SE), $p = .009$.

[Table 1]

Insomnia during COVID-19

The average ISI score was 12.1 ± 0.48 SE (see Figure 1 for ISI by group), with 34.1% reporting moderate to severe insomnia symptoms ($ISI \geq 15$). Adjusting for baseline insomnia symptoms, ISI scores during the pandemic were significantly associated with the impact of the coronavirus assessed by CIS scores, $B = 0.52 \pm 0.05$ SE, $p < .001$ (see Figure 1). Similarly, those who reported that the pandemic had a larger impact on their sleep also exhibited more severe ISI scores, $B = 0.83 \pm 0.05$ SE, $p < .001$. Together, these data indicate a robust association between insomnia symptoms and the COVID-19 pandemic.

Consistent with our hypothesis, results also revealed that those who previously received dCBT-I exhibited less severe insomnia symptoms during the pandemic, $b = -2.9 \pm 0.8$ SE, $p = .001$ ($B = -0.41$), indicating that ISI scores were approximately 3 points lower in the dCBT-I group compared to the control group during the pandemic. The effect size of dCBT-I on insomnia symptoms during COVID-19 were comparable between individuals directly impacted by the coronavirus ($B = -0.43$) compared to those who were not directly impacted ($B = -0.37$). Sensitivity analysis also revealed that odds of resurgent moderate to severe insomnia during COVID-19 in those who previously reported symptom resolution ($ISI < 8$ at one-year follow-up) was 51% lower in those who received dCBT-I relative to control, OR = 0.49, 95% CI [0.25, 0.96], $p < .001$.

[Figure 1]

Stress during COVID-19

The sample reported a mean general stress score of 2.3 ± 0.07 SE (see Figure 2 for means by condition). Consistent with our hypothesis, those who previously received dCBT-I also demonstrated a trend of less overall stress levels during the COVID-19 pandemic compared to the control condition, $b = -0.2 \pm 0.1$ SE, $p = .055$ sample ($B = -0.25$), despite similar exposure to disruptions due to COVID-19. The buffer effect of dCBT-I on stress was larger for those reporting direct impact by the coronavirus ($B = -0.36$) compared to those who were not directly impacted ($B = -0.09$).

In terms of COVID-19 specific stress, the sample reported a mean $IES_{COVID-19}$ score of 26.5 ± 1.0 SE (see Figure 2 for component scores by condition). Those who previously received dCBT-I reported lower total $IES_{COVID-19}$ scores compared to the control condition, $b = -4.1 \pm 1.9$ SE, $p = .03$. Analyses of the $IES_{COVID-19}$ component scores revealed a significant effect for cognitive intrusion, $b = -0.3 \pm 0.1$ SE, $p = .03$ ($B = -0.30$), a marginal effect for hyperarousal, $b = -0.2 \pm 0.1$ SE, $p = .08$ ($B = -0.23$), but no significant effect for avoidance ($p = .23$). The buffer effect of dCBT-I against cognitive intrusion was comparable for those who were directly impacted by the coronavirus ($B = -0.31$) than those who were not directly impacted ($B = -0.26$).

[Figure 2]

Health during COVID-19

Depression. The sample reported a mean QIDS-SR₁₆ score of 10.6 ± 0.3 SE (see Figure 3 for means by condition). Those who previously received dCBT-I also showed lower depressive symptoms, $b = -1.3 \pm 0.5$ SE, $p = .01^1$. Importantly, the odds of moderate to severe depressive symptoms during COVID-19 was 57% lower in those who had received dCBT-I compared to those who received sleep education, OR = 0.43, 95% CI [0.30, 0.61], $p < .001$.

Global Health. The sample's average Global Physical Health (GPH) and Global Mental Health (GMH) t-scores were 45.7 ± 0.5 SE and 43.4 ± 0.6 SE, respectively (see Figure 3 for means by condition). Results from the multivariate regression indicated that who previously received dCBT-I reported better global health, $F(2,204) = 3.77$, $p = .02$.

Consistent with the other results, those who previously received dCBT-I exhibited better Global Physical Health, $b = 2.76 \pm 1.01$ SE, $p = .006$. As the subscales are normed on a T-score distribution ($M = 50$, $SD = 10$), this indicates that while both groups show GPH scores within a SD of the population mean, the deviation from population mean in the control group (5.9 points) was approximately two-fold higher than that of the dCBT-I group (3.1 points), indicating better global physical health in the dCBT-I group relative to the control group. In contrast, group differences on the GMH

¹ Results were not substantively different when sleep items were removed from the QIDS-SR₁₆.

component did not achieve statistical significance, $b = 1.46 \pm 1.10 \text{ SE}$, $p = .18$ (effect size corresponds to a Cohen's d of 0.15).

Given that the sample was powered to detect moderate effect sizes, additional analyses were conducted to examine the odds of low GPH and GMH scores, defined as scores half a standard deviation lower than the population norm ($t\text{-score} \leq 45$, corresponding to a Cohen's d of 0.5). These variables were then used in a logistic regression with Condition as the predictor of interest. Results indicated that the odds of reporting low GPH and GMH was 58% and 42% lower in the dCBT-I group relative to the control group, respectively (see Figure 3), GPH: OR = 0.42, 95% CI [0.29, 0.62], $p = .002$; GMH: OR = 0.58, 95% CI [0.42, 0.82], $p < .001$.

[Figure 3]

Discussion

The results of this study support the overall hypothesis that dCBT-I treatment increases health resilience during the COVID-19 pandemic. Consistent with other early reports of COVID-related health outcomes,^{3,5} COVID-related disruptions in daily life were associated with insomnia symptoms. At the time of data collection, southeast Michigan was a significant hotspot of COVID-19 cases (approximately 40,000 cases and 3800 deaths) and it is therefore not surprising that most of the participants in this sample reported that the COVID-19 pandemic had a direct impact on their lives. On average, the sample also reported insomnia symptoms, moderate overall stress, clinically significant COVID-related stress, moderate depressive symptoms, and moderate overall health.

Considered together, these results provide evidence that dCBT-I promotes health resilience in an adult population with a history of insomnia and ongoing mild to moderate psychiatric symptoms. Results showed that, relative to a sleep education intervention, adults who completed dCBT-I in 2016-2017 had lower symptoms of insomnia, lower general stress and COVID-related cognitive intrusions (intrusive thoughts, feelings, and imagery; nightmares; dissociative-like re-experiencing), lower depressive symptom severity, and better global health. Moreover, these data suggest that dCBT-I helped build resilience during the COVID-19 pandemic. Relative to sleep education, dCBT-I prevented a resurgence of clinically significant insomnia during the pandemic by 50%. Additionally, the odds of moderate to severe depression in those who received dCBT-I was almost 60% lower than the sleep education group. These health outcomes were observed in April 2020 of the COVID-19 pandemic, 3-4 years after treatment completion, suggesting that dCBT-I offers long-lasting protection across multiple health domains and in the context of a global health threat. In light of the pressing mental health needs associated with the COVID-19 pandemic, the significance of these results is difficult to overstate. They add to a growing literature suggesting that behavioral insomnia treatment improves health,^{39,59,66,68-70} and provide the first prospective evidence that dCBT-I increases health resilience during later stressors — in this case the COVID-19 pandemic.

This study was not designed to examine mechanisms of insomnia treatment; however, there are a number of potential mechanisms by which dCBT-I may promote health resilience. Hyperarousal models posit that insomnia may be related to disruptions in biological (autonomic and central nervous system functioning⁸⁵⁻⁸⁸) and cognitive systems (cognitive arousal, worry and rumination,

and emotional distress^{58,89–91}) that support energy mobilization.^{92–96} CBT-I can mitigate these underlying mechanisms,^{41,59,97,98} especially as the components of CBT-I (e.g., cognitive restructuring, relaxation, sleep hygiene) are designed to target the arousal system as a barrier to sleepiness around the sleep period. Indeed, our results suggest that those who previously completed dCBT-I experienced less stress, and cognitive intrusion and hyperarousal on the IES_{COVID-19}. Additionally, research should also further examine different mechanisms by which insomnia contributes to adverse outcomes relevant to the COVID-19 pandemic, such as stress and trauma-related symptoms (e.g., vigilance to threat) versus depressive symptoms (e.g., hedonic processing). Different types of insomnia (e.g., short sleep and non-restorative phenotypes) may also differentially impact biological and cognitive-emotional systems. For example, whereas medial prefrontal cortex functioning has been implicated in the relationship between non-restorative sleep and later depressive symptoms, it does not have a significant role in the relationship between nocturnal insomnia symptoms and later depressive symptoms.⁹⁹ Understanding the mechanisms by which insomnia contributes to different types of mental health problems (multifinality), or how different types of insomnia contribute to the same mental health problem (equifinality) may facilitate personalized and mechanistically-specific treatment approaches.

It is now evident that the COVID-19 pandemic will likely be a long-term stressor for many, particularly as the temporary support systems begin to subside (e.g., federal stimulus payments, enhanced unemployment), and as additional waves recur. This has potential to perpetuate a compounding cascade of negative consequences for mental and physical health, particularly as chronic stress increases allostatic load and erodes biological and emotional-cognitive systems.¹⁰⁰ As such, it is also important to consider the role of insomnia treatment during the COVID-19 pandemic, particularly as untreated insomnia is both common and debilitating. Epidemiological data prior to the pandemic indicates that approximately 9% of adults within the United States report daily or near daily insomnia symptoms,¹⁰¹ and this number may have increased due to COVID-19. Additionally, the economic burden of insomnia is significant,^{102–106} and is worsened by its medical comorbidities.^{107,108} Together, these data suggest that the prevalence and costs of insomnia are likely to increase as the COVID-19 pandemic continues.^{3,9} dCBT-I offers an accessible (self-paced, geographically unrestricted, low cost), acceptable, and efficacious intervention for insomnia and mental health.^{39,109} Our data suggest that we could capitalize on the promise of dCBT-I to promote mental and physical health during the COVID-19 pandemic.

This study has both strengths and limitations that impact the significance of these results. Strengths of this study include the assessment of long-term effects of dCBT-I during a natural and chronic stressor that will have a myriad of major implications for public health. Most of the sample reported direct impact from the coronavirus, providing face validity to our claim that dCBT-I improves health resilience. The study sample ($n = 208$) allowed for adequate power to detect moderate effects of dCBT-I on health outcomes. Furthermore, the study included sleep education as an active comparison condition to control for non-specific effects of intervention effort and attention on health outcomes. The sample was also heterogeneous with regard to race/ethnicity and socioeconomic status, providing greater confidence in the generalizability of the results.

Limitations of this study include potential selection bias considering the context of a global pandemic; however, the analyses utilized sampling weights to mitigate this limitation. Additionally, the measure of general stress included sleep disturbance as part of the phenomenology of stress

response. The sample size, although large enough to detect moderately large effects of interest, was not sufficient to detect potential moderators of dCBT-I outcomes (e.g., COVID-exposure, COVID-related stress, race, socioeconomic status) in primary hypothesis tests. We also did not design the study to examine mechanisms by which dCBT-I may differentially predict individual health outcomes (i.e., insomnia symptoms, COVID-related intrusions, depressive symptoms, global physical health). Finally, data collected in April 2020 may not represent the effects of dCBT-I later in the course of the COVID-19 pandemic. Additional follow-up assessments could help establish the persistence of benefit from dCBT-I and the relationship between mental health symptoms and COVID-19 chronicity and impact over time.

Conclusion

This study examined the role of prior treatment of insomnia with digital CBT-I (dCBT-I) on health resilience during the COVID-19 pandemic. Results demonstrated that those who received dCBT-I reported less insomnia, stress, depression, and better global physical health compared to those who received a sleep education control. Indeed, the risk of clinically significant insomnia and depression during the pandemic was reduced approximately by half in the dCBT-I group relative to the control group. Future research should examine the mechanisms by which insomnia treatment may enhance resilience, and the role of dCBT-I in mitigating the adverse health consequences of the COVID-19 pandemic.

Author Contributions

Concept and Design: Cheng, Casement, Drake

Acquisition, Analysis, or Interpretation of data: Cuamatzi Castelan, Cheng, Casement

Drafting of manuscript: Cheng, Casement

Critical revision of the manuscript for important intellectual content: Kalmbach, Drake

Acknowledgements

Support for PC was provided from the National Heart Lung and Blood Institute (K23HL138166). We would also like to thank the Division of Sleep Medicine and the staff at the Thomas Roth Sleep Disorders and Research Center at the Henry Ford Health System for their continued support.

Disclosures

Financial Disclosures: PC is funded by the National Heart Lung and Blood Institute (K23HL138166)

Non-financial Disclosures: none

References

1. Nelson BW, Pettitt AK, Flannery J, Allen NB. Rapid Assessment of Psychological and Epidemiological Correlates of COVID-19 Concern, Financial Strain, and Health-Related Behavior Change in a Large Online Sample. *PsyArXiv*; 2020.
2. Wang C, Pan R, Wan X, et al. Immediate Psychological Responses and Associated Factors during the Initial Stage of the 2019 Coronavirus Disease (COVID-19) Epidemic among the General Population in China. *Int J Environ Res Public Health*. 2020;17(5):1729.
3. Huang Y, Zhao N. Generalized anxiety disorder, depressive symptoms and sleep quality during COVID-19 outbreak in China: a web-based cross-sectional survey. *Psychiatry Res*. 2020;288:112954.
4. Rajkumar RP. COVID-19 and mental health: A review of the existing literature. *Asian J Psychiatry*. 2020;52:102066.
5. Cellini N, Canale N, Mioni G, Costa S. Changes in sleep pattern, sense of time and digital media use during COVID-19 lockdown in Italy. *J Sleep Res*. n/a(n/a):e13074.
6. Torales J, O'Higgins M, Castaldelli-Maia JM, Ventriglio A. The outbreak of COVID-19 coronavirus and its impact on global mental health. *Int J Soc Psychiatry*. 2020;66(4):317-320.
7. World Health Organization. Global Health Estimates 2016: Disease burden by Cause, Age, Sex, by Country and by Region, 2000-2016. Published online 2018.
8. Holmes EA, O'Connor RC, Perry VH, et al. Multidisciplinary research priorities for the COVID-19 pandemic: a call for action for mental health science. *Lancet Psychiatry*. 2020;7(6):547-560.
9. Altena E, Baglioni C, Espie CA, et al. Dealing with sleep problems during home confinement due to the COVID-19 outbreak: practical recommendations from a task force of the European CBT-I Academy. *J Sleep Res*. n/a(n/a).
10. Galea S, Merchant RM, Lurie N. The Mental Health Consequences of COVID-19 and Physical Distancing: The Need for Prevention and Early Intervention. *JAMA Intern Med*. 2020;180(6):817-818.
11. Pfefferbaum B, North CS. Mental Health and the Covid-19 Pandemic. *N Engl J Med*. 2020;0(0):null.
12. Germain A, McKeon AB, Campbell RL. Sleep in PTSD: Conceptual model and novel directions in brain-based research and interventions. *Curr Opin Psychol*. 2017;14:84-89.
13. Manber R, Chambers AS. Insomnia and depression: A multifaceted interplay. *Curr Psychiatry Rep*. 2009;11:437-442.
14. Vargas I, Perlis ML. Insomnia and depression: clinical associations and possible mechanistic links. *Curr Opin Psychol*. 2020;34:95-99.
15. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *J Affect Disord*. 2011;135:10-19.

16. Hertenstein E, Feige B, Gmeiner T, et al. Insomnia as a predictor of mental disorders: A systematic review and meta-analysis. *Sleep Med Rev.* 2019;43:96-105.
17. Pigeon WR, Bishop TM, Krueger KM. Insomnia as a Precipitating Factor in New Onset Mental Illness: a Systematic Review of Recent Findings. *Curr Psychiatry Rep.* 2017;19(8):44.
18. Li L, Wu C, Gan Y, Qu X, Lu Z. Insomnia and the risk of depression: a meta-analysis of prospective cohort studies. *BMC Psychiatry.* 2016;16(1):375.
19. Soehner AM, Kaplan KA, Harvey AG. Prevalence and clinical correlates of co-occurring insomnia and hypersomnia symptoms in depression. *J Affect Disord.* 2014;167:93-97.
20. O'Brien EM, Chelminski I, Young D, Dalrymple K, Hrabosky J, Zimmerman M. Severe insomnia is associated with more severe presentation and greater functional deficits in depression. *J Psychiatr Res.* 2011;45(8):1101-1105.
21. Gehrman P, Seelig AD, Jacobson IG, et al. Predeployment Sleep Duration and Insomnia Symptoms as Risk Factors for New-Onset Mental Health Disorders Following Military Deployment. *Sleep.* 2013;36(7):1009-1018.
22. Brownlow JA, McLean CP, Gehrman PR, Harb GC, Ross RJ, Foa EB. Influence of Sleep Disturbance on Global Functioning After Posttraumatic Stress Disorder Treatment. *J Trauma Stress.* 2016;29(6):515-521.
23. Mason EC, Harvey AG. Insomnia before and after treatment for anxiety and depression. *J Affect Disord.* 2014;168:415-421.
24. Pigeon WR, Campbell CE, Possemato K, Ouimette P. Longitudinal relationships of insomnia, nightmares, and PTSD severity in recent combat veterans. *J Psychosom Res.* 2013;75(6):546-550.
25. Schnurr PP, Lunney CA. Residual symptoms following prolonged exposure and present-centered therapy for PTSD in female veterans and soldiers. *Depress Anxiety.* 2019;36(2):162-169.
26. Larsen SE, Fleming CJE, Resick PA. Residual symptoms following empirically supported treatment for PTSD. *Psychol Trauma Theory Res Pract Policy.* 2019;11(2):207-215.
27. Xiao L, Feng L, Zhu X, et al. Comparison of residual depressive symptoms and functional impairment between fully and partially remitted patients with major depressive disorder: a multicenter study. *Psychiatry Res.* 2018;261:547-553.
28. Carney CE, Segal ZV, Edinger JD, Krystal AD. A comparison of rates of residual insomnia symptoms following pharmacotherapy or cognitive-behavioral therapy for major depressive disorder. *J Clin Psychiatry.* 2007;68(2):254-260.
29. Nierenberg AA, Husain MM, Trivedi MH, et al. Residual symptoms after remission of major depressive disorder with citalopram and risk of relapse: a STAR*D report. *Psychol Med.* 2010;40(1):41-50.
30. Kalmbach DA, Pillai V, Arnedt JT, Anderson JR, Drake CL. Sleep system sensitization: evidence for changing roles of etiological factors in insomnia. *Sleep Med.* 2016;21:63-69.

31. Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. *Psychosom Med*. 2003;65(2):259–267.
32. Palagini L, Moretto, Umberto, Novi, Martina, et al. Lack of Resilience Is Related to Stress-Related Sleep Reactivity, Hyperarousal, and Emotion Dysregulation in Insomnia Disorder. *J Clin Sleep Med*. 2018;14(05):759-766.
33. Ebert DD, Cuijpers P. It Is Time to Invest in the Prevention of Depression. *JAMA Netw Open*. 2018;1(2):e180335-e180335.
34. Germain A, Dretsches M. Sleep and resilience—a call for prevention and intervention. *Sleep*. 2016;39(5):963–965.
35. Taylor DJ, Lichstein KL, Durrence HH. Insomnia as a Health Risk Factor. *Behav Sleep Med*. 2003;1(4):227-247.
36. Dolsen MR, Asarnow LD, Harvey AG. Insomnia as a transdiagnostic process in psychiatric disorders. *Curr Psychiatry Rep*. 2014;16(9):471.
37. Qaseem A, Kansagara D, Forcica MA, Cooke M, Denberg TD. Management of Chronic Insomnia Disorder in Adults: A Clinical Practice Guideline From the American College of Physicians. *Ann Intern Med*. 2016;165(2):125-133.
38. Riemann D, Baglioni C, Bassetti C, et al. European guideline for the diagnosis and treatment of insomnia. *J Sleep Res*. 2017;26(6):675-700.
39. Cheng P, Luik AI, Fellman-Couture C, et al. Efficacy of digital CBT for insomnia to reduce depression across demographic groups: a randomized trial. *Psychol Med*. 2019;49(3):491-500.
40. Pillai V, Anderson JR, Cheng P, et al. The Anxiolytic Effects of Cognitive Behavior Therapy for Insomnia: Preliminary Results from a Web-delivered Protocol. Published online 2015:7.
41. Cheng P, Kalmbach DA, Castelan AC, Murugan N, Drake CL. Depression prevention in digital cognitive behavioral therapy for insomnia: Is rumination a mediator? *J Affect Disord*. 2020;273:434–441.
42. Jungquist CR, O'Brien C, Matteson-Rusby S, et al. The Efficacy of Cognitive Behavioral Therapy for Insomnia in Patients with Chronic Pain. *Sleep Med*. 2010;11(3):302-309.
43. Espie CA, Emsley R, Kyle SD, et al. Effect of Digital Cognitive Behavioral Therapy for Insomnia on Health, Psychological Well-being, and Sleep-Related Quality of Life: A Randomized Clinical Trial. *JAMA Psychiatry*. 2019;76(1):21-30.
44. Cheng P, Kalmbach D, Tallent G, Joseph C, Espie CA, Drake C. Depression Prevention Via Digital CBT for Insomnia: A Randomized Controlled Trial. *SLEEP*. Published online 2019.
45. Schutte-Rodin S, Broch L, Buysse D, Dorsey C, Sateia M. Clinical Guideline for the Evaluation and Management of Chronic Insomnia in Adults. *J Clin Sleep Med JCSM Off Publ Am Acad Sleep Med*. 2008;4(5):487-504.

46. van Straten A, van der Zweerde T, Kleiboer A, Cuijpers P, Morin CM, Lancee J. Cognitive and behavioral therapies in the treatment of insomnia: A meta-analysis. *Sleep Med Rev.* 2018;38:3-16.
47. Mitchell MD, Gehrman P, Perlis M, Umscheid CA. Comparative effectiveness of cognitive behavioral therapy for insomnia: a systematic review. *BMC Fam Pract.* 2012;13(1):40.
48. Riemann D, Perlis ML. The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. *Sleep Med Rev.* 2009;13(3):205-214.
49. Cunningham JEA, Shapiro CM. Cognitive Behavioural Therapy for Insomnia (CBT-I) to treat depression: A systematic review. *J Psychosom Res.* 2018;106:1-12.
50. Ye Y, Zhang Y, Chen J, et al. Internet-Based Cognitive Behavioral Therapy for Insomnia (ICBT-i) Improves Comorbid Anxiety and Depression—A Meta-Analysis of Randomized Controlled Trials. *PLoS ONE.* 2015;10(11).
51. Taylor DJ, Pruiksma KE. Cognitive and behavioural therapy for insomnia (CBT-I) in psychiatric populations: A systematic review. *Int Rev Psychiatry.* 2014;26(2):205-213.
52. Ballesio A, Aquino MRJV, Feige B, et al. The effectiveness of behavioural and cognitive behavioural therapies for insomnia on depressive and fatigue symptoms: A systematic review and network meta-analysis. *Sleep Med Rev.* 2018;37:114-129.
53. Ho FY-Y, Chan CS, Tang KN-S. Cognitive-behavioral therapy for sleep disturbances in treating posttraumatic stress disorder symptoms: A meta-analysis of randomized controlled trials. *Clin Psychol Rev.* 2016;43:90-102.
54. Wu JQ, Appleman ER, Salazar RD, Ong JC. Cognitive Behavioral Therapy for Insomnia Comorbid With Psychiatric and Medical Conditions: A Meta-analysis. *JAMA Intern Med.* 2015;175:1461-1472.
55. Benz F, Knoop T, Ballesio A, et al. The efficacy of cognitive and behavior therapies for insomnia on daytime symptoms: A systematic review and network meta-analysis. *Clin Psychol Rev.* Published online June 5, 2020:101873.
56. Garland SN, Vargas I, Grandner MA, Perlis ML. Treating insomnia in patients with comorbid psychiatric disorders: A focused review. *Can Psychol Can.* 2018;59(2):176-186.
57. Gebara MA, Siripong N, DiNapoli EA, et al. Effect of insomnia treatments on depression: A systematic review and meta-analysis. *Depress Anxiety.* 2018;35(8):717-731.
58. Palagini L, Moretto U, Novi M, et al. Lack of Resilience Is Related to Stress-Related Sleep Reactivity, Hyperarousal, and Emotion Dysregulation in Insomnia Disorder. *J Clin Sleep Med JCSM Off Publ Am Acad Sleep Med.* 2018;14(5):759-766.
59. Kalmbach DA, Cheng P, Arnedt JT, et al. Treating insomnia improves depression, maladaptive thinking, and hyperarousal in postmenopausal women: comparing cognitive-behavioral therapy for insomnia (CBTI), sleep restriction therapy, and sleep hygiene education. *Sleep Med.* 2019;55:124–134.

60. Drake CL. The Promise of Digital CBT-I. *Sleep*. 2016;39(1):13-14.
61. Lancee J, van Straten A, Morina N, Kaldo V, Kamphuis JH. Guided Online or Face-to-Face Cognitive Behavioral Treatment for Insomnia: A Randomized Wait-List Controlled Trial. *Sleep*. 2016;39(1):183-191.
62. Luik AI, Bostock S, Chisnall L, et al. Treating Depression and Anxiety with Digital Cognitive Behavioural Therapy for Insomnia: A Real World NHS Evaluation Using Standardized Outcome Measures. *Behav Cogn Psychother*. 2017;45(1):91-96.
63. Luik AI, Marsden A, Emsley R, et al. Long-term benefits of digital cognitive behavioural therapy for insomnia: Follow-up report from a randomized clinical trial. *J Sleep Res*. n/a(n/a):e13018.
64. Zachariae R, Lyby MS, Ritterband LM, O'Toole MS. Efficacy of internet-delivered cognitive-behavioral therapy for insomnia – A systematic review and meta-analysis of randomized controlled trials. *Sleep Med Rev*. 2016;30:1-10.
65. Seyffert M, Lagisetty P, Landgraf J, et al. Internet-Delivered Cognitive Behavioral Therapy to Treat Insomnia: A Systematic Review and Meta-Analysis. *PLOS ONE*. 2016;11(2):e0149139.
66. Zweerde T van der, Straten A van, Eftting M, Kyle SD, Lancee J. Does online insomnia treatment reduce depressive symptoms? A randomized controlled trial in individuals with both insomnia and depressive symptoms. *Psychol Med*. 2019;49(3):501-509.
67. Cheng SK, Dizon J. Computerised Cognitive Behavioural Therapy for Insomnia: A Systematic Review and Meta-Analysis. *Psychother Psychosom*. 2012;81(4):206-216.
68. Cheng P, Kalmbach DA, Tallent G, Joseph CL, Espie CA, Drake CL. Depression prevention via digital cognitive behavioral therapy for insomnia: a randomized controlled trial. *Sleep*. Published online 2019:1-9.
69. Christensen H, Batterham PJ, Gosling JA, et al. Effectiveness of an online insomnia program (SHUTi) for prevention of depressive episodes (the GoodNight Study): a randomised controlled trial. *Lancet Psychiatry*. 2016;3(4):333-341.
70. Batterham PJ, Christensen H, Mackinnon AJ, et al. Trajectories of change and long-term outcomes in a randomised controlled trial of internet-based insomnia treatment to prevent depression. *BJPsych Open*. 2017;3(5):228-235.
71. Cheng P, Luik AI, Fellman-Couture C, et al. Efficacy of digital CBT for insomnia to reduce depression across demographic groups: a randomized trial. *Psychol Med*. Published online 2018:1–10.
72. Kessler RC, Coulouvrat C, Hajak G, et al. Reliability and Validity of the Brief Insomnia Questionnaire in the America Insomnia Survey. *Sleep*. 2010;33(11):1539-1549.
73. Espie CA, Kyle SD, Hames P, Cyhlarova E, Benzeval M. The daytime impact of DSM-5 insomnia disorder: comparative analysis of insomnia subtypes from the Great British Sleep Survey. *J Clin Psychiatry*. 2012;73(12):e1478–84.

74. Gray MJ, Litz BT, Hsu JL, Lombardo TW. Psychometric properties of the life events checklist. *Assessment*. 2004;11(4):330–341.
75. National Institutes of Health. Your guide to healthy sleep. Published online 2011.
76. Bastien CH, Vallières A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med*. 2001;2(4):297-307.
77. Elo A-L, Leppänen A, Jahkola A. Validity of a single-item measure of stress symptoms. *Scand J Work Environ Health*. Published online 2003:444–451.
78. Salminen S, Kouvonen A, Koskinen A, Joensuu M, Väänänen A. Is a single item stress measure independently associated with subsequent severe injury: a prospective cohort study of 16,385 forest industry employees. *BMC Public Health*. 2014;14(1):543.
79. Weiss DS, Marmar CR, Wilson JP, Keane TM. Assessing psychological trauma and PTSD. *Impact Events Scale—Revised*. 1997;19:399–411.
80. Miller WR, Rollnick S. *Motivational Interviewing: Helping People Change*. Guilford press; 2012.
81. Rush AJ, Trivedi MH, Ibrahim HM, et al. The 16-Item quick inventory of depressive symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biol Psychiatry*. 2003;54(5):573-583.
82. Cella D, Riley W, Stone A, et al. The Patient-Reported Outcomes Measurement Information System (PROMIS) developed and tested its first wave of adult self-reported health outcome item banks: 2005-2008. *J Clin Epidemiol*. 2010;63(11):1179-1194.
83. Zhao Q, Ju N, Bacallado S. BETS: The dangers of selection bias in early analyses of the coronavirus disease (COVID-19) pandemic. *ArXiv Prepr ArXiv200407743*. Published online 2020.
84. Sullivan SG. The need for robust epidemiological evidence during a pandemic. *Clin Infect Dis*. Published online 2020.
85. Nano M-M, Fonseca P, Vullings R, Aarts RM. Measures of cardiovascular autonomic activity in insomnia disorder: A systematic review. *PLOS ONE*. 2017;12(10):e0186716.
86. Dodds KL, Miller CB, Kyle SD, Marshall NS, Gordon CJ. Heart rate variability in insomnia patients: A critical review of the literature. *Sleep Med Rev*. 2017;33:88-100.
87. Carter JR, Grimaldi D, Fonkoue IT, Medalie L, Mokhlesi B, Van Cauter E. Assessment of sympathetic neural activity in chronic insomnia: evidence for elevated cardiovascular risk. *Sleep*. 2018;41(6).
88. Jarrin DC, Ivers H, Lamy M, Chen IY, Harvey AG, Morin CM. Cardiovascular autonomic dysfunction in insomnia patients with objective short sleep duration. *J Sleep Res*. 2018;27(3):e12663.

89. Kalmbach DA, Buysse DJ, Cheng P, Roth T, Yang A, Drake CL. Nocturnal cognitive arousal is associated with objective sleep disturbance and indicators of physiologic hyperarousal in good sleepers and individuals with insomnia disorder. *Sleep Med.* 2020;71:151-160.
90. Wassing R, Benjamins JS, Dekker K, et al. Slow dissolving of emotional distress contributes to hyperarousal. *Proc Natl Acad Sci.* 2016;113(9):2538-2543.
91. Kuisk LA, Bertelson AD, Walsh JK. Presleep Cognitive Hyperarousal and Affect as Factors in Objective and Subjective Insomnia. *Percept Mot Skills.* 1989;69(3-2):1219-1225.
92. Bonnet MH, Arand DL. Hyperarousal and insomnia: State of the science. *Sleep Med Rev.* 2010;14(1):9-15.
93. Kalmbach DA, Cuamatzi-Castelan AS, Tonnu CV, et al. Hyperarousal and sleep reactivity in insomnia: current insights. *Nat Sci Sleep.* 2018;10:193-201.
94. Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Med Rev.* 2010;14(1):19-31.
95. Levenson JC, Kay DB, Buysse DJ. The Pathophysiology of Insomnia. *Chest.* 2015;147(4):1179-1192.
96. Riemann D, Nissen C, Palagini L, Otte A, Perlis ML, Spiegelhalder K. The neurobiology, investigation, and treatment of chronic insomnia. *Lancet Neurol.* 2015;14(5):547-558.
97. Irwin MR, Olmstead R, Breen EC, et al. Cognitive Behavioral Therapy and Tai Chi Reverse Cellular and Genomic Markers of Inflammation in Late-Life Insomnia: A Randomized Controlled Trial. *Biol Psychiatry.* 2015;78(10):721-729.
98. Marques DR, Gomes AA, Clemente V, dos Santos JM, Caetano G, Castelo-Branco M. Neurobiological Correlates of Psychological Treatments for Insomnia. *Eur Psychol.* 2016;21(3):195-205.
99. Casement MD, Keenan KE, Hipwell AE, Guyer AE, Forbes EE. Neural Reward Processing Mediates the Relationship between Insomnia Symptoms and Depression in Adolescence. *Sleep.* 2016;39(2):439-447.
100. Cannon WB. Bodily Changes in Pain, Hunger, Fear, and Rage: An Account of Recent Research Into the Function of Emotional Excitement. 2nd ed. Appleton-Century-Crofts; 1929.
101. Ancoli-Israel S, Roth T. Characteristics of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. I. *Sleep.* 1999;22 Suppl 2:S347-353.
102. Sarsour K, Kalsekar A, Swindle R, Foley K, Walsh JK. The Association between Insomnia Severity and Healthcare and Productivity Costs in a Health Plan Sample. *Sleep.* 2011;34(4):443-450.
103. Stoller MK. Economic effects of insomnia. *Clin Ther Int Peer-Rev J Drug Ther.* 1994;16(5):873-897.

104. McCrae Christina S., Bramoweth Adam D., Williams Jacob, Roth Alicia, Mosti Caterina. Impact of Brief Cognitive Behavioral Treatment for Insomnia on Health Care Utilization and Costs. *J Clin Sleep Med*. 2014;10(02):127-135.
105. Snedecor SJ, Botteman MF, Bojke C, Schaefer K, Barry N, Pickard AS. Cost-Effectiveness of Eszopiclone for the Treatment of Adults with Primary Chronic Insomnia. *Sleep*. 2009;32(6):817-824.
106. Jhaveri M, Seal B, Pollack M, Wertz D. Will insomnia treatments produce overall cost savings to commercial managed-care plans? A predictive analysis in the United States. *Curr Med Res Opin*. 2007;23(6):1431-1443.
107. Asche CV, Joish VN, Camacho F, Drake CL. The direct costs of untreated comorbid insomnia in a managed care population with major depressive disorder. *Curr Med Res Opin*. 2010;26(8):1843-1853.
108. Bramoweth AD, Taylor DJ. Chronic Insomnia and Health Care Utilization in Young Adults. *Behav Sleep Med*. 2012;10(2):106-121.
109. Ho FY-Y, Chung K-F, Yeung W-F, et al. Self-help cognitive-behavioral therapy for insomnia: A meta-analysis of randomized controlled trials. *Sleep Med Rev*. 2015;19:17-28.

Tables and Figure Captions

Figure 1. Insomnia symptom severity during the COVID-19 pandemic. Panel A: Bubble chart of the weighted association between insomnia symptoms and disruptions to daily life due to COVID-19 ($r = 0.60$). Larger points indicate stronger weighting. Panel B: Estimated marginal means for insomnia severity by group. Error bars represent one standard error. *** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$, † $p < .10$

Figure 2. Estimated marginal means for stress by group. Panel A: General stress; Panel B: COVID-19 specific stress as measured on the Impact of Events Scale specific to COVID-19. Error bars represent one standard error. *** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$, † $p < .10$

Figure 3. Odds ratios of clinical outcomes in the dCBT-I relative to the control group. Resurgent Ins = resurgence of moderate to severe insomnia during COVID-19 in individuals who showed symptom resolution following the SPREAD trial; Mod-Sev Dep = moderate to severe depression on the Quick Inventory of Depressive Symptomatology (QIDS-SR₁₆); Low GPH = Global Physical Health scores below half a standard deviation of the population norm; Low GMH = Global Mental Health scores below half a standard deviation of the population norm. Error bars represent the 95% confidence intervals.

Table 1. Baseline sample characteristics by group. dCBT-I = digital Cognitive Behavioral Therapy for Insomnia; CIS = Coronavirus Impact Scale. No group differences were detected at $p < .05$.

	Control (n=106)	dCBT-I (n=102)
Age (M ± SD)	44.7 ± 14.2	44.6 ± 14.1
Sex (Female)	84.0%	72.5%
Race		
White	68.8%	73.5%
Black	27.4%	18.6%
Other	3.8%	7.8%
2019 Household Income		
Very low (<15k)	9.4%	5.0%
Low (<35k)	23.6%	19.6%
Middle (<75k)	34.0%	43.1%
High (≥75k)	33.0%	32.4%
Married/Partnered	48.1%	59.8%
Living alone	28.3%	24.5%
Pre-treatment ISI (M ± SD)	17.0 ± 4.12	18.0 ± 3.8
COVID-19 direct impact	67.0%	67.6%
CIS (M ± SD)	12.1 ± 5.3	11.4 ± 4.3

Figure 1

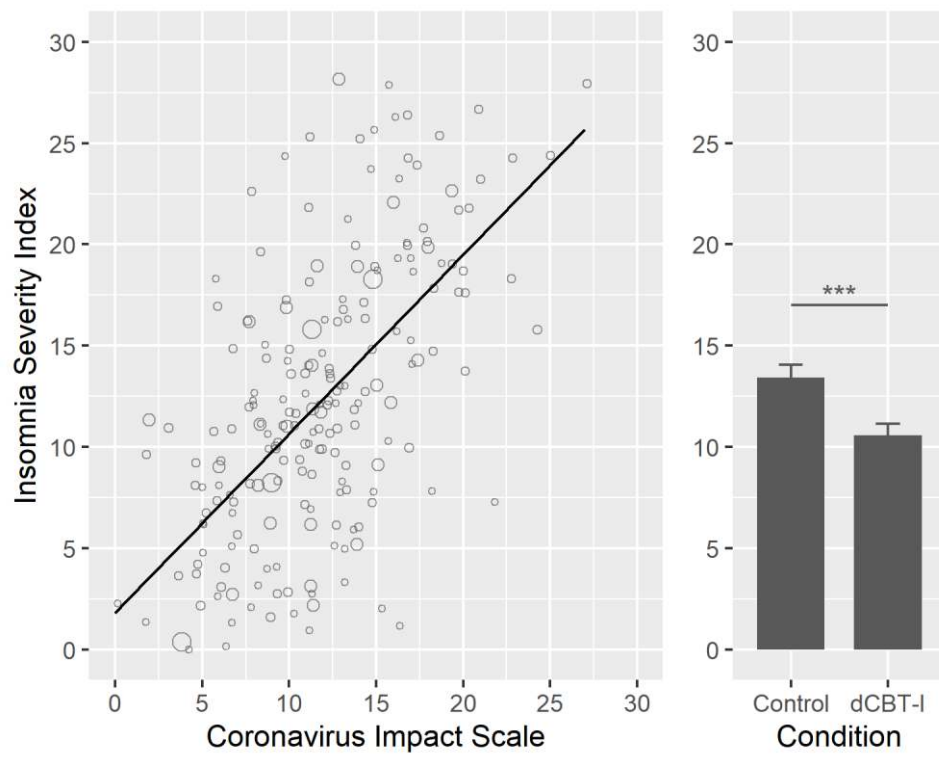


Figure 2

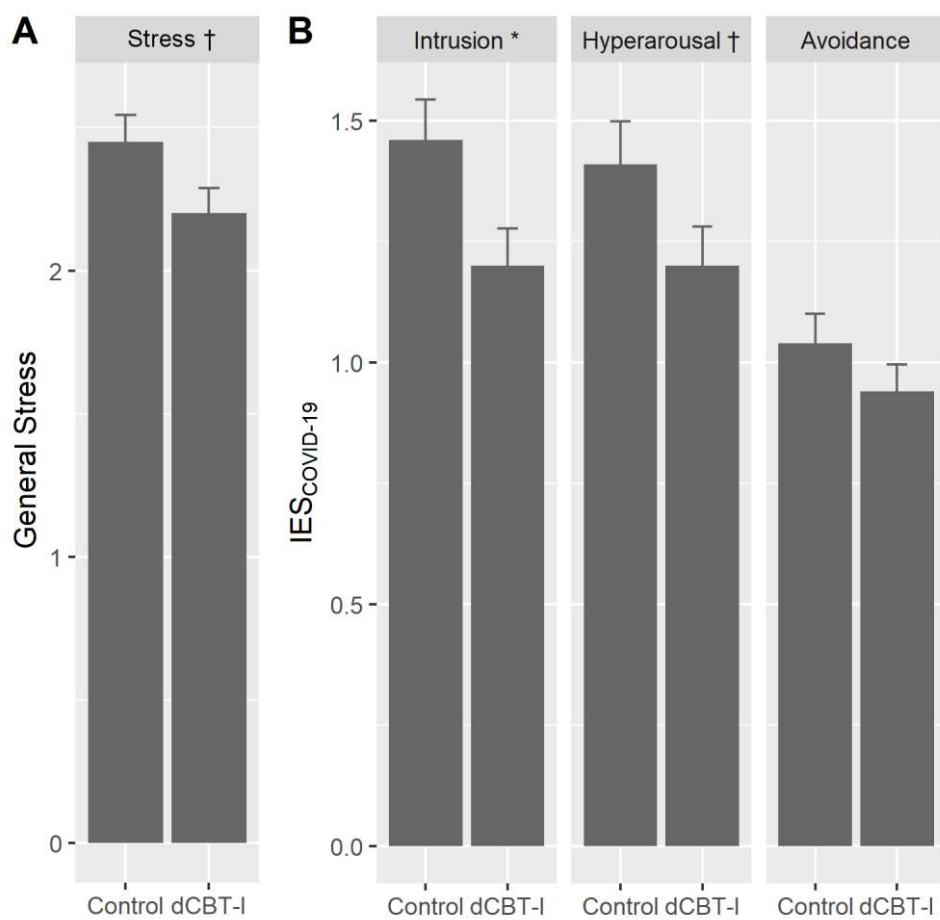


Figure 3

