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RESEARCH ARTICLE

Pre-pandemic sleep reactivity prospectively predicts distress during the COVID-19 pandemic: The protective effect of insomnia treatment

Anthony N. Reffi[1] | Christopher L. Drake[1] | David A. Kalmbach[1] | Tanja Jovanovic[2] | Seth D. Norrholm[2] | Thomas Roth[1] | Melynda D. Casement[3] | Philip Cheng[1]

[1]Sleep Disorders & Research Center, Henry Ford Health System, Detroit, Michigan, USA

[2]Neuroscience Center for Anxiety, Stress, and Trauma (NeuroCAST), Department of Psychiatry and Behavioral Neurosciences, Wayne State University School of Medicine, Detroit, Michigan, USA

[3]Department of Psychology, University of Oregon, Eugene, Oregon, USA

Correspondence
Anthony N. Reffi, Thomas Roth Sleep Disorders & Research Center, Department of Sleep Medicine, Henry Ford Health System, 39450 West Twelve Mile Road, Novi, Michigan, USA.
Email: areffi1@hfhs.org

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Summary

The COVID-19 pandemic is a rare stressor that has precipitated an accompanying mental health crisis. Prospective studies traversing the pandemic's onset can elucidate how pre-existing disease vulnerabilities augured risk for later stress-related morbidity. We examined how pre-pandemic sleep reactivity predicted maladaptive stress reactions and depressive symptoms in response to, and during, the pandemic. This study is a secondary analysis of a randomised controlled trial from 2016 to 2017 comparing digital cognitive behavioural therapy for insomnia (dCBT-I) against sleep education ($N = 208$). Thus, we also assessed whether dCBT-I moderated the association between pre-pandemic sleep reactivity and pandemic-related distress. Pre-pandemic sleep reactivity was measured at baseline using the Ford Insomnia Response to Stress Test. In April 2020, participants were recontacted to report pandemic-related distress (stress reactions and depression). Controlling for the treatment condition and the degree of COVID-19 impact, higher pre-pandemic sleep reactivity predicted more stress reactions ($\beta = 0.13$, ± 0.07 SE, $p = 0.045$) and depression ($\beta = 0.22$, ± 0.07 SE, $p = 0.001$) during the pandemic. Further, the odds of reporting clinically significant stress reactions and depression during the pandemic were over twice as high in those with high pre-pandemic sleep reactivity. Notably, receiving dCBT-I in 2016–2017 mitigated the relationship between pre-pandemic sleep reactivity and later stress reactions (but not depression). Pre-pandemic sleep reactivity predicted psychological distress 3–4 years later during the COVID-19 pandemic, and dCBT-I attenuated its association with stress reactions, specifically. Sleep reactivity may inform prevention and treatment efforts by identifying individuals at risk of impairment following stressful events.

KEYWORDS
adversity, chronic stress, longitudinal, prevention, resilience, risk

1 | INTRODUCTION

The 2019 coronavirus disease (COVID-19) pandemic has been a protracted global stressor with pernicious effects on mental health (Aknin et al., 2021). While many individuals have shown resilience (Gambin et al., 2021; Kimhi et al., 2021; Saunders et al., 2021), consistent with health trajectories following trauma (Galatzer-Levy et al., 2018), others have experienced steadily worsening symptoms

under this chronic stressor (Kimhi et al., 2021). Prospective studies assessing health factors prior to and during the COVID-19 pandemic are well positioned to help identify individuals who cope with chronic stress maladaptively (Bonanno, 2021), thereby elucidating disease risk factors that can be targeted to reduce morbidity in populations most vulnerable to dysfunction in response to stress (Chen et al., 2020). Accumulating evidence points to variability in stress system regulation as an important determinant of individual responses to adversity (Galatzer-Levy et al., 2018). Given its transdiagnostic nature, stress-related sleep dysregulation has gained interest as a robust risk factor for mental illness in response to stress exposure (Kalmbach et al., 2018). Further, the recent pandemic illustrated the need to better understand the role of sleep system dysregulation in the context of a global stressor.

Sleep reactivity is a vulnerability to sleep disturbances in response to a stressor (broadly defined) (Drake et al., 2004). Individuals with highly reactive sleep systems experience sleep disruptions in response to myriad laboratory challenges (Kalmbach et al., 2018) and naturalistic stressors (Petersen et al., 2013) that, in turn, presage the development of insomnia and mental illness over time (Drake et al., 2014; Kalmbach, Pillai, Arnedt, Anderson, & Drake, 2016). Importantly, sleep disturbances serve as barriers to adapting to adversity, augmenting emotional sensitivity and increasing perceived stress (Krause et al., 2017). For instance, sleep disturbances following trauma are associated with increased risk for symptoms of depression and post-traumatic stress disorder (PTSD) (Cox et al., 2017; Fan et al., 2017; Kim et al., 2021; Koren et al., 2002; von Känel et al., 2021). Yet, it is unclear who is at greatest risk of developing these sleep disturbances following exposure to potential trauma, hindering our ability to optimally prevent their downstream effects. Given that sleep reactivity precedes sleep disturbances, sleep reactivity may itself be an important predictor of psychological reactions to adverse life events, such as the COVID-19 pandemic.

Indeed, Neylan et al. (2021) recently found evidence that sleep reactivity prospectively predicts outcomes in the wake of trauma (Neylan et al., 2021). In their ongoing large-scale study of survivors of motor vehicle collisions (MVCs), participants reported pre-MVC sleep reactivity upon admission to the emergency department (among other sleep characteristics). Notably, pre-MVC sleep reactivity was the most consistent sleep-related predictor of posttraumatic reactions. Specifically, higher pre-MVC sleep reactivity predicted PTSD and major depressive episodes at 8 weeks (Neylan et al., 2021). While consistent with the role of sleep reactivity in depression (Kalmbach et al., 2018), their novel findings extend cross-sectional research on sleep reactivity in PTSD (Sanchez et al., 2020) by indicating pre-trauma sleep reactivity prospectively predicts posttraumatic psychopathology. In doing so, their study suggests sleep reactivity might help to identify individuals at risk of dysfunction following stressful and potentially traumatic events. Taken with emerging evidence that behavioural treatment effectively reduces sleep reactivity (Cheng et al., 2022), it may then be feasible to target sleep reactivity to prevent these deleterious outcomes.

One intervention that has shown promising preventative effects is digital cognitive behavioural therapy for insomnia (dCBT-I). dCBT-I is the delivery of CBT-I in a fully automated, online format via a computer and/or mobile device. Like its face-to-face counterpart, dCBT-I reliably improves insomnia (Zachariae et al., 2016), and accumulating evidence shows that ameliorating insomnia produces an "upward spiral" effect, whereby improved sleep begets broad, durable improvements across other health domains (Batterham et al., 2017; Cheng et al., 2021; Cheng, Kalmbach, et al., 2019). For instance, in our previous investigation, dCBT-I improved resilience that in turn buffered against insomnia and depression 1 year later (Cheng et al., 2022). Importantly, this protective effect was achieved, in part, by reducing sleep reactivity. Thus, sleep reactivity is a modifiable risk factor that could be targeted via dCBT-I to promote resilience to the COVID-19 pandemic.

The current study tested sleep reactivity as a prospective predictor of COVID-19 pandemic-related distress and built on the above work by addressing two important limitations. First, Neylan et al. assessed pre-MVC sleep reactivity retrospectively while participants were in the emergency department, thus potentially introducing recall bias (Neylan et al., 2021). Second, the researchers used an abridged measure of the sleep reactivity construct to minimise participant burden. In contrast, the current study used a full, psychometrically sound measure of sleep reactivity assessed *prior* to the COVID-19 pandemic in 2016–2017 to evaluate the predictive value of sleep reactivity. In addition, we further extended the aforementioned study with a preliminary test of dCBT-I modifying the relationship between sleep reactivity and pandemic-related distress (i.e. stress reactions and depressive symptoms). Consistent with the literature reviewed, we expected that higher pre-pandemic sleep reactivity would predict more severe stress reactions and depressive symptoms during the pandemic. Furthermore, we hypothesised that receiving dCBT-I in 2016–2017 would diminish the predictive effects of pre-pandemic sleep reactivity on subsequent pandemic-related distress.

2 | METHOD

2.1 | Participants and procedure

This study utilised data from participants recruited in southeastern Michigan for a randomised controlled trial comparing the efficacy of self-guided digital cognitive behavioural therapy for insomnia (dCBT-I; $n = 358$) against a sleep education control condition ($n = 300$) in treating insomnia (Cheng, Luik, et al., 2019) and in preventing incident depression (SPREAD trial; NCT02988375) (Cheng, Kalmbach, et al., 2019). Participants initially enrolled in the SPREAD trial between 2016 and 2017. Eligible participants met criteria for insomnia disorder via an online screener based on the DSM-5 (American Psychiatric Association, 2013). Participants were excluded from the SPREAD trial if they reported a diagnosis of any untreated sleep disorders besides insomnia (e.g. obstructive sleep apnea, restless legs, 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, Kalmbach, et al., 2019) for addition details). Individuals randomised to the dCBT-I condition completed six sessions of self-guided dCBT-I directed by an animated "virtual therapist" who reviewed and guided the participants' progress. Individuals randomised to the online sleep education condition received six weekly e-mails based on the NIH guide to healthy sleep (National Institutes of Health, 2011).

All participants in the SPREAD trial were eligible for follow-up in the present study (Cheng et al., 2021). Email invitations were sent during the last week of April 2020, 5 weeks into Michigan's stay-at-home order, with approximately 40,000 cases and 3800 deaths reported across the state (Dong et al., 2020). Enrolment was closed in the first week of May 2020 after achieving the target sample of 200 participants (final $N = 208$ [dCBT-I: $n = 102$; control: $n = 106$]; 78.4% women, $M_{age} = 44.67$, SD = 14.13, range = 18–80). Participants reported being mostly White (71.2%), followed by Black (23.1%), and Other (5.7%). Informed consent was obtained from all participants, and all procedures were approved by the Institutional Review Board at Henry Ford Health.

2.2 | Measures

2.2.1 | Pre-pandemic sleep reactivity

Participants reported their pre-pandemic levels of sleep reactivity in 2016–2017 using the Ford Insomnia Response to Stress Test (FIRST) (Drake et al., 2004). The FIRST is a nine-item questionnaire of sleep reactivity as operationalised by a vulnerability to experience sleep disturbances when faced with different stressors (e.g. "After an argument"). Participants reported the likelihood of having difficulty sleeping when experiencing stressors using a four-point scale ranging from not likely (1) to very likely (4), with higher values indicating greater sleep reactivity prior to the pandemic. The FIRST has good psychometric properties (Kalmbach et al., 2018), including temporal stability and test-retest reliability across stressors (Drake et al., 2014; Jarrin et al., 2016), making it an ideal candidate risk factor for psychological dysfunction during an ongoing pandemic marked by changing and unpredictable challenges. Further, there is evidence the FIRST measures a unique sleep-specific component of stress reactivity that is distinct from general trait hyperarousal (Jarrin et al., 2014). We used the pre-pandemic FIRST as our predictor of pandemic outcomes by computing both a sum score (range = 9–36) and a dichotomised variable, with scores ≥18 indicating high sleep reactivity prior to the pandemic (Kalmbach, Pillai, Arnedt, Anderson, & Drake, 2016). The internal consistency for the FIRST in this study was good ($\alpha = 0.88$).

2.2.2 | Pandemic-related stress reactions

Participants reported their stress reactions to the pandemic using the Impact of Events Scale – Revised (IES-R) (Weiss, 2007). The IES-R is a 22-item questionnaire that measures distress over the past week in response to a stressful or potentially traumatic event along three symptom clusters: intrusions, hyperarousal, and avoidance. We tailored the IES-R instructions to ask participants about their stress reactions to "the COVID-19 pandemic", specifically (e.g. "I had waves of strong feelings about it"). Participants indicated how distressed they were by each stress reaction using a five-point scale ranging from not at all (0) to extremely (4), with higher scores indicating more severe stress reactions to the pandemic.

The IES-R is regarded as a measure of post-trauma phenomena rather than general distress (Beck et al., 2008). It has good psychometric properties across several trauma-exposed samples, including convergent validity with PTSD symptoms and the ability to discriminate between individuals with and without interview-assessed PTSD (Adkins et al., 2008; Beck et al., 2008; Rash et al., 2008). Several cut-offs have been developed for the IES-R to facilitate interpretation of stress reaction severity: IES-R scores ≥24 indicate clinical concern (Asukai et al., 2002), and scores ≥33 indicate clinically significant impairment, providing diagnostic sensitivity of 0.91 and specificity of 0.82 in detecting DSM-IV PTSD (Creamer et al., 2003). We utilised these cut-offs in addition to the total IES-R score (range = 0–88) as our outcome of maladaptive stress reactions to the pandemic, and mean scores from each subscale in follow-up analyses. The internal consistency for the overall IES score and its subscales in this study were as follows: total score $\alpha = 0.91$; intrusions $\alpha = 0.91$; hyperarousal $\alpha = 0.85$; avoidance $\alpha = 0.73$.

It is important to note that the original IES was developed at a time that predated the inclusion of PTSD as a diagnosis in the DSM-III (American Psychiatric Association, 1980), and the most recent version (IES-R) queries about "stressful life events" that may or may not meet full diagnostic criteria for a DSM-5 Criterion A trauma, an ICD-11 PTSD qualifying event, or both (Norrholm et al., 2021). However, the IES-R remains an effective tool for assessing the distressful impact of a significant life event such as the COVID-19 pandemic, at least as a screening instrument, whereas ultimate PTSD diagnostic status should be evaluated clinically with full measures and interview.

2.2.3 | Pandemic-concurrent depressive symptoms

Participants reported their depressive symptoms during the pandemic using the 16-item Quick Inventory of Depressive Symptomatology self-report (QIDS-SR_{16}) (Rush et al., 2003). Participants indicated the severity of their depression over the past week using a 4-point scale, with higher scores indicating more severe symptoms. The QIDS-SR_{16} is a reliable and validated instrument for measuring depressive symptoms (Reilly et al., 2015). Scores on the QIDS-SR_{16} range from 0 to 27, with scoring criteria ranging from 0 to 5 (normal), 6 to 10 (mild), 11 to 15 (moderate), 16 to 20 (severe), and 21 to 27 (very severe) (Rush et al., 2003). We used both the QIDS-SR_{16} total score as our outcome of depressive symptoms and a cut-off score ≥ 11 to detect clinically significant depression during the pandemic (Lamoureux et al., 2010). The internal consistency for the overall QIDS-SR_{16} score was good ($\alpha = 0.81$).

2.2.4 | Degree of COVID-19 impact

When assessing for the ability of individual characteristics to predict adjustment to adversity, it is important to consider exposure severity to the event itself (Bonanno, 2020; Chen et al., 2020). Therefore, we used a modified version of the Life Events Checklist (LEC) (Weathers et al., 2013) to isolate the predictive effects of sleep reactivity beyond the degree to which participants were impacted by COVID-19.

Participants were presented with three events on the LEC: (1) exposure to the coronavirus; (2) life-threatening illness or injury related to the coronavirus; and (3) severe human suffering related to the coronavirus. Participants indicated the degree to which they experienced any event along the following response scale: (1) it happened to me; (2) I witnessed it happening to someone else; (3) I learned about it happening to a close friend or family member; (4) I was exposed to it as part of my job; (5) not sure; or (6) it does not apply to me. Direct impact from COVID-19 was operationalised as any endorsement of responses 1 to 4 on at least one of the three items described and used as a covariate.

2.3 | Data analysis

We first screened for data quality and examined correlations and descriptive statistics using IBM SPSS version 21 (Armonk, NY). We then performed regression models in R using the *stats* package (R Core Team, 2021). We ran the following models:

1. hierarchical linear regressions to analyse pre-pandemic sleep reactivity (FIRST sum score) as a predictor of pandemic-related stress reactions (IES-R sum score) and pandemic-concurrent depression (QIDS-SR_{16} sum score);
2. logistic regressions to estimate the odds ratio (OR) of reporting clinically significant stress reactions (IES-R score ≥ 33) and depressive symptoms (QIDS-SR_{16} score ≥ 11) during the pandemic among those with high pre-pandemic sleep reactivity (FIRST score ≥ 18); and
3. moderation analyses to test whether receiving dCBT-I in 2016–2017 modified the relationship between pre-pandemic sleep reactivity and later distress. Specifically, we tested a two-way interaction between pre-pandemic sleep reactivity (FIRST sum score) and treatment condition (0 = control, 1 = dCBT-I) in predicting pandemic-related stress reactions (IES-R sum score) and pandemic-concurrent depressive symptoms (QIDS-SR_{16} sum score).

All analyses covaried for treatment condition and COVID-19 impact (0 = no direct impact, 1 = direct impact). We also assessed age in years (from 2020) and sex as potential covariates and retained each when significant. Additional sensitivity analyses were conducted to assess changes in non-sleep symptoms; we ran models with and without the sleep items from the IES-R (intrusion and hyperarousal subscales; items 2, 15, and 20) and QIDS-SR_{16} (items 1–4). Beta weights (β) represent standardised regression coefficients.

Performing research during a pandemic might be prone to selection bias (Sullivan, 2020; Zhao et al., 2021). Hence, we utilised 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. This sampling weight approach is described in more detail in our previous publication (Cheng et al., 2021).

3 | RESULTS

3.1 | Sample characteristics

This sample's average FIRST score was above the 18-point cut-off, indicating high levels of sleep reactivity prior to the pandemic. The average IES-R exceeded the 24-point cut-off indicating clinically concerning stress reactions to the pandemic, and 30.3% of participants scored above the 33-point threshold suggestive of clinically significant and impairing stress reactions. Lastly, the average QIDS-SR_{16} score during the pandemic was nearing the 11-point cut-off marking the lower bound of the moderate depressive symptom range.[1] All other descriptive statistics and correlations are presented in Table 1.

Most participants (67.3%) reported being directly impacted by COVID-19. The breakdown of exposure severity among this portion of the sample is presented in Table 2.

3.2 | Sleep reactivity predicts pandemic-related stress reactions 3–4 years later

Our first linear regression modelled pre-pandemic sleep reactivity predicting pandemic-related stress reactions (see Table S1). As expected, higher pre-pandemic sleep reactivity predicted more severe stress reactions to the pandemic (β = 0.13, ± 0.07 SE, $p = 0.045$). Age and sex were not significant covariates. Follow-up analyses indicated the effect of pre-pandemic sleep reactivity was most prominent for hyperarousal (β = 0.17, ± 0.07 SE, $p = 0.012$) and intrusions (β = 0.15, ± 0.07 SE, $p = 0.027$), but not significant for avoidance.[2]

We then conducted a logistic regression to estimate the odds of reporting clinically significant stress reactions for individuals with high pre-pandemic sleep reactivity (see Table S2).[3] The findings revealed that individuals with higher pre-pandemic sleep reactivity had over twice the odds of reporting clinically significant stress reactions to the pandemic (OR = 2.65, 95% CI [1.51, 4.67]).

3.3 | dCBT-I mitigates effect of sleep reactivity on pandemic-related stress reactions

Our moderation analyses revealed that receiving dCBT-I prior to the pandemic exerted a protective effect against pandemic-related stress

TABLE 1 Descriptive statistics and bivariate correlations among study variables

	Scale[a]	1	2	3	4	5	6	7	8
1.	COVID-19 impact	–							
2.	Sleep reactivity	0.05	–						
3.	Stress reactions	0.27**	0.16*	–					
4.	Intrusions	0.33**	0.17*	0.91**	–				
5.	Hyperarousal	0.20**	0.19**	0.91**	0.80**	--			
6.	Avoidance	0.11	0.02	0.69**	0.39**	0.47**	–		
7.	Depression	0.13	0.22**	0.68**	0.59**	0.75**	0.36**	–	
8.	Age	−0.12	−0.16*	−0.14*	−0.09	−0.14*	−0.13	−0.19**	–
Min		0	9	0	0	0	0	2	18
Max		1	36	77	3.63	3.83	3.38	24	80
Mean		0.67	24.70	26.84	1.34	1.33	1.02	10.69	44.67
Median		1.00	25.00	25.00	1.25	1.17	0.88	10.00	45.00
SD		0.47	6.67	14.47	0.87	0.91	0.58	4.35	14.13

Pearson and point-biserial correlations. COVID-19 impact = severity of COVID-19 exposure (no direct impact = 0, direct impact = 1); sleep reactivity = Ford Insomnia Response to Stress Test sum score; stress reactions = pandemic-related stress reactions (Impact of Events Scale – Revised (IES-R) sum score); intrusions, hyperarousal, and avoidance = IES-R subscale mean scores; depression = pandemic-concurrent depressive symptoms (16-item self-report Quick Inventory of Depressive Symptomatology); age = age in years.

[a]All variables measured in April–May 2020, except sleep reactivity (measured between 2016 and 2017).

*$p < 0.05$. **$p < 0.01$.

TABLE 2 Breakdown of COVID-19 exposure severity

COVID-19 event	It happened to me	I witnessed it happening to someone else	I learned about it happening to a close friend or family member	I was exposed to it as part of my job
Exposure to coronavirus	10.3%	10.3%	64.4%	14.9%
Life-threatening illness or injury related to the coronavirus	5.7%	16.1%	73.6%	4.6%
Severe human suffering related to the coronavirus	5.6%	20.2%	68.5%	5.6%

Data are based on a modified version of the Life Events Checklist.

reactions among individuals with high pre-pandemic sleep reactivity (β = −0.35, ± 0.13 SE, $p = 0.008$), such that they reported significantly lower levels of stress reactions to the pandemic if they received dCBT-I in 2016–2017 rather than the sleep education control (see Figure 1).[4] Table S3 contains all results from the final step of the moderation model.

3.4 | Sleep reactivity predicts pandemic-concurrent depressive symptoms 3–4 years later

Our second linear regression examined pre-pandemic sleep reactivity as a predictor of depressive symptoms during the pandemic (see Table S1). Consistent with our hypothesis, pre-pandemic sleep reactivity significantly predicted pandemic-concurrent depressive symptoms (β = 0.22, ± 0.07 SE, $p = 0.001$).[5] Age was retained as a covariate in this and subsequent models of depression, but sex was not significant.

Our final logistic regression estimated the odds of reporting clinically significant depressive symptoms for individuals with high pre-pandemic sleep reactivity (see Table S2). Like our previous model, individuals with high sleep reactivity before the pandemic were over twice as likely to report clinically significant depressive symptoms during the pandemic (OR = 2.72, 95% CI [1.69, 4.38]), even while adjusting for age (OR = 2.58, 95% CI [1.59, 4.16]).

3.5 | dCBT-I does not mitigate effect of sleep reactivity on pandemic-concurrent depression

Conversely, prior dCBT-I did not significantly moderate the relationship between pre-pandemic sleep reactivity and subsequent depression during the pandemic, although the beta was in the hypothesised direction (β = −0.16, ± 0.14 SE, $p = 0.232$).[6] Said differently, individuals with high pre-pandemic sleep reactivity reported comparable levels of depression during the pandemic, regardless of whether they received dCBT-I or the sleep education control in 2016–2017 (see Figure 2). See

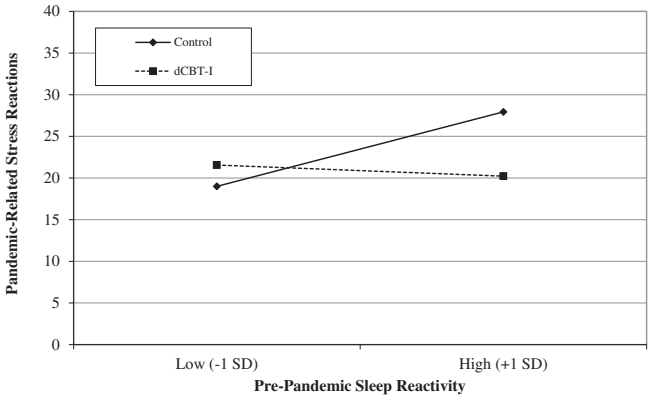

FIGURE 1 dCBT-I buffers the effect of pre-pandemic sleep reactivity on pandemic-related stress reactions. Control = sleep education; dCBT-I = digital cognitive behavioural therapy for insomnia; pandemic-related stress reactions = impact of events scale – revised sum score (measured in April–May 2020); low versus high pre-pandemic sleep reactivity = 1 standard deviation below and above the mean of the Ford Insomnia Response to Stress Test sum score (measured in 2016–2017). Model adjusted for severity of COVID-19 exposure (no direct impact = 0, direct impact = 1)

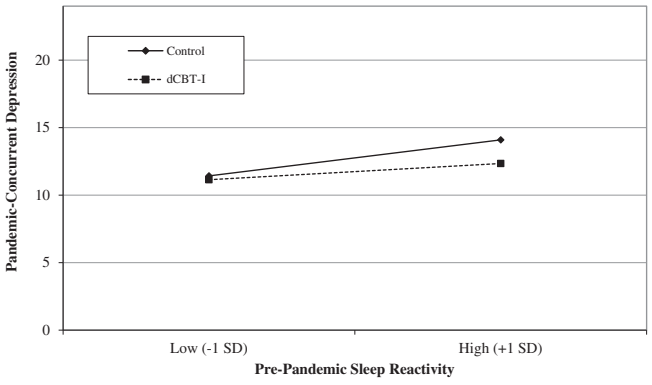

FIGURE 2 dCBT-I does not buffer the effect of pre-pandemic sleep reactivity on pandemic-concurrent depression. Control = sleep education; dCBT-I = digital cognitive behavioural therapy for insomnia; pandemic-concurrent depression = 16-item self-report quick inventory of depressive symptomatology sum score (measured in April–May 2020); low versus high pre-pandemic sleep reactivity = 1 standard deviation below and above the mean of the Ford Insomnia Response to Stress Test sum score (measured in 2016–2017). Model adjusted for severity of COVID-19 exposure (no direct impact = 0, direct impact = 1), as well as age during the pandemic (in years) because it was a significant covariate with depression (results did not vary substantially after removing age from model)

Table S3 for all results from the final step of both moderation models.

4 | DISCUSSION

This study tested pre-pandemic sleep reactivity as a prospective predictor of distress 3–4 years later, early into the COVID-19 pandemic's first wave in the USA, and whether receiving dCBT-I in 2016–2017 prevented these outcomes. As expected, sleep reactivity from 2016 to 2017 predicted more severe stress reactions and depressive symptoms in response to, and during, the pandemic in April–May 2020. More precisely, compared with low reactive sleepers, individuals who were highly reactive sleepers prior to the pandemic were over twice as likely to report clinically significant stress reactions and depressive symptoms one month into the pandemic. These effects were independent of the degree to which COVID-19 impacted participants, thereby supporting sleep reactivity as an important predictor of pandemic-related adjustment that cuts across exposure severity (Bonanno, 2020). Our findings also build on previous evidence that sleep reactivity predicts posttraumatic sequelae (Neylan et al., 2021) and demonstrate its predictive utility generalises to other significant and novel stressors. Moreover, receiving dCBT-I prior to the pandemic mitigated the predictive effect of pre-pandemic sleep reactivity on stress reactions to the pandemic. Taken together, sleep reactivity may identify individuals at risk of clinically significant distress following stressors and potential trauma, and dCBT-I could be one preventative intervention to enhance resilience among this vulnerable group.

Sleep reactivity reflects a predisposition to difficulties falling and/or staying asleep when confronted with a stressor or challenge (Drake et al., 2004). As such, one explanation for our findings might be found in the way disturbed sleep impacts the ability to cope with stressors (Vandekerckhove et al., 2018). When stressed, highly reactive sleepers exhibit decreased rapid eye movement (REM) sleep and increased nocturnal arousals (particularly during REM) (Petersen et al., 2013). It is possible these sleep disruptions hinder the overnight lowering of noradrenaline during REM, resulting in elevated noradrenergic tone during the day that helps to sustain the intensity of emotional experiences and exacerbates affective reactivity (Krause et al., 2017; Vandekerckhove et al., 2018). This aligns with our exploratory findings that sleep reactivity predicted the specific stress responses of intrusions (e.g. "I thought about it when I didn't mean to") and hyperarousal (e.g. "I was jumpy and easily startled"). Though more work is needed to test these putative mechanisms, our finding that prior insomnia treatment prevented stress reactions associated with sleep reactivity underscores the influential role of sleep in this relationship.

Relative to the sleep education control, individuals with high pre-pandemic sleep reactivity who received dCBT-I in 2016–2017 exhibited significantly lower levels of pandemic-related stress reactions in 2020. However, receiving dCBT-I did not significantly alter depression severity during the pandemic for individuals who were reactive sleepers before the pandemic. This suggests dCBT-I confers protective effects for reactive sleepers against maladaptive stress reactions, but not depression. This is difficult to reconcile with our previous study among this same sample, in which we found dCBT-I resulted in less severe depressive symptoms during the pandemic (Cheng et al., 2021). Perhaps the inclusion of sleep reactivity in the current study accounts for this discrepancy. However, reactive sleepers' risk for depression is largely mediated by sleep disturbances (Kalmbach et al., 2018), and so it is unclear why treating insomnia would be

insufficient in alleviating the depression risk associated with sleep reactivity. Alternatively, it is conceivable that we had insufficient power for these analyses, especially considering dCBT-I interacted with sleep reactivity in the expected direction (i.e. predicting less depression). Additional research should be carried out in larger samples before drawing firm conclusions about these preliminary data.

Overall, our results converge with those of Neylan et al., who reported sleep reactivity predicted PTSD and major depressive episodes 8 weeks after motor vehicle collisions (MVCs) (Neylan et al., 2021). Due to their study's design in recruiting acutely traumatised patients, however, pre-MVC sleep reactivity was assessed retrospectively while in the emergency department and using an abridged version of the FIRST. Our study therefore builds on theirs by using pre-event data on the full FIRST as a prospective predictor of post-event distress. Further, our use of pandemic outcomes, measured 3–4 years after the FIRST, provides evidence that the predictive effect of sleep reactivity is both generalisable to a unique stressor and stable across relatively distal timepoints. Our findings also indicate sleep reactivity was not simply predicting the sleep disturbances associated with either depression or the hyperarousal and intrusion stress reactions, as they remained significantly related even when these sleep items were removed from their respective scales. Taken together, sleep reactivity appears to be a potential risk factor for deleterious outcomes after traumatic and adverse life events that could be prevented with insomnia treatment. Individuals most vulnerable to experiencing dysfunction following stressful life events may be identified a priori using the FIRST and subsequently triaged by high versus low sleep reactivity to receive CBT-I to prevent or minimise posttraumatic sequelae. More studies are needed to evaluate this approach, however, including whether CBT-I confers resilience when delivered shortly *after* trauma exposure (e.g. emergency department patients).

4.1 | Limitations and strengths

Our findings must be interpreted within the context of several limitations. First, our sample had insomnia during our assessment of pre-pandemic sleep reactivity. Individuals with insomnia have significantly elevated FIRST scores relative to those without a lifetime history of the disorder (Kalmbach, Pillai, Arnedt, & Drake, 2016), perhaps due in part to the development of insomnia itself (Kalmbach et al., 2016). Although this limits the generalisability of our study, it is notable that our findings nonetheless dovetail with those from trauma survivors presenting to the emergency department (Neylan et al., 2021). Taken together, this convergent evidence supports a sensitive sleep system as a prognostic indicator for future clinically significant distress following adversity.

Second, our data on pandemic-related functioning were collected at a single timepoint shortly after the onset of the pandemic. Thus, although we discuss sleep reactivity as a potential risk factor for psychological distress during the pandemic, it is possible we captured transient reactions that would have remitted over repeated assessments or later waves of the pandemic. Future research may examine how sleep reactivity predicts outcome trajectories across multiple timepoints to elucidate its relationship with longer-term adjustment to adversity, including how it distinguishes between individuals with acute, delayed, or chronic reactions (Bonanno, 2020). Still, our finding that pre-existing sleep reactivity predicts variability in post-event distress satisfies a necessary step toward including sleep reactivity in studies designed to forecast risk for psychopathology following potential trauma (Chen et al., 2020). Moreover, despite the proliferation of research uncovering predictors of pandemic-related distress, much of this relies on cross-sectional data that severely restricts their generalisability (Manchia et al., 2022). This study addresses an urgent need for researchers to utilise prospective designs to more precisely uncover variables relevant to psychological adjustment during the COVID-19 pandemic (Bonanno, 2020).

Third, we must emphasise our outcomes on stress reactions do not necessarily reflect PTSD symptoms. This is because the COVID-19 pandemic subsumes myriad experiences of variable intensity that do not always meet the Criterion A definition of trauma required for PTSD, as outlined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (American Psychiatric Association, 2013; Norrholm et al., 2021). Additional work using more methodologically rigorous designs (e.g. interviews) is needed to delineate adequately the conditions under which individual COVID-19 experiences qualify as Criterion A traumas. Even so, our use of the LEC allowed us to characterise participants' COVID-19 experiences more precisely, revealing that nearly three quarters of our sample learned about a close friend or family member enduring life-threatening illness or injury related to COVID-19. This distinguishes the relatively acute nature of our sample's COVID-19 exposure from the lower magnitude, chronic threat of potential exposure befalling the broader population (Norrholm et al., 2021). Taken together with previous evidence sleep reactivity predicts PTSD onset (Neylan et al., 2021), these data collectively point to sleep reactivity as a candidate risk factor for PTSD warranting further investigation.

5 | CONCLUSION

A pre-existing vulnerability to sleep disturbances (sleep reactivity) was related to more distress 3–4 years later in response to, and during, the COVID-19 pandemic. More specifically, the odds of reporting clinically significant stress reactions and depression in April–May 2020 were over twice as high among individuals who were highly reactive sleepers in 2016–2017. Yet, receiving dCBT-I in 2016–2017 buffered the effects of pre-pandemic sleep reactivity on subsequent stress reactions to the pandemic (but not depression). Sleep reactivity may detect individuals most vulnerable to dysfunction after adversity and implementing dCBT-I prior to stressors may promote resilience among this at-risk group.

AUTHOR CONTRIBUTIONS

Concept and Design: Reffi, Cheng, Casement, Drake. Acquisition, Analysis, or Interpretation of data: Cheng, Reffi, Casement. Drafting

of manuscript: Reffi, Cheng. Critical revision of the manuscript for important intellectual content: Drake, Kalmbach, Jovanovic, Norrholm, Casement, Roth, Cheng.

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CONFLICT OF INTEREST

None declared.

DATA AVAILABILITY STATEMENT

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

ORCID

Anthony N. Reffi https://orcid.org/0000-0002-2783-1678
David A. Kalmbach https://orcid.org/0000-0003-3107-0522
Melynda D. Casement https://orcid.org/0000-0002-5906-0994

ENDNOTES

[1] Comparable to pre-treatment QIDS-SR_{16}: $M = 11$, $SD = 4.69$.

[2] After removing the sleep items from the IES-R, the linear relationship between pre-pandemic sleep reactivity and the overall IES-R became null ($\beta = 0.11$, ± 0.07 *SE*, $p = 0.116$), yet pre-pandemic sleep reactivity remained a significant predictor of both the hyperarousal ($\beta = 0.14$, ± 0.07 *SE*, $p = 0.042$) and intrusions subscales ($\beta = 0.14$, ± 0.06 *SE*, $p = 0.034$). This suggests the relationship between sleep reactivity and the overall IES-R may have been obfuscated by the inclusion of the avoidance subscale, while also highlighting that the relationship between sleep reactivity, hyperarousal, and intrusions is not specific to the sleep disturbances associated with these symptom clusters.

[3] Logistic regression models were not re-run with sleep items omitted from the IES-R and QIDS-SR_{16} because doing so would alter these scales' psychometrically derived cutoff scores we used for our outcomes.

[4] Moderation results remained consistent after removing the sleep items from the IES-R ($\beta = -0.38$, ± 0.13 *SE*, $p = 0.004$).

[5] Linear regression results remained consistent after removing the sleep items from the QIDS-SR_{16} ($\beta = 0.20$, ± 0.07 *SE*, $p = 0.002$).

[6] Moderation results remained consistent after removing the sleep items from the QIDS-SR_{16} ($\beta = -0.16$, ± 0.14 *SE*, $p = 0.240$).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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