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An Analysis of Sleep Disturbances Interacting with Rumination and Childhood Adversity as a Longitudinal Predictor for Depression and Anxiety

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Abstract: During the early stages of the COVID-19 pandemic, many individuals experienced turbulent changes in their mental wellbeing and daily routines. To investigate whether and how sleep disturbance predicted depression and anxiety during this unprecedented public health crisis, this study used a longitudinal dataset (N = 1,518) spanning across the first two years of the pandemic, from March 2020 to November 2021. Additionally, this study examined how rumination and childhood adversity may interact with sleep disturbance to prospectively affect depression or anxiety. Depression and anxiety at the end of 2020 and 2021 were regressed on sleep disturbance, at the beginning of 2020 and 2021, in interaction with rumination and childhood adversity as predictor variables. It was determined that high rumination always exacerbated the relationship between sleep disturbances and affective psychopathology. On the other hand, the 2021 models showed that high childhood adversity, when paired with low rumination, can actually have an inverse effect on the aforementioned correlation. If these results can be explained by the potentially inoculating effect of childhood adversity, then these results may imply potential areas of research in combining current psychiatric inoculation strategies with cognitive behavioral therapy, a way to remedy rumination, as a preventative measure for those at high risk for depression or anxiety.

Keywords: sleep disturbance, rumination, adverse childhood experience, anxiety, depression, COVID-19 pandemic

1. Introduction

1.1. Pre-Pandemic Studies on Sleep and Psychopathology

The relationship between sleep and mental health has long been a field of study, even during pre-pandemic times, since a large number of psychological disorders are accompanied by side effects that disturb sleep. As evidenced by a meta-analysis of 177 studies, most patients with psychiatric disorders have sleep total time and efficiency that differed from control groups [1]. Patients with major depressive disorder tend to have trouble falling and staying asleep, as well as insufficient sleep quality and nightmares; those in the manic phase of bipolar disorder also see decreased need to sleep, but those in the depressed phase tend to have hypersomnia; and those with generalized

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anxiety disorder also have trouble falling and staying asleep [2]. Several studies have confirmed a correlational relationship between the two, including the Benca et al. meta-analysis, which demonstrated that total sleep time and efficiency differ greatly for patients with affective disorders and those without, and a cross-sectional study on American adults established a strong positive correlation between inadequate sleep and frequent mental distress [3]. Few studies have started to shed light on possible causal relationships. For example, a pilot study done on sleep extension in a population of young women who get insufficient sleep showed that those who increased their sleep time showed lessened depressive symptoms and morning sleepiness [4]. Other studies have cited sleep disturbances as a possible risk factor for different forms of psychopathology [5] and stated that the disruptions can be a precursor to psychopathologic deterioration and relapses [6]. However, more studies need to be done to corroborate this potential causal effect and explore possible mediators or moderators.

Inversely, depression and anxiety can be a strong risk factor for insomnia, which is one of the most common sleep disorders [7]. Moreover, a pre-pandemic group-based multi-trajectory model of data from 41,094 participants from the UK Biobank cohort has proven that depressive symptoms are a mediator for the relationship between socio-economic deprivation and poor sleep [8]. Another study that focused on twins aged 8-10 showed that not only can sleep problems at 8 years old predict depression at 10 years old, but genetics have a greater influence on the association between sleep problems and depression at 8 years old, as monozygotic twins showed a much stronger correlation than dizygotic twins at that age. On the other hand, environmental factors had a greater influence on the same association at age 10, showing that genetics' role in the correlation between sleep problems and depression ebb over time [9]. It can be seen that depression and anxiety have a complex correlation with sleep disturbances, which indicates that potential treatments or preventions for one may help with managing the other. This is an important field of potential research because both affective psychopathology and sleep problems are extremely common, including as comorbid conditions, and have profound negative effects on patients' mental and physical health. As there is no current cure for depression or anxiety, gaining insight into preventative measures may be.

1.2. Sleep and Affective Psychopathology During the COVID-19 Pandemic

The onset of the COVID-19 pandemic mainly affected psychopathology and mental wellbeing in one of two ways: 1) through the preventative measures taken to contain the spread of the virus which resulted in major changes in lifestyle for many via the lockdowns and social isolation imposed worldwide during the spring of 2020 [10], or 2) the sequelae of the disease itself and how patients with COVID-19 tend to suffer from psychological distress, likely as a result of prolonged systemic inflammation that puts them at greater risk [11]. Other factors may include external stressors such as financial or social changes, which researchers have found can lead to brain inflammation that can negatively affect mental health even in people uninfected by COVID-19 [12].

Another way the pandemic affected psychopathology was through interactions between individuals and those who have had COVID. A study done in Saudi Arabia showed that there was a significant correlation between COVID-related anxiety and problems affecting routine work. The same study also found that those who had associations with COVID-19 patients were more likely to experience sleep disturbances [13]. Thus, it can be concluded that COVID-19 exacerbated issues of sleep and anxiety in individuals who have associations with COVID-related work or COVID-affected people.

Likewise, sleep was also severely impacted by pandemic safety measures such as working from home and online schooling. The sudden increase in screen time and change in daily routines affected sleep time and quality in many. Electrolyte abnormalities, hyper-inflammation, and

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isolation due to public health concerns are some of the biological and environmental factors that account for sleep disturbances during the pandemic [14]. An early study done in March 2020 in the U.K. found that half the population experienced more disturbed sleep than pre pandemic times [15]; a later cross-sectional online survey-based study found that the rate of sleep disturbances had increased to 69.4% in U.K. adults [16]. An exception to the increase in sleep disturbance during the pandemic was in preschool children, which was lower than pre-pandemic times. However, the literature used in the meta-analysis that came to this conclusion were limited and didn't include sufficient studies done on heterogeneous populations [17].

The correlation between sleep disturbances and affective psychopathology was exacerbated during the pandemic. Medicated insomnia sleepers exhibited greater depression variability and magnitude during the first phases of COVID-related restrictions in March of 2020 than inefficient or healthy sleepers [18]. Not many studies have researched the topic of sleep disturbance and affective psychopathology later on in the pandemic, especially after the initial lockdowns of 2020. Consequently, research comparing the effects of the pandemic in 2020 on sleep and affective psychopathology with those of later time periods, or from 2021 and onwards, is lacking.

Overall, the COVID-19 pandemic both directly - through infection - and indirectly - through safety measures and social changes - affected sleep and affective psychopathology negatively. Furthermore, it impacted existing correlations between sleep disturbances and affective psychopathology, particularly for those already affected by sleep disturbance, depression, or anxiety.

1.3. Childhood Adversity on the Correlation Between Sleep and Psychopathology

Research has shown that people with certain characteristics or experiences are at higher risk for affective psychopathology and sleep disorders, although not many involve all three variables. One such example is the presence of childhood adversity. Adverse childhood experiences (ACE) can include experiences involving harm or threat of harm to the child, such as physical or sexual abuse, domestic violence, or exposure to violence in the community, and experiences that involve deprivation and social disadvantage, such as neglect, the absence or limited availability of a caregiver, poverty and insecure access to food [19]. In cases of individuals with childhood adversity, the population-attributable risk proportions of mental disorders ranged from 15.7% for phobia and panic disorders to 40.7% for behavioral disorders, with an overall association of 28.2% with all psychiatric disorders [20]. Likewise, those with childhood adversity are at higher risk for insomnia and sleep disorders. Not only can it amplify the effects of sleep loss on markers of physical inflammation [21], childhood adversity also leads to higher rates of insomnia in adolescents when compared to those without the same adverse experiences, even after all results were adjusted for recent psychiatric disorders [22].

1.4. Rumination on the Correlation Between Sleep and Psychopathology

Similarly, rumination, which is defined by the APA Dictionary of Psychology as "obsessional thinking involving excessive, repetitive thoughts or themes that interfere with other forms of mental activity," has heavy implications on sleep and psychopathology [23]. High rumination is linked to higher risk of anxiety and depression [24], and a study found that it can be used to predict psychological distress in adolescents. Not only does rumination prolong negative mood, thereby increasing the risk for depression, but it also disrupts sleep -- self-proclaimed poor sleepers also had a higher tendency to ruminate than good sleepers in general [25]. Other meta-analyses have shown correlations between rumination and later sleep onset latency, poorer sleep quality, and shorter total sleep time [26]. A study also demonstrated how rumination directly mediates the relationship between depression and sleep disturbances. Among relatively healthy young adults, depressed

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mood did not directly affect sleep quality, but was rather correlated with rumination, which then predicted sleep quality and self-reported health. This mediation was true regardless of whether or not anxiety was controlled for [27]. Another study confirmed the role of both sleep and rumination in predicting depression, regardless of anxiety (in this study, social anxiety). Likewise, it also showed that the relationship between sleep and social anxiety was mediated by rumination [28]. This proves that rumination plays a significant part in the established predictive correlation between sleep and affective psychopathology. In particular, in the early stages of the COVID-19 pandemic, when psychological wellbeing worsened, lower rumination indicated better mental health across timepoints [29].

These findings together suggest that rumination plays an important role in either directly affecting depression or anxiety, or mediating the correlation between them and sleep disturbances. However, few studies considered the tendency to ruminate as a potential moderator between sleep disturbance and psychopathology.

1.5. The Current Investigation

The current study aims to investigate how sleep disturbance, in interaction with rumination and ACEs, predicted depression and anxiety symptoms during the early phases of the pandemic. Specifically, the study focused on the roles of rumination and childhood adversity as moderators of said prediction. Further, given that stress associated with the pandemic may be qualitatively different between 2020 and 2021, the author examined how sleep disturbances predicted affective psychopathology symptoms in 2020 and 2021, respectively, in order to find how different social contexts may affect the correlation. The current hypothesis is that there will be predictive significance between the predictor variables and affective psychopathology, but that the significance will differ for 2020 and 2021.

2. Methods

2.1. Participants and Procedure

The current study used an open access longitudinal dataset from the Boston College Daily Sleep and Well-Being Survey Data during the COVID-19 Pandemic [30]. The dataset included demographic measures, daily online self-report surveys, and eight rounds of assessments. The daily surveys were self-reported and assessed variables including but not limited to sleep logs, exercise, depression symptoms, and coronavirus symptoms or diagnoses. The eight assessment rounds included several one-time assessments with variables such as sleep disturbances, general anxiety, childhood adversity, and cognitive emotion regulation. The first round was administered on May 19, 2020, and the last round was administered on October 28, 2021, with varied time intervals between rounds. The participants were recruited primarily through social media advertisements and received compensation in the form of raffle entries for gift cards. The participants were English-speaking and were primarily White, female, between 18-90 years of age (M = 37.65, SD = 16.33), and from the United States.

To optimize comparisons between the present study's three main variables of sleep, depression, and anxiety, this study utilized data from Round 1 (May 19, 2020), Round 4 (September 28, 2020), Round 5 (February 27, 2021), and Round 8 (October 28, 2021), as those four rounds included assessments of generalized anxiety and sleep disturbances. The final analyses were based on 827 participants with an age range between 18 and 90 (M = 37.65, SD = 16.33). The next step was to compile the data on depression symptoms based on the daily surveys.

Following this, the author created a 14-day window for each of the rounds (7 days before and after the date of each round) and obtained the average as the depression score for each round. This

DOI: 10.54254/2753-7048/17/20231259

dataset was then combined with the other data from the four assessment rounds based on each participant's unique subject ID. Other assessed variables in these rounds that were used include cognitive emotional regulation (rumination) and childhood adversity.

2.2. Measures

2.2.1. Depression

Depression was assessed via participants' self-reported Patient Health Questionnaire (PHQ-9; [31]), minus the variable of suicidality at the request of the IRB, in the daily surveys. The PHQ-9 assesses an individual's degree of depression severity on a scale of 0 (not at all) to 3 (nearly every day) for each variable. The nine variables of the original questionnaire reflect the nine diagnostic criteria for major depressive disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) that apply to a patient's two-week period. With the removal of the suicidality variable, the total score for the assessed PHQ-9 ranges from 0-24, with higher scores indicating greater depression. To obtain a depression estimate for each round, the author averaged the PHQ-9 total sum scores jia7 days within ± the date of each round assessment.

2.2.2. Generalized Anxiety

The General Anxiety Disorder-7 (GAD-7; [32]) was used to measure generalized anxiety symptoms for each participant. The GAD-7 was administered in all four included rounds, consisting of seven items that measure anxiety symptoms on a scale from 0 (not at all) to 3 (nearly every day) for a total score of 0 (not at all for all seven items) to 21 (nearly every day for all seven items). A higher score indicates higher levels of anxiety, with a score of 10 or higher being a likely indicator for generalized anxiety disorder. For the current study, the total score for each subject was used.

2.2.3. Sleep

The Pittsburgh Sleep Quality Index (PSQI; [33]) is an effective instrument used to measure the quality and patterns of sleep in adults. It differentiates "poor" from "good" sleep quality by measuring seven areas (components): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction over the last month. For the current study, the total score for all the subjects combined was used.

2.2.4. Childhood Adversity

Childhood adversity was measured via the Adverse Childhood Experience questionnaire (ACE; [34]) in round 5. The ACE measures 10 types of childhood adversities with the first five relating to personal circumstances (e.g. abuse or neglect) and the last five relating to familial conditions (e.g. domestic abuse or parental separation). Each variable is assigned one point, so the total possible score ranges from 0-10, with 10 indicating the greatest range of childhood adversities.

2.2.5. Rumination

Rumination was measured by two items from the Cognitive Emotion Regulation Questionnaire (CERQ; [35]): "I am preoccupied with what I think and feel about what I have experienced" and "I often think about how I feel about what I have experienced." Each item of the CERQ was measured on a 5-point Likert scale ranging from 1 (almost never) to 5 (almost always), so the total rumination scores ranged from 2 (almost never for both) to 10 (almost always for both).

DOI: 10.54254/2753-7048/17/20231259

2.3. Data Analytic Plan

In R Version 4.1.2, statistical analyses were performed using the tidyverse, dplyr, DMwR2, sjPlot, and summarytools packages [36][37][38][39][40]. The author combined the datasets of Rounds 1, 4, 5, and 8, including the data on depression from the daily surveys, and performed 10-nearest neighbor imputation on the combined dataset. To ensure the accuracy of imputation, additional measures of psychopathology and emotion regulation were included to facilitate the identification of 10-nearest neighbors for all missing data values except for demographics.

To understand the longitudinal relationships from sleep disturbance, childhood adversity, and rumination to depression and anxiety in 2020 and 2021 separately, the author built two sets of multivariable linear regression models with symptoms of depression and anxiety at Round 4 (September, 2020) and Round 8 (October, 2021) as the outcome variables.

The 2020 set of models used the standardized values of sleep disturbance at Round 5, and placed them in interaction with ACEs and rumination levels to predict depression and anxiety symptoms at Round 4. The author further controlled baseline values of affective psychopathology at Round 1 to ensure that the predictive results were robust. Similarly, the 2021 set of models used the standardized values of sleep disturbance at Round 5 in interaction with ACEs and rumination levels to predict depression and anxiety symptoms at Round 8. The author also controlled baseline values for affective psychopathology at Round 5 to ensure that results were robust.

3. Results

3.1. Predicting 2020 Anxiety and Depression

When predicting depression values at Round 4, baseline depression at Round 1 was the only significant predictor, $\beta = 0.36$, $p < .001$. The interaction of rumination, ACE, and sleep disturbance was not significant, $\beta < .001$, $p > .05$, in predicting depression levels. Similarly, baseline anxiety at Round 1 was the only significant predictor, $\beta = .42$, $p < .001$ for the depression values at Round 4.

When plotted, neither the 2020 depression (Figure 1) nor anxiety (Figure 2) regression models showed any significant association between any of the predictor variable interactions and the outcome variables.

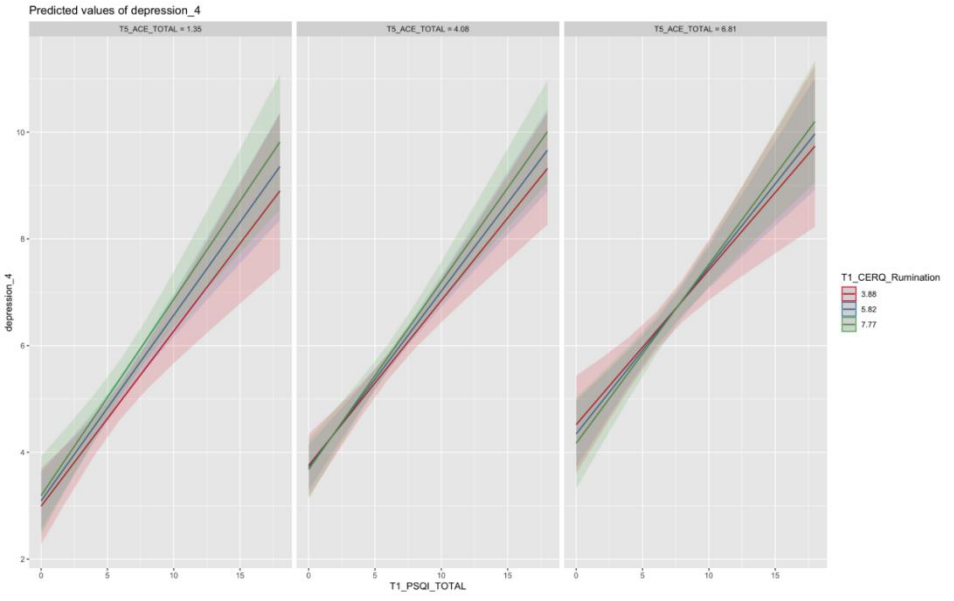

Figure 1: Predictive variables' significance for 2020 depression.

DOI: 10.54254/2753-7048/17/20231259

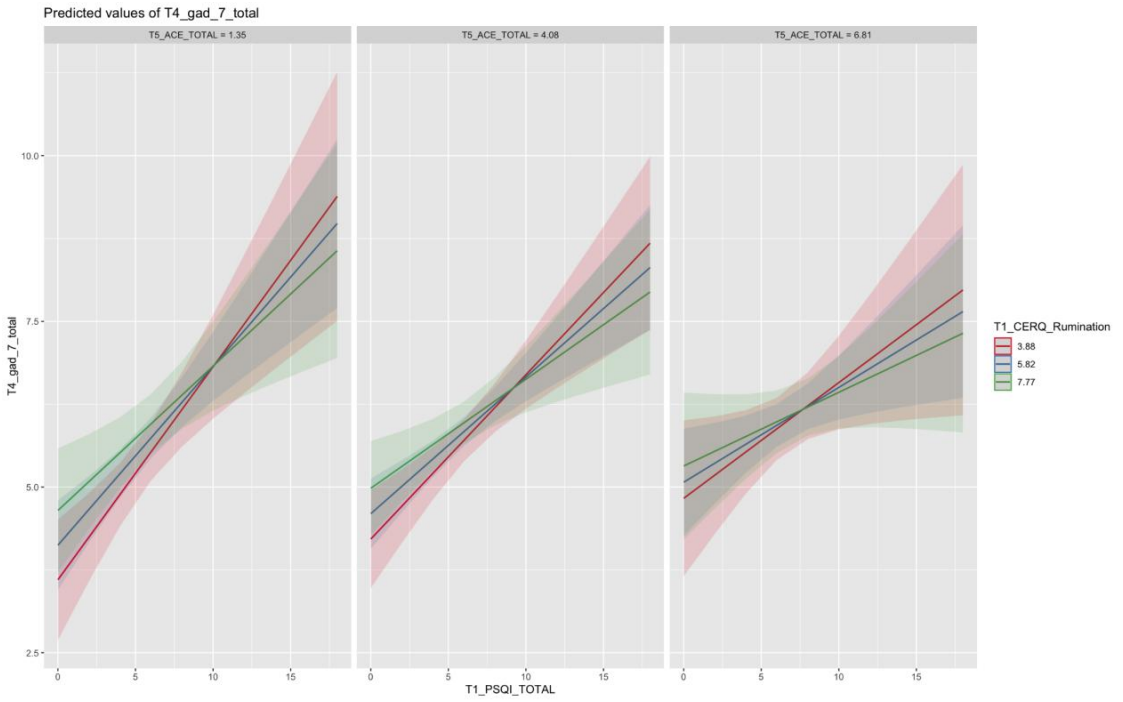

Figure 2: Predictive variables' significance for 2020.

3.2. Predicting 2021 Anxiety and Depression

When predicting Round 8 depression symptoms, baseline depression (Round 5), $\beta = .55$, $p < .001$, and sleep disturbance, $\beta = .37$, $p < .05$, emerged as significant predictors, whereas rumination and ACEs were not significant, $p > .05$. Moreover, there was a significant three-way interaction among sleep disturbance, ACE, and rumination, $\beta = .009$, $p < .05$, as a predictor variable.

Likewise, the 2021 anxiety regression model (Figure 3) showed a strong significance for predictive correlation between baseline anxiety, $\beta = .53$, $p < .001$, ACEs, $\beta = .76$, $p < .01$, and sleep disturbance-ACE-rumination interaction, $\beta = .02$, $p < .05$, with Round 8 anxiety. All standardized beta coefficients for the 2020 and 2021 regression models are listed in Table 1.

The plots for both three-way interaction models, made using the sjPlot package, showed similar trends: those with low ACE scores showed a neutral to positive linear correlation between sleep disturbance and affective psychopathology, regardless of rumination levels. As ACEs increased, the correlation between sleep disturbance and affective psychopathology varied a lot more, on account of different rumination levels. In groups with higher ACE, as rumination decreased, so did the slope of the linear correlation between sleep disturbances and anxiety or depression. Those with higher rumination scores showed the same positive correlation, those with intermediate rumination scores showed a more neutral correlation, and those with lower rumination scores showed a negative correlation between sleep disturbances and affective psychopathology.

DOI: 10.54254/2753-7048/17/20231259

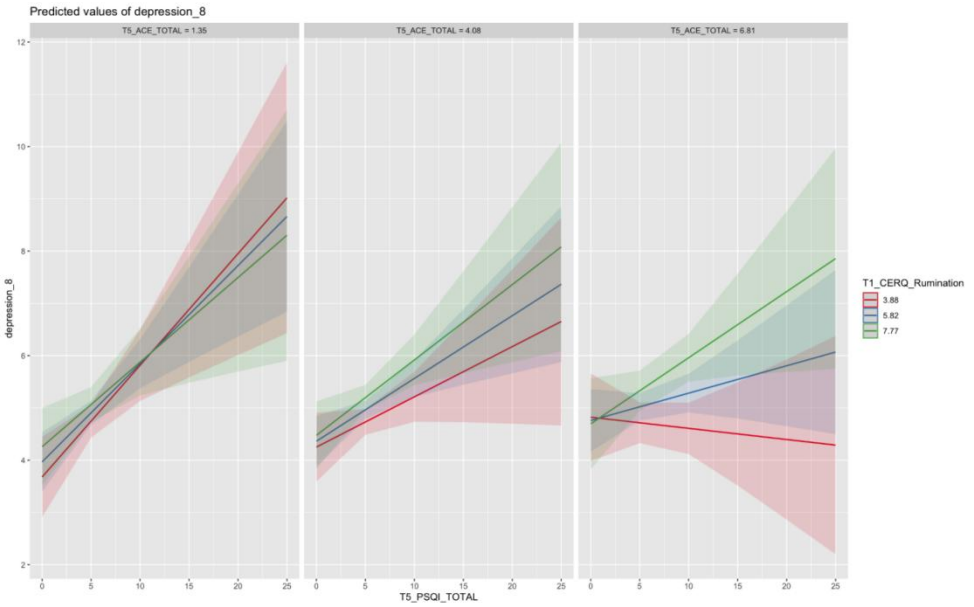

Figure 3: Predictive variables' significance for 2021 depression.

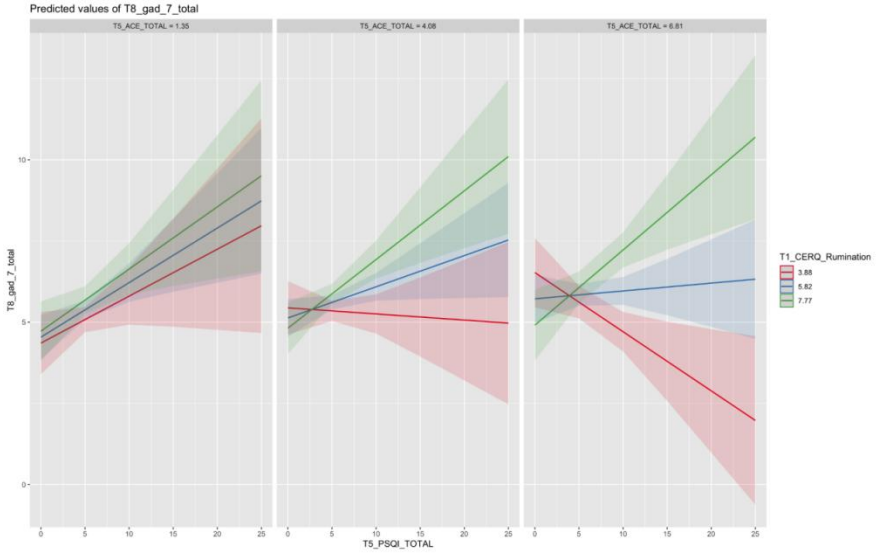

Figure 4: Predictive variables' significance for 2021 anxiety.

Table 1: Predictive significance of sleep, rumination, and ACEs on affective psychopathology.

Predictor Variables	2020 Depression (Standardized β)	2020 Anxiety (Standardized β)	2021 Depression (Standardized β)	2021 Anxiety (Standardized β)
Baseline Depression/Anxiety	.36***	.42***	.55***	.53 ***
Sleep Disturbance	.30	.47*	.37*	.27

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Table 1: (continued).

Rumination	.09	.30	.19	.22
ACE	.34	.33	.34	.76**
Sleep*Rumination* ACE	< .001	.002	.009*	.02**

Note:
*p < .05
**p < .01
***p < .001

4. Discussion

Overall, this study showed that while affective psychopathology and sleep disturbances are mostly positively correlated, the association was much more nuanced than previously established. In particular, there are varying trends of correlation depending on an individual's childhood experiences and emotion regulation as well as social contexts. In 2020, neither rumination nor ACE affected the predictive relationship between sleep disturbances and depression or anxiety; however, in 2021, both rumination and ACE affected how sleep disturbances predicted depression and anxiety. Specifically, at lower levels of ACEs, greater sleep disturbance was associated with more anxiety and depression symptoms, regardless of one's tendency to ruminate; however, at higher levels of ACEs, the results were divergent. Among those who experienced higher ACEs and lower rumination, sleep disturbance was inversely associated with depression and anxiety symptoms, suggesting that those with sleep disturbance may have fewer depression and anxiety symptoms. One possible explanation for this may be that those with higher ACEs yet lower rumination might develop a greater tolerance of distress, making them less susceptible to the influence of sleep disturbances on emotional health.

Consistent with previous literature, both ACEs and rumination played a role in the relationship between sleep disturbances and affective psychopathology, under certain conditions. In particular, higher levels of rumination almost always correlated with, or predicted, higher levels of depression and anxiety [24][41]. Furthermore, consistent with the meta-analyses done by Clancy et al. [26], high levels of rumination exacerbated the correlation between sleep disturbance and affective psychopathology. With the exception of one specific population group, this study was also consistent with existing literature that claims that ACEs are positively correlated with affective psychopathology [42] and sleep disturbances [43]. However, in previous studies examining the association among ACEs, rumination, and affective psychopathology, rumination has often been considered a mediator between sleep disturbance and affective psychopathology [27][44][45]. Similarly, existing studies have mostly studied ACEs as a predictor of sleep disturbance, anxiety, and depression, sometimes with sleep disturbance as a mediator for the correlation with anxiety and depression [46]. One possible yet understudied interaction between these four variables (ACEs, rumination, sleep disturbances, and affective psychopathology) is that rumination, with its relative stability over time, acts as a moderator between sleep disturbance and affective psychopathology, particularly when interacting with ACEs. In this study, the author found that even in populations with high levels of ACEs, those with low rumination exhibited a negative correlation between sleep disturbance and both anxiety and depression, despite the previously established trend of high ACEs exacerbating affective psychopathology. An explanation for this effect may be that the coupling of low rumination and high ACEs may reflect a potential state of resilience against the typically significant negative effects of sleep disturbances. An alternative possibility is such resilience acts

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directly against depression and anxiety, minimizing any effects sleep disturbances may have on them.

These findings may suggest further research combining inoculation techniques, such as stress inoculation training (SIT; [47]), and rumination-focused cognitive behavioral therapy (RFCBT; [48]) in the prevention of depression and anxiety in those at higher risk. In this study, childhood adversity may have acted as a stress inoculator, presenting a vaccine-like effect in individuals with low rumination tendencies by pre-exposing them to, and therefore allowing them to easier adapt to, harsh stressors. Combined inoculation and RFCBT could serve to lower future depression and anxiety levels or risk, as well as minimize any significant impacts sleep disturbances may have on mental wellbeing, in general populations.

This study also showed a significant disparity between the interactions of the predictor, moderator, and outcome variables in 2020 and 2021. In 2020, higher sleep disturbances predicted higher levels of anxiety and depression, regardless of rumination and ACE levels; in 2021, this predictive relationship was affected by both rumination and ACE levels. This difference may be accounted for by the abnormal circumstances in 2020, when the COVID-19 pandemic was still in its early stages and strict safety measures were first implemented. The stress, which then directly correlates to both sleep disturbances and affective psychopathology, created by such sudden and large-scale changes may have outweighed any personal stress levels that could be attributed to rumination or childhood adversity. Once people started adjusting, however, differences in stress levels — and hence, sleep and anxiety and depression — once again reflected individual rumination and ACE levels, as can be seen in 2021. Another possible external factor that could have influenced the sample population's stress levels is the 2020 election. Since a large portion of the sample population used in the given dataset was liberal, former President Donald J. Trump's time in office and the preceding presidential election could also have caused abnormal spikes in stress levels. Simply put, the author proposes that the 2021 depression and anxiety models be considered as more accurate under normal circumstances, for the high-stress circumstances of 2020 seem to negate any significant effects of the predictor variables. Assuming that the 2021 models are mostly accurate, this study could also have implications for identifying individuals more at risk for depression and anxiety by checking for rumination levels and past childhood adversity. Combining these two traits with recent sleep behaviors could help predict depression and anxiety, as well.

These results should be viewed in light of certain limitations of the used dataset. For one, the majority of participants in the Boston College daily sleep and well-being survey data were white, liberal, non-Hispanic women residing in Massachusetts. Other limitations include the small number of participants with high ACE scores, and possible inconsistencies due to individual differences in interpreting questionnaires.

5. Conclusion

This study demonstrated a three-way interaction among sleep disturbance, tendency to ruminate, and childhood adversity as predictors of depression and anxiety in 2020 and 2021. The results indicate that almost all people who exhibit poor sleep are at a higher risk for depression and anxiety, especially those with high rumination and ACEs. The one exception is those with high ACEs and low rumination levels, for whom higher sleep disturbances paradoxically predict lower depression and anxiety. Furthermore, this specific model does not hold true under abnormally high-stress circumstances, in which the positive predictive correlation between sleep disturbance and affective psychopathology is unaffected by rumination and childhood adversity.

The results of this study indicate areas for further research regarding possible prevention methods for depression and anxiety through inoculation, lowering rumination, and remedying sleep disturbances. These findings suggest that, when paired with low rumination, childhood adversity

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serves as an effective inoculator for anxiety and depression later in life. For individuals who have not experienced ACEs, a version of the existing stress inoculation therapy (SIT), currently mainly used to prevent PTSD, can be developed and used to serve as an inoculator for depression and anxiety. Then, the inoculation, whether from therapy or childhood adversity, can be coupled with cognitive behavioral therapy (CBT) or similar methods to lower rumination levels to decrease morbidity rates for depression and anxiety drastically. Among individuals with prominent sleep problems, targeting sleep disturbance (e.g., cognitive behavioral therapy for insomnia) can be fruitful in preventing affective psychopathology. If effective, this prevention method can be administered to those at high risk for depression and anxiety (e.g. individuals with a family history of depression or anxiety, experienced childhood adversity, working in high-stress jobs such as first care, living in areas prone to natural disasters, etc.).

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