THE PENNSYLVANIA STATE UNIVERSITY
SCHREYER HONORS COLLEGE

DEPARTMENT OF PSYCHOLOGY

Why Sleep is Key: Poor Sleep Quality is a Mechanism for the Bidirectional Relationship between Major Depressive Disorder and Generalized Anxiety Disorder Across 18 Years

VIVIAN NGUYEN
SPRING 2021

A thesis
submitted in partial fulfillment
of the requirements
for a baccalaureate degree
in Psychology
with honors in Psychology

Reviewed and approved* by the following:

Michelle Newman
Professor of Psychology
Thesis Supervisor

Frank Hillary
Associate Department Head of Psychology
Professor of Psychology
Honors Advisor

* Electronic approvals are on file.

ABSTRACT

Background: Generalized anxiety disorder (GAD) and major depressive disorder (MDD) reliably precede and predict one another. However, there is insufficient data on *mechanisms* through which the longitudinal GAD-MDD association unfolds. Based on insomnia theories, such as the hyperarousal model of sleep, we tested the degree to which poor global sleep quality functioned as a mediator of the prospective bidirectional anxiety-depression relationship. **Method:** Participants were 3,294 community-dwelling adults who partook in 3 waves of measurement spaced 9 years apart. GAD and MDD were assessed in-person using the Composite International Diagnostic Interview-Short Form at baseline (Time 1 [T1]) and 18 years later (T3). T2 global sleep quality was measured using the multiple-domain Pittsburgh Sleep Quality Index self-report at T2. Longitudinal structural equation modeling mediation analysis was used. **Results:** Analyses showed that higher T1 MDD and GAD severity individually predicted lower T2 global sleep quality (Cohen's d = -0.570–-0.457), and less T2 global sleep quality thereby forecasted both higher T3 MDD and GAD (d = -0.487–-0.422). Poorer T2 global sleep quality significantly mediated the T1 GAD–T3 MDD relation (controlling for T1 MDD), accounting for 83.93% of the association. Poorer global sleep quality at T2 also significantly mediated the T1 MDD–T3 GAD association (controlling for T1 GAD), accounting for 100% of the relation. **Conclusions:** Findings offer evidence for transdiagnostic theories of sleep and insomnia. Theoretical and clinical implications, such as the importance of prioritizing sleep improvement in cognitive behavioral therapies, are also discussed.

Keywords: sleep quality; depression; anxiety; longitudinal; mechanism; comorbidity

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGEMENTS

I would like to extend my gratitude to the people who have been instrumental in helping me write my honors thesis. First, I would like to thank Dr. Michelle Newman, as her expert advice and continuous support throughout this process has made this thesis possible. Additionally, Nur Hani Zainal, a graduate student in the Newman Laboratory, served as an outstanding mentor throughout my undergraduate research experience. Her willingness to teach me new concepts, flexibility with scheduling meetings, and thoughtful comments guided me tremendously in writing this thesis. Lastly, many thanks to Dr. Frank Hillary, my honors advisor, and the Schreyer Honors College staff for enriching my undergraduate education experience.

Chapter 1

Introduction

Approximately one-fifth of the population suffers from anxiety or depressive disorders in a given year (cf. recent meta-analysis on global prevalence rates of mental disorders; Steel et al., 2014). Further, 49 to 81% of those suffering from a depressive disorder will also meet diagnostic criteria for an anxiety disorder in their lifetime, and the opposite relation has been found for 47 to 88% of anxiety-disordered individuals (Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998). When compared with either diagnosis alone, comorbid major depressive disorder (MDD) and generalized anxiety disorder (GAD) have been connected to greater symptom severity, chronicity, and impairment (Kessler et al., 2008a). Simultaneously, MDD and GAD diagnoses have been shown to dovetail with heightened interpersonal distress (Uhmann, Beesdo-Baum, Becker, & Hoyer, 2010), lower income (Kessler et al., 2008b), and a plethora of physical ailments related to immune, cardiorespiratory, and major neurocognitive disorders (Butnoriene et al., 2015; Zainal & Newman, in press-a; Zainal & Newman, in press-b). Due to the consequences of its comorbidity, better understanding of the MDD-GAD relation is essential.

On top of the wealth of research demonstrating concurrent MDD-GAD relations, an abundance of evidence suggests that the relation holds across multiple time-lags. *Prospective comorbidity* theories have proposed that anxiety can provoke hopelessness and helplessness, which in turn prompts future depression (Swendsen, 1997). Similarly, anxiety theorists have posited that higher levels of anxiety results in greater levels of inhibition and prolonged behavioral inactivity, which over time evokes depression (Rosellini & Brown, 2011). Supporting those theories, a recent meta-analysis by (Jacobson & Newman, 2017) pooled across 66 studies observed equally large effect sizes for anxiety disorders predicting future disorders of depression

(odds ratio [OR] = 2.73), and vice versa (OR = 2.77). To date, however, the mechanisms underlying the longitudinal MDD-GAD comorbidity remain understudied.

One potential mechanism could be sleep. The *transdiagnostic cognitive theory of sleep* posits that insomnia is a candidate mediator in explaining the longitudinal relation between GAD and MDD across extended timeframes (Harvey, 2008). The theory asserts that over protracted timescales, insomnia can mediate the GAD-MDD relation as it decreases quality of life, in part due to economic and physical health consequences, such as work absenteeism and over-reliance on medical services. Moreover, sleep deprivation may reduce one's ability to optimally manage emotions and acute stress triggers, contributing to the onset and maintenance of psychological disorders over time. However, depression and anxiety disorders themselves can also aggravate sleep patterns in the long-term future (Gregory et al., 2005; Morphy, Dunn, Lewis, Boardman, & Croft, 2007). Collectively, theory and evidence thus far suggest a *reciprocal* prospective connection between insomnia and anxiety or depressive disorders and symptoms (e.g., up to as long as 50 years) (Jansson-Fröjmark & Lindblom, 2008; Kim et al., 2009; Sivertsen et al., 2012).

Further, the *hyperarousal model of insomnia* (Riemann et al., 2010) postulates that insomnia is a *psychobiological* disorder, involving both psychological disturbances and biological imbalances, across long time-points. Psychological disturbances may manifest as maladaptive sleep behaviors; creating associations between sleep and negative mood, excessive worry, rumination, and dysfunctional beliefs and attitudes toward sleep. Such psychological factors tend to interact with biological disharmony of the neuroendocrine and immunological systems (e.g., excessive cortisol, insulin, and pro-inflammatory cytokines such as interleukin-6) (Cho, Seeman, Kiefe, Lauderdale, & Irwin, 2015; O'Connor et al., 2014; Stahl et al., 2020). On the whole, the model proposes that wear-and-tear of biological systems from poor sleep habits

can account for the bi-directional association between poor sleep quality and depression and anxiety symptoms across long periods.

Five longitudinal studies have examined the proposition that poorer sleep quality precedes and predicts anxiety and depressive disorders. For example, a study of 9,683 Australian young adult women observed that sleep difficulties contributed to the onset, relapse, and maintenance of anxiety and depression disorders following 9 years (Jackson, Sztendur, Diamond, Byles, & Bruck, 2014); however, due to its all-female sample, it is unclear if findings extend to the general population. A study on a Chinese cohort of male college students suggested that possibility, as it found that poor sleep quality at baseline predicted a higher risk of anxiety symptoms at one-year follow-up (Zou et al., 2020). Likewise, a recent study observed that poorer sleep quality forecasted anxiety (OR 95% confidence interval [CI]: 2.38 to 7.53) and depression (OR: 1.54 to 3.71) at three time points throughout all stages of pregnancy in Chinese women (Yu et al., 2017); nonetheless, whether the findings generalize across other cultures remains open to question. Another study investigated whether sleep problems predicted higher depressive symptoms 14 months later in depressed cancer survivors (Hsiao et al., 2013); however, they did not find a relation between baseline sleep problems and future depressive symptoms. Relatedly, a study among Swiss adults observed a prospective link between insomnia and subsequent depressive episodes across 20 years (Buysse et al., 2008). Based on these findings, it is plausible that lower global sleep quality could precede and predict MDD and GAD.

Simultaneously, four studies thus far investigated if anxiety and depressive disorders predicted diverse sleep problems at a later time. For instance, in a study of adolescents who survived the 2008 Wenchuan earthquake, higher anxiety severity predicted poorer sleep quality generally, and shorter sleep duration in particular, after 1.5 years (Geng et al., 2018); despite

that, it is unclear whether such pattern of findings would hold across decades. On a similar note, another study found a prospective relationship between depressive symptoms and sleep disturbance (i.e., shorter vs. longer sleep duration) over four years (Sun et al., 2018). However, the measure of sleep disturbance was limited to sleep duration, and other markers of sleep quality (e.g., daytime dysfunction, sleep latency, use of sleep medications) that could be longitudinally related to anxiety and depression were not examined. Further, a twin study assessed the bi-directional relation between sleep problems (i.e., difficulties falling or staying asleep, excessive sleep, or issues with experiencing restful sleep) and depression (Gregory et al., 2005). Although Gregory and colleagues (2005) found that sleep problems predicted depressive symptoms 2 years later, they did not find the reverse relationship. Also, a study on patients with Parkinson's Disease (PD) observed prospective relations between baseline state anxiety and depressive symptoms and the incidence of insomnia six months later (Rutten et al., 2017); however, the sample comprised only PD patients, thereby limiting generalizability. Taken together, these findings support the idea that anxiety and depressive disorders can predict future sleep problems across long durations.

Building on existing research, this study aimed to investigate if the 18-year longitudinal relations between MDD and GAD severity were mediated by subjective sleep quality problems assessed during mid-point. This study adds to current literature in several ways. First, the current literature consists of mostly cross-sectional studies, which preclude causal inferences due to the absence of temporal precedence. Our longitudinal study over a longer 18-year time-span contributes to a potential cause-effect understanding of the bi-directional relations between sleep problems and MDD or GAD. Second, our study examined community-dwelling individuals using the Diagnostic and Statistical Manual–Third Edition–Revised (DSM-III-R)–aligned

Composite International Diagnostic Interview–Short Form (CIDI-SF). Previous studies investigating the bidirectional MDD–GAD relationship included scales utilizing limited clinical diagnostic criteria (e.g., Hospital Anxiety and Depression Scale) (Jansson-Fröjmark & Lindblom, 2008). Third, we used a measure of global sleep quality that contained different facets of the sleep experience. Fourth, this is the first study to examine if overall sleep quality is a mediator (or mechanism) of the 18-year relation between MDD and GAD severity.

Based on theory and research, we predicted that higher MDD and GAD symptom severity would forecast reduced future global sleep quality (indexed by subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, presence of use of sleep medications, daytime dysfunction) 9 years later. Moreover, we hypothesized that lower global sleep quality would predict greater MDD and GAD symptom severity following 9 years. In addition, we hypothesized that global sleep quality measured at mid-point would mediate the 18-year relations between MDD and GAD symptom severity.

Chapter 2

Methods

Participants

Participants comprised 3,294 community-dwelling adults who consented to take part in the Midlife Development in the United States (MIDUS) project at Time 1 (T1) (1995-1996), T2 (2004-2005), and T3 (2013-2014) (Brim et al., 2019; Ryff et al., 2017; Ryff et al., 2019). Participants were aged 45.62 years on average (*SD* = 11.41, range = 20 to 74 years), 54.61% were females, and 92.64% identified as Whites compared to African Americans, Hispanics, Asians, Pacific Islanders, or others. Since this study is a secondary analysis of a publicly available data set, it was exempt from institutional review board approval.

Procedures

We selected participants who consented to complete in-person clinical interviews to determine their diagnostic status of MDD and GAD at T1 and T3. Participants completed in-person clinical interviews at T1 and T3, as well as a self-report measure of multiple dimensions of sleep quality at T2. Global sleep quality was not assessed at T1 and T3.

Measures

Major Depressive Disorder. MDD severity was measured using the Diagnostic and Statistical Manual–Third Edition–Revised (DSM-III-R)–aligned Composite International Diagnostic Interview–Short Form (CIDI-SF) (Kessler et al., 1998). Participants reported if they experienced depression symptoms (7-item; i.e., loss of interest in pleasurable activities, anhedonia, depressed mood, appetite changes, fatigue, suicidal ideation, sleep disturbance) in the past 12-months by answering ‘Yes’ (coded as ‘1’) or ‘No’ (coded as ‘0’) to each symptom. The MDD symptom severity scale score can thus range from 0 to 6. The CIDI-SF has exhibited good

internal consistency (Cronbach's α = 0.94 for both T1 and T3 in this study), strong retest-reliability, and excellent sensitivity (93.9%), and specificity (89.6%) (Wittchen, 1994).

Generalized Anxiety Disorder. GAD severity was also assessed using the DSM-III-R-consistent CIDI-SF (Kessler et al., 1998). Participants recorded the degree to which they experienced a series of symptoms resulting from their worries for most days in the past year (10-item; i.e., restlessness, feeling keyed up, irritability, trouble sleeping, difficulties focusing, fatigue, muscle tension). The GAD symptom severity score can therefore range from 0 to 10. For GAD, the CIDI-SF has demonstrated good internal consistency (α = .80 at T1 and .83 at T3 in this study), strong retest-reliability, and excellent sensitivity (99.8%) and specificity (89.6%) (Abel & Borkovec, 1995; Wittchen, 1994).

Sleep quality. Past-month sleep quality and disturbances were assessed with the 19-item Pittsburgh Sleep Quality Index (PSQI) self-report (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI comprises 7 components. *Subjective sleep quality* was assessed with a 1-item overall rating of participants' subjective appraisal of sleep quality (1 = *very bad* to 4 = *very good*). *Sleep latency* (2-item; e.g., "During the past month, how long (in minutes has it taken you to fall asleep at night?") assessed the average time one takes to fall asleep. *Sleep duration* (1-item) measured the average number of hours one actually sleeps each night. *Habitual sleep efficiency* (3-item) assessed the percentage of time in bed that was spent actually sleeping (e.g., time participants went to bed and woke up). *Sleep disturbances* (9-item) assessed the frequency with which various scenarios interrupted sleep (i.e., coughing and snoring, bad dreams, pain) (1 = *Three or more times per week* to 4 = *Not during the past month*). *Use of sleeping medication* (1-item) assessed how often one consumed prescribed or "over the counter" medication to assist with sleep (1 = *Three or more times per week* to 4 = *Not during the past month*). *Daytime*

dysfunction (2-item) inquired on degree of difficulty to stay awake (1 = *A very big problem* to 4 = *No problem at all*). Most items were rated on a 0-3 Likert scale, and summed to give a global PSQI score, ranging from 0-21. Higher scores indicate better global sleep quality. The PSQI has been shown to have good internal consistency for the 7 components (α = .83) and 19 individual items (α = .83), high retest reliability (*r* = .85), convergent validity, and discriminant validity (Buysse et al., 1989). In this study, the PSQI had good internal consistency (α = 0.70).

Data Analyses

We conducted longitudinal structural equation modeling analyses using the *lavaan* package in *R* (Rosseel, 2012). Depending on the hypothesis in question, MDD or GAD at T1 served as either a predictor or covariate. T2 global sleep quality was examined as a mediator. Either T3 MDD or T3 GAD severity served as outcomes. For a stringent test of our analyses, we adjusted for baseline disorder comorbidity in all our models. Many of the indicators of the latent constructs were non-normally distributed ordinal data. Thus, we conducted all analyses using maximum likelihood with robust standard errors estimator (MLR) which can accommodate non-normality (Rhemtulla, Brosseau-Liard, & Savalei, 2012; Wang & Cunningham, 2005).

Mediation analyses were conducted by carrying out a product-of-coefficients approach of the indirect effects ($a \times b$) for the regression coefficients of the predictor forecasting the mediator (*a* path), and the mediator predicting T3 MDD or GAD symptoms (*b* path). We presented the unstandardized regression coefficients and used bootstrapping with 10,000 resampling draws (Cheung & Lau, 2008). The mediation effect size is the ratio of the indirect effect (a*b) to the total effect, c = a*b + c', expressed in the percentage of variance that the mediator accounts for the predictor forecasting the outcome (Preacher & Kelley, 2011; Wen & Fan, 2015). Also, for each regression estimate, Cohen's *d* effect size was calculated using the formula $d = 2t/\sqrt{(df)}$

(Dunlap, Cortina, Vaslow, & Burke, 1996; Dunst, Hamby, & Trivette, 2004; Lakens, 2013). Thus, *d* values of 0.2, 0.5, and 0.8, represent small, moderate, and large effect sizes, respectively (Cohen, 1988). In total, the 1.9% missing data points were handled with full information maximum likelihood (Graham, 2009).

Tests of model's assumptions were conducted before analysis of each hypothesis. We assessed each model's empirical identification status by comparing the model's fully standardized solutions against conventionally acceptable loadings (Graham, 2005). The pattern of standardized factor loadings suggested that all measurement models were empirically identified. For large samples, χ^2 tends to be statistically significant (Brown, 2006), despite the candidate model differing from the true saturated model by trivial amounts. To assess each model's goodness-of-fit, we used practical goodness-of-fit indices and heuristic cut-offs (Kline, 2016b, 2016a): Tucker-Lewis Index (TLI; Tucker & Lewis, 1973; TLI $\geq$ .95), confirmatory fit index (CFI; Bentler, 1990; CFI $\geq$ .95), root mean square error of approximation (RMSEA; Steiger, 1990; RMSEA and its 90% confidence interval [CI] $\leq$ .50), and standardized root mean square residual (SRMR; Hu & Bentler, 1999; SRMR with 90% CI $\leq$ .080).

Chapter 3

Results

Hypothesis 1: T1 GAD → T3 MDD mediated by T2 Global Sleep Quality

First, we examined if the T1 GAD–T3 MDD relation would be mediated by T2 global sleep quality. Based on the practical fit indices, this mediation model showed good fit (χ^2 (*df* = 294) = 341.60, *p* = .029, CFI = .962, TLI = .958, RMSEA = .024, SRMR = .048). Supporting Hypothesis 1, higher T1 GAD symptom severity significantly predicted lower T2 global sleep quality (β = -0.332, *SE* = 0.073, *z* = -4.525, *p* < .001, *d* = -0.528), and less T2 global sleep quality was significantly related to higher T3 MDD severity (β = -0.142, *SE* = 0.040, *z* = -3.520, *p* < .001, *d* = -0.411). Simultaneously, the indirect effect (T1 GAD → T2 global sleep quality → T3 MDD) was significant (β = 0.047, *SE* = 0.017, *z* = 2.807, *p* = .005, *d* = 0.327). T2 global sleep quality accounted for 83.92% of the T1 GAD–T3 MDD association. Figure 1 in the presents the full SEM model of Hypothesis 1.

Hypothesis 2: T1 MDD → T3 GAD mediated by T2 Global Sleep Quality

Next, we examined if T2 global sleep quality would mediate the T1 MDD–T3 GAD relation. Based on the practical fit indices, this mediation model showed good fit (χ^2 (*df* = 270) = 310.94, *p* = .044, CFI = .941, TLI = .934, RMSEA = .036, SRMR = .068). Higher T1 MDD symptom severity was significantly associated with lower T2 global sleep quality (β = -1.067, *SE* = 0.314, *z* = -3.399, *p* < .001, *d* = -0.414), and reduced T2 global sleep quality was significantly linked to greater T3 MDD severity (β = -1.250, *SE* = 0.404, *z* = -3.097, *p* = .002, *d* = -0.377). Simultaneously, the indirect effect (T1 MDD → T2 global sleep quality → T3 GAD) was statistically significant (β = 1.334, *SE* = 0.469, *z* = 2.846, *p* = .004, *d* = 0.346). T2 Global Sleep

Quality fully mediated or accounted for 100% of the T1 MDD–T3 GAD relation. Therefore, Hypothesis 2 was supported (Figure 2).[1]

[1] For both Hypotheses 1 and 2, we determined that the same pattern of findings remained after removing the sleep disturbance symptom items from the MDD and GAD measures in the longitudinal SEM mediation analyses.

Chapter 4

Discussion

To our knowledge, this is the first study to test if poorer global subjective sleep quality mediated GAD predicting MDD, and vice versa. We found that poorer global sleep quality mediated the relation between T1 MDD and T3 GAD (controlling for T1 GAD), as well as the association between T1 GAD and T3 MDD (adjusting for T1 MDD). Specifically, T2 global sleep quality accounted for 83.93% of the T1 GAD–T3 MDD association, and 100% of the T1 MDD–T3 GAD relation. Collectively, our results thus lend support to the *transdiagnostic theory of sleep* (Harvey, 2008). We provide potential explanations for this pattern of findings.

Why did T1 MDD and GAD predict T2 global subjective sleep quality? One plausible explanation is that sleep issues and MDD and GAD have similar underlying risk factors, such as repetitive negative thinking patterns. Consistent with Riemann's *hyperarousal model*, over time, insomnia could exacerbate a cycle of pre-sleep negative perseverative cognitions, some of which are often implicated in depression and anxiety disorder-prone individuals (e.g., undue worry, rumination, limiting core beliefs). It is possible that repetitive negative thinking patterns hinder the ability to achieve good quality sleep across time (Jansson & Linton, 2007; Mazzer, Boersma, & Linton, 2019; Norell-Clarke, Jansson-Fröjmark, Tillfors, Harvey, & Linton, 2014; Riemann et al., 2010).

Findings that greater MDD and GAD symptom severity predicted lower global sleep quality may also be partly explained by *scar theories*. These theories propose that higher MDD and GAD symptoms may create a "scarring effect" over time by predisposing individuals to poorer sleep quality through various deficient health behaviors, such as unhealthy diet and excessive technology use. Plausibly, those exhibiting elevated anxiety and depressive symptoms

predispose themselves to future sleep issues by making poor diet choices. There is a growing body of evidence for links between more unhealthy dietary choices and increased anxiety and depressive symptoms in the long term (Daneshzad, Keshavarz, Qorbani, Larijani, & Azadbakht, 2020; Tan et al., 2016). Relatedly, prolonged smartphone overuse amongst anxiety and depression-prone individuals may influence the link between T1 MDD and GAD and T2 subjective sleep quality by affecting sleep architecture, altering melatonin levels via excessive exposure to bright lights during bedtime, and inducing cognitive, emotional, and physiological arousal (Dinis & Bragança, 2018). Clearly, these conjectures warrant testing by future studies.

Additionally, why did T2 global subjective sleep quality predict T3 MDD and GAD? It is possible that suboptimal stress coping strategies play a role in mediating the association between poor sleep quality and MDD or GAD 9 years later. Prolonged stress reactivity has been linked to sleep problems, possibly due to fatigue and other physiological markers associated with high stress reactivity (e.g., higher resting heart rate and blood pressure) (Herr et al., 2018). As a result, sleep issues may, over time, increase vulnerability to stress, which has already been identified as a significant risk factor for anxiety and depressive disorder-prone individuals (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Zhang, Yan, Shum, & Deng, 2020). Our findings also extend a recent study that showed poor sleep quality was associated with larger rise in somatic symptoms of depression (e.g., fatigue) among college students (and vice versa) (Shim, Noh, Yoon, Mun, & Hahm, 2019). This pattern may be attributed to daytime dysfunction reducing serotonergic and dopaminergic transmission critical for regulating sleep-wake cycle biological rhythms, and the adverse effects of poor sleep on cognitive functions (Harvey, Murray, Chandler, & Soehner, 2011; Waller et al., 2016). Future research on sleep deprived or sleep behavior-disordered individuals could provide insight on the role of dysregulated dopamine

on long-term anxiety and depressive symptoms. Specifically, the impacts on reward brain circuitry (e.g., nucleus accumbens, orbitofrontal cortex, striatum) may have direct effects on reward processing such that sleep-disturbed individuals demonstrate depression-like behaviors (i.e., helplessness, anhedonia, inability to concentrate) (Auerbach, Admon, & Pizzagalli, 2014; Blake, Trinder, & Allen, 2018; Martin-Soelch, 2009). These hypotheses merit empirical testing.

Overall, our findings that T2 global sleep quality mediates the prospective 18-year MDD-GAD bi-directional relation is consistent with some prior longitudinal research. For instance, at least four studies have shown that sleep duration, disturbance, and associated insomnia markers were reciprocally related to depression and anxiety disorder severity in diverse adolescent, pregnant women, and community-dwelling samples (Geng et al., 2018; Goldstone et al., 2020; Liu et al., 2020; van der Zwan et al., 2017). The current paper builds upon these studies by examining suboptimal sleep as a *mechanism* of the longitudinal MDD-GAD comorbidity.

Our study presents with a number of limitations. First, we did not make use of any objective measures of sleep quality (e.g., electroencephalogram, polysomnography). Future studies should consider utilizing measures such as the multiple sleep latency test, to better capture the psychobiological sleep profiles (Brand et al., 2014; Plante, Finn, Hagen, Mignot, & Peppard, 2017; Zhu et al., 2020). Also, given the use of DSM-III-R-defined CIDI-SF interviews, future research can determine if a similar pattern of findings is replicated with the use of DSM-5 measures. In addition, to increase generalizability, it would be optimal for upcoming studies to recruit diverse samples in terms of socio-economic status and ethnicity, given the sample homogeneity in this study. Nonetheless, our study has a number of strengths. For instance, the longitudinal nature of our study provides more insight into the temporal relation between sleep issues and two common psychiatric disorders. Additionally, the data set included a wide age

range of participants. As sleep issues influence people throughout the lifespan, our research sufficiently captures how sleep difficulties may precede or predict psychological disorders across various developmental stages.

If our pattern of findings is replicated by future studies, some clinical implications merit consideration. For instance, therapies which address sleep issues as well as psychiatric disorders, such as cognitive behavioral therapy for insomnia (CBTI), appear to have promising outcomes for improving symptoms. Examples include the effectiveness of CBTI to change maladaptive thinking and somatic hyperarousal, which are characteristic of both insomnia, depression, and anxiety disorders (Kalmbach et al., 2019). Another type of therapy that employs this tactic of addressing both sleep and psychiatric issues is mindfulness-based cognitive therapy (MBCT). Research has consistently shown that MBCT is effective at significantly reducing anxiety and depressive symptoms alongside improving subjective sleep quality (Britton, Haynes, Fridel, & Bootzin, 2010). The mechanisms of treatment have included modifications in pre-sleep arousal, such as decrements in unhelpful avoidance behaviors and classically conditioned anxious arousal or pathological worry with the bed (Blake et al., 2017). Further, several preliminary studies have demonstrated that music therapy may elicit psychophysiological responses to musical pitch and rhythm, resulting in reductions in depressive symptoms alongside sleep issues (Chan, Chan, Mok, & Kwan Tse, 2009; Chan, Chan, & Mok, 2010; Wang, Sun, & Zang, 2014).

In closing, our findings further refine the longitudinal between-subject relationship between MDD and GAD across 18 years. Global subjective sleep quality at T2 mediated the T1 MDD–T3 GAD relation, and vice versa. Future studies should consider utilizing objective measures of sleep quality and DSM-5 measures as well as recruiting culturally diverse samples.

Appendix A

Figures

Figure 1 T1 GAD-T3 MDD relation mediated by T2 Global Sleep Quality

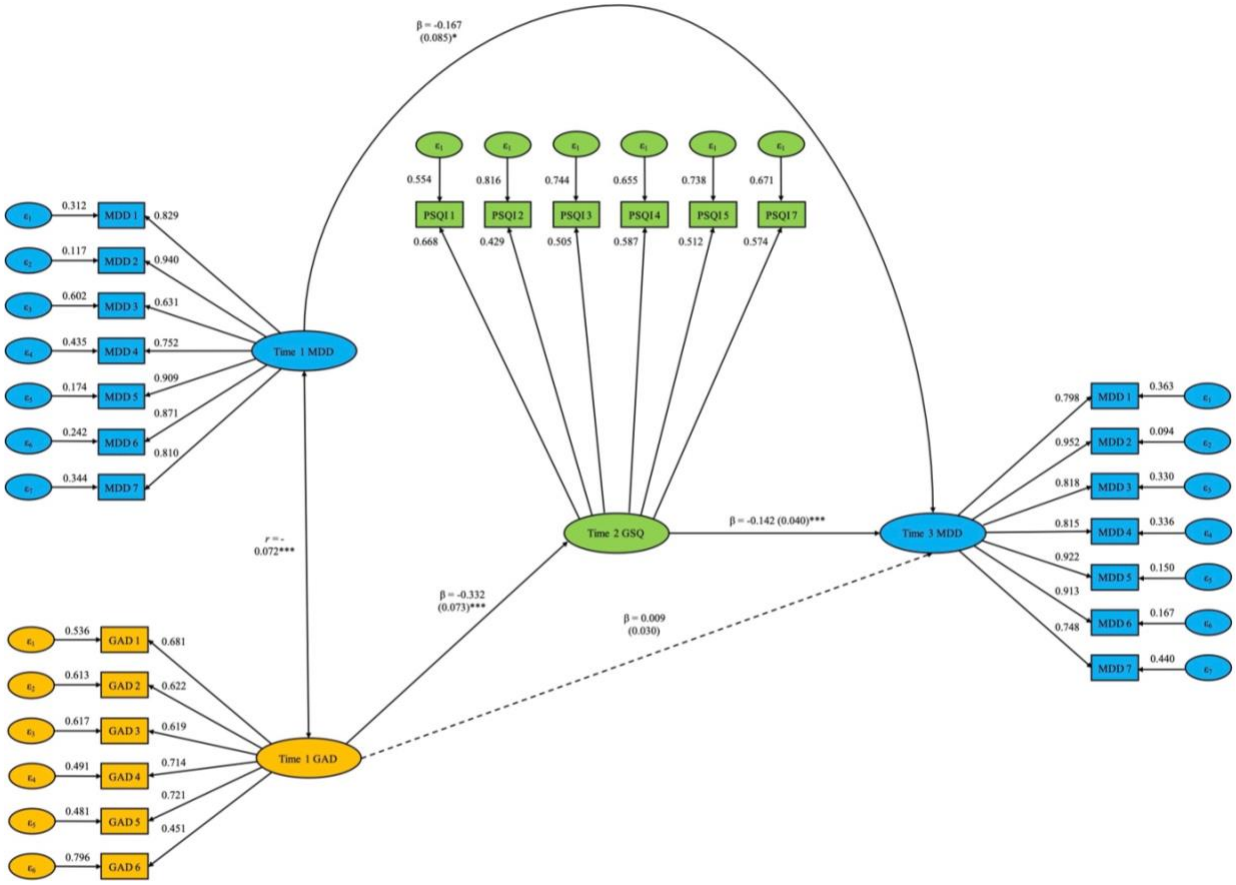
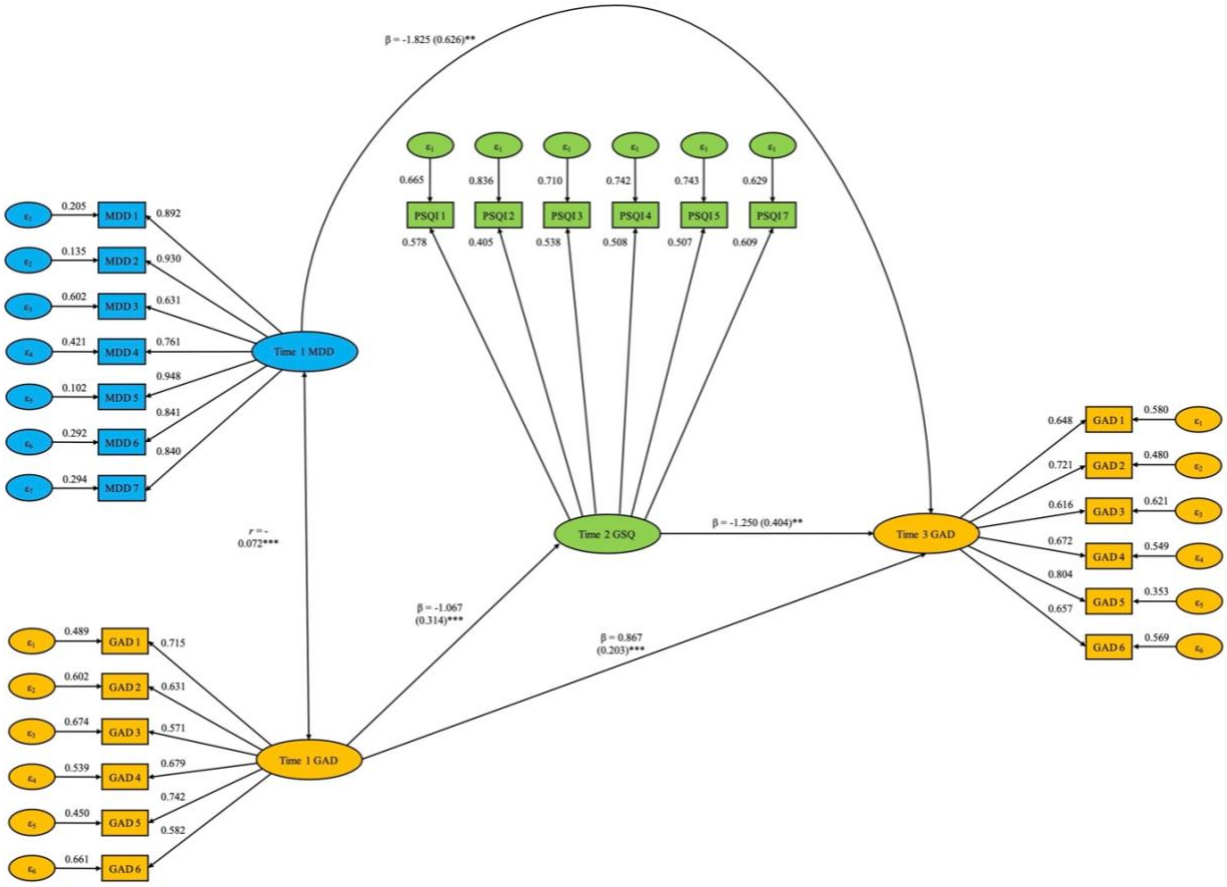


Figure 2 T1 MDD-T3 GAD relation mediated by T2 Global Sleep Quality



Appendix B

Tables

Table 1 Correlation Matrix of Study Variables

	1	2	3	4	5	6	7
1. Age	–						
2. Gender	-.002	–					
3. T1 MDD	-.092***	.075***	–				
4. T1 GAD	-.044*	.075***	.288***	–			
5. T3 MDD	-.088***	.083***	.279***	.182***	–		
6. T3 GAD	-.086***	.066***	.159***	.354***	.340***	–	
7. T2 GSQ	-.041	.144***	.224***	.179***	.291***	.181***	–
M or %	45.62	54.61	0.69	0.14	0.6	0.13	6.09
SD	11.41	–	1.82	0.86	1.71	0.92	3.58
Min	20.00	–	0.00	0.00	0.00	0.00	0.00
Max	74.00	–	7.00	10.00	7.00	10.00	19.00
Skewness	0.24	–	2.44	7.47	2.69	7.89	0.93
Kurtosis	-0.70	–	4.35	60.84	5.74	65.66	0.57

Note. * $p < .05$; ** $p < .01$; *** $p < .001$.
GAD = generalized anxiety disorder; GSQ = global sleep quality; MDD = major depressive disorder; *M* = mean; Max = maximum; Min = minimum; *SD* = standard deviation; T1 = time 1; T2 = time 2; T3 = time 3.

References

Abel, J. L., & Borkovec, T. D. (1995). Generalizability of DSM-III-R generalized anxiety disorders to proposed DSM-IV criteria and cross-validation of proposed changes. *Journal of Anxiety Disorders, 9*, 303-315. doi:10.1016/0887-6185(95)00011-C

Auerbach, R. P., Admon, R., & Pizzagalli, D. A. (2014). Adolescent depression: Stress and reward dysfunction. *Harvard Review of Psychiatry, 22*, 139-148. doi:10.1097/HRP.0000000000000034

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107,* 238-246. doi:10.1037/0033-2909.107.2.238

Blake, M., Schwartz, O., Waloszek, J. M., Raniti, M., Simmons, J. G., Murray, G., . . . Allen, N. B. (2017). The SENSE study: Treatment mechanisms of a cognitive behavioral and mindfulness-based group sleep improvement intervention for at-risk adolescents. *Sleep, 40*. doi:10.1093/sleep/zsx061

Blake, M. J., Trinder, J. A., & Allen, N. B. (2018). Mechanisms underlying the association between insomnia, anxiety, and depression in adolescence: Implications for behavioral sleep interventions. *Clinical Psychology Review, 63*, 25-40. doi:10.1016/j.cpr.2018.05.006

Brand, S., Kalak, N., Gerber, M., Kirov, R., Pühse, U., & Holsboer-Trachsler, E. (2014). High self-perceived exercise exertion before bedtime is associated with greater objectively assessed sleep efficiency. *Sleep Medicine, 15*, 1031-1036. doi:10.1016/j.sleep.2014.05.016

Brim, O. G., Baltes, P. B., Bumpass, L. L., Cleary, P. D., Featherman, D. L., Hazzard, W. R., . . . Shweder, R. A. (2019). Midlife in the United States (MIDUS 1), 1995-1996: Inter-university Consortium for Political and Social Research [distributor].

Britton, W. B., Haynes, P. L., Fridel, K. W., & Bootzin, R. R. (2010). Polysomnographic and subjective profiles of sleep continuity before and after mindfulness-based cognitive therapy in partially remitted depression. *Psychosomatic Medicine, 72*, 539-548. doi:10.1097/PSY.0b013e3181dc1bad

Brown, T. A. (2006). *Confirmatory factor analysis for applied research*. New York: The Guilford Press.

Butnoriene, J., Bunevicius, A., Saudargiene, A., Nemeroff, C. B., Norkus, A., Ciceniene, V., & Bunevicius, R. (2015). Metabolic syndrome, major depression, generalized anxiety disorder, and ten-year all-cause and cardiovascular mortality in middle aged and elderly patients. *International Journal of Cardiology, 190*, 360-366. doi:10.1016/j.ijcard.2015.04.122

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193-213. doi:10.1016/0165-1781(89)90047-4

Buysse, D. J., Angst, J., Gamma, A., Ajdacic, V., Eich, D., & Rössler, W. (2008). Prevalence, course, and comorbidity of insomnia and depression in young adults. *Sleep, 31*, 473-480. doi:10.1093/sleep/31.4.473

Chan, M. F., Chan, E. A., Mok, E., & Kwan Tse, F. Y. (2009). Effect of music on depression levels and physiological responses in community-based older adults. *Int J Ment Health Nurs, 18*, 285-294. doi:10.1111/j.1447-0349.2009.00614.x

Chan, M. F., Chan, E. A., & Mok, E. (2010). Effects of music on depression and sleep quality in elderly people: A randomised controlled trial. *Complement Ther Med, 18*, 150-159. doi:10.1016/j.ctim.2010.02.004

Cheung, G. W., & Lau, R. S. (2008). Testing mediation and suppression effects of latent variables: Bootstrapping with structural equation models. *Organizational Research Methods, 11*, 296-325. doi:10.1177/1094428107300343

Cho, H. J., Seeman, T. E., Kiefe, C. I., Lauderdale, D. S., & Irwin, M. R. (2015). Sleep disturbance and longitudinal risk of inflammation: Moderating influences of social integration and social isolation in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Brain, Behavior, and Immunity, 46*, 319-326. doi:10.1016/j.bbi.2015.02.023

Cohen, J. (1988). *Statistical power for the social sciences*. Hillsdale, NJ: Laurence Erlbaum and Associates.

Daneshzad, E., Keshavarz, S.-A., Qorbani, M., Larijani, B., & Azadbakht, L. (2020). Association between a low-carbohydrate diet and sleep status, depression, anxiety, and stress score. *Journal of the Science of Food and Agriculture, 100*, 2946-2952. doi:10.1002/jsfa.10322

Dinis, J., & Bragança, M. (2018). Quality of sleep and depression in college students: A systematic review. *Sleep Science, 11*, 290-301. doi:10.5935/1984-0063.20180045

Dunlap, W. P., Cortina, J. M., Vaslow, J. B., & Burke, M. J. (1996). Meta-analysis of experiments with matched groups or repeated measures designs. *Psychological Methods, 1*, 170-177. doi:10.1037/1082-989x.1.2.170

Dunst, C. J., Hamby, D. W., & Trivette, C. M. (2004). Guidelines for calculating effect sizes for practice-based research syntheses. *Centerscope, 3*, 1-10.

Geng, F., Liu, X., Liang, Y., Shi, X., Chen, S., & Fan, F. (2018). Prospective associations between sleep problems and subtypes of anxiety symptoms among disaster-exposed adolescents. *Sleep Medicine, 50,* 7-13. doi:10.1016/j.sleep.2018.05.017

Goldstone, A., Javitz, H. S., Claudatos, S. A., Buysse, D. J., Hasler, B. P., de Zambotti, M., . . . Baker, F. C. (2020). Sleep disturbance predicts depression symptoms in early adolescence: Initial findings from the adolescent brain cognitive development study. *Journal of Adolescent Health, 66*, 567-574. doi:10.1016/j.jadohealth.2019.12.005

Graham, J. W. (2005). *Structural Equation Modeling*. Unpublished Manuscript, The Pennsylvania State University.

Graham, J. W. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology, 60,* 549–576. doi:10.1146/annurev.psych.58.110405.085530

Gregory, A. M., Caspi, A., Eley, T. C., Moffitt, T. E., Oconnor, T. G., & Poulton, R. (2005). Prospective longitudinal associations between persistent sleep problems in childhood and anxiety and depression disorders in adulthood. *Journal of Abnormal Child Psychology, 33*, 157-163. doi:10.1007/s10802-005-1824-0

Harvey, A. G. (2008). Insomnia, psychiatric disorders, and the transdiagnostic perspective. *Current Directions in Psychological Science, 17*, 299-303. doi:doi.org/10.1111/j.1467-8721.2008.00594.x

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review, 31,* 225-235. doi:10.1016/j.cpr.2010.04.003

Herr, R. M., Barrech, A., Riedel, N., Gündel, H., Angerer, P., & Li, J. (2018). Long-term effectiveness of stress management at work: Effects of the changes in perceived stress

reactivity on mental health and sleep problems seven years later. *International Journal of Environmental Research and Public Health, 15*. doi:10.3390/ijerph15020255

Hsiao, F. H., Chang, K. J., Kuo, W. H., Huang, C. S., Liu, Y. F., Lai, Y. M., . . . Chan, C. L. (2013). A longitudinal study of cortisol responses, sleep problems, and psychological well-being as the predictors of changes in depressive symptoms among breast cancer survivors. *Psychoneuroendocrinology, 38*, 356-366. doi:10.1016/j.psyneuen.2012.06.010

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*, 1-55. doi:10.1080/10705519909540118

Jackson, M. L., Sztendur, E. M., Diamond, N. T., Byles, J. E., & Bruck, D. (2014). Sleep difficulties and the development of depression and anxiety: A longitudinal study of young Australian women. *Archives of Women's Mental Health, 17*, 189-198. doi:10.1007/s00737-014-0417-8

Jacobson, N. C., & Newman, M. G. (2017). Anxiety and depression as bidirectional risk factors for one another: A meta-analysis of longitudinal studies. *Psychological Bulletin, 143*, 1155-1200. doi:10.1037/bul0000111

Jansson, M., & Linton, S. J. (2007). Psychological mechanisms in the maintenance of insomnia: Arousal, distress, and sleep-related beliefs. *Behaviour Research and Therapy, 45*, 511-521. doi:10.1016/j.brat.2006.04.003

Jansson-Fröjmark, M., & Lindblom, K. (2008). A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population. *Journal of Psychosomatic Research, 64*, 443-449. doi:10.1016/j.jpsychores.2007.10.016

Kalmbach, D. A., Cheng, P., Arnedt, J. T., Anderson, J. R., Roth, T., Fellman-Couture, C., . . . Drake, C. L. (2019). 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 medicine, 55*, 124-134. doi:10.1016/j.sleep.2018.11.019

Kessler, R. C., Wittchen, H.-U., Abelson, J. M., Mcgonagle, K., Schwarz, N., Kendler, K. S., . . . Zhao, S. (1998). Methodological studies of the Composite International Diagnostic Interview (CIDI) in the US national comorbidity survey (NCS). *International Journal of Methods in Psychiatric Research, 7*, 33-55. doi:10.1002/mpr.33

Kessler, R. C., Gruber, M., Hettema, J. M., Hwang, I., Sampson, N., & Yonkers, K. A. (2008a). Co-morbid major depression and generalized anxiety disorders in the National Comorbidity Survey Follow-Up. *Psychological Medicine, 38*, 365-374. doi:10.1017/S0033291707002012

Kessler, R. C., Heeringa, S., Lakoma, M. D., Petukhova, M., Rupp, A. E., Schoenbaum, M., . . . Zaslavsky, A. M. (2008b). Individual and societal effects of mental disorders on earnings in the United States: Results from the national comorbidity survey replication. *American Journal of Psychiatry, 165*, 703-711. doi:10.1176/appi.ajp.2008.08010126

Kim, J.-M., Stewart, R., Kim, S.-W., Yang, S.-J., Shin, I., & Yoon, J.-S. (2009). Insomnia, depression, and physical disorders in late life: A 2-year longitudinal community study in Koreans. *Sleep, 32*, 1221-1228. doi:10.1093/sleep/32.9.1221

Kline, R. B. (2016b). Chapter 12: Global fit testing *Principles and practice of structural equation modeling* (pp. 262–299). New York: The Guilford Press.

Kline, R. B. (2016a). Chapter 11: Estimation and local fit testing. In R. B. Kline (Ed.), *Principles and practice of structural equation modeling* (pp. 231–261). New York: The Guilford Press.

Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and ANOVAs. *Frontiers in Psychology, 4*, 863. doi:10.3389/fpsyg.2013.00863

Liu, B.-P., Wang, X.-T., Liu, Z.-Z., Wang, Z.-Y., An, D., Wei, Y.-X., . . . Liu, X. (2020). Depressive symptoms are associated with short and long sleep duration: A longitudinal study of Chinese adolescents. *Journal of Affective Disorders, 263*, 267-273. doi:10.1016/j.jad.2019.11.113

Martin-Soelch, C. (2009). Is depression associated with dysfunction of the central reward system? *Biochemical Society Transactions, 37*, 313-317. doi:10.1042/bst0370313

Mazzer, K., Boersma, K., & Linton, S. J. (2019). A longitudinal view of rumination, poor sleep and psychological distress in adolescents. *Journal of Affective Disorders, 245*, 686-696. doi:10.1016/j.jad.2018.11.053

Michl, L. C., McLaughlin, K. A., Shepherd, K., & Nolen-Hoeksema, S. (2013). Rumination as a mechanism linking stressful life events to symptoms of depression and anxiety: Longitudinal evidence in early adolescents and adults. *Journal of Abnormal Psychology, 122*, 339-352. doi:10.1037/a0031994

Morphy, H., Dunn, K. M., Lewis, M., Boardman, H. F., & Croft, P. R. (2007). Epidemiology of Insomnia: A Longitudinal Study in a UK Population. *Sleep, 30*, 274-280. doi:10.1093/sleep/30.3.274

Norell-Clarke, A., Jansson-Fröjmark, M., Tillfors, M., Harvey, A. G., & Linton, S. J. (2014). Cognitive processes and their association with persistence and remission of insomnia: Findings from a longitudinal study in the general population. *Behaviour Research and Therapy, 54,* 38-48. doi:10.1016/j.brat.2014.01.002

O'Connor, T. G., Tang, W., Gilchrist, M. A., Moynihan, J. A., Pressman, E. K., & Blackmore, E. R. (2014). Diurnal cortisol patterns and psychiatric symptoms in pregnancy: Short-term longitudinal study. *Biological Psychology, 96,* 35-41. doi:10.1016/j.biopsycho.2013.11.002

Plante, D. T., Finn, L. A., Hagen, E. W., Mignot, E., & Peppard, P. E. (2017). Longitudinal associations of hypersomnolence and depression in the Wisconsin Sleep Cohort Study. *Journal of Affective Disorders, 207,* 197-202. doi:10.1016/j.jad.2016.08.039

Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods, 16,* 93-115. doi:10.1037/a0022658

Regier, D. A., Rae, D. S., Narrow, W. E., Kaelber, C. T., & Schatzberg, A. F. (1998). Prevalence of anxiety disorders and their comorbidity with mood and addictive disorders. *British Journal of Psychiatry, 173*, 24-28. doi:10.1192/S0007125000293483

Rhemtulla, M., Brosseau-Liard, P. É., & Savalei, V. (2012). When can categorical variables be treated as continuous? A comparison of robust continuous and categorical SEM estimation methods under suboptimal conditions. *Psychological Methods, 17*, 354-373. doi:10.1037/a0029315

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews, 14*, 19-31. doi:10.1016/j.smrv.2009.04.002

Rosellini, A. J., & Brown, T. A. (2011). The NEO Five-Factor Inventory: Latent structure and relationships with dimensions of anxiety and depressive disorders in a large clinical sample. *Assessment, 18*, 27-38. doi:10.1177/1073191110382848

Rosseel, Y. (2012). Lavaan: An R package for structural equation modeling. *Journal of Statistical Software, 48*, 1–36. doi:10.18637/jss.v048.i02

Rutten, S., Vriend, C., van der Werf, Y. D., Berendse, H. W., Weintraub, D., & van den Heuvel, O. A. (2017). The bidirectional longitudinal relationship between insomnia, depression and anxiety in patients with early-stage, medication-naïve Parkinson's disease. *Parkinsonism & related disorders, 39*, 31-36. doi:10.1016/j.parkreldis.2017.01.015

Ryff, C., Almeida, D. M., Ayanian, J., Carr, D. S., Cleary, P. D., Coe, C., . . . Williams, D. (2017). Midlife in the United States (MIDUS 2), 2004-2006: Inter-university Consortium for Political and Social Research [distributor].

Ryff, C., Almeida, D., Ayanian, J., Binkley, N., Carr, D. S., Coe, C., . . . Williams, D. (2019). Midlife in the United States (MIDUS 3), 2013-2014: Inter-university Consortium for Political and Social Research [distributor].

Shim, E.-J., Noh, H.-l., Yoon, J., Mun, H. s., & Hahm, B.-J. (2019). A longitudinal analysis of the relationships among daytime dysfunction, fatigue, and depression in college students. *Journal of American College Health, 67*, 51-58. doi:10.1080/07448481.2018.1462819

Sivertsen, B., Salo, P., Mykletun, A., Hysing, M., Pallesen, S., Krokstad, S., . . . Øverland, S. (2012). The bidirectional association between depression and insomnia: The HUNT study. *Psychosom Med, 74*, 758-765. doi:10.1097/PSY.0b013e3182648619

Stahl, S. T., Smagula, S. F., Rodakowski, J., Dew, M. A., Karp, J. F., Albert, S. M., . . . Reynolds, C. F. (2020). Subjective Sleep Quality and Trajectories of Interleukin-6 in Older Adults. *The American Journal of Geriatric Psychiatry*. doi:10.1016/j.jagp.2020.06.019

Steel, Z., Marnane, C., Iranpour, C., Chey, T., Jackson, J. W., Patel, V., & Silove, D. (2014). The global prevalence of common mental disorders: A systematic review and meta-analysis 1980–2013. *International Journal of Epidemiology, 43*, 476-493. doi:10.1093/ije/dyu038

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25*, 173-180. doi:10.1207/s15327906mbr2502_4

Sun, Y., Shi, L., Bao, Y., Sun, Y., Shi, J., & Lu, L. (2018). The bidirectional relationship between sleep duration and depression in community-dwelling middle-aged and elderly individuals: evidence from a longitudinal study. *Sleep Medicine, 52*, 221-229. doi:10.1016/j.sleep.2018.03.011

Swendsen, J. D. (1997). Anxiety, depression, and their comorbidity: An experience sampling test of the helplessness-hopelessness theory. *Cognitive Therapy and Research, 21*, 97-114. doi:Doi 10.1023/A:1021872410824

Tan, X., Alén, M., Wang, K., Tenhunen, J., Wiklund, P., Partinen, M., & Cheng, S. (2016). Effect of six-month diet intervention on sleep among overweight and obese men with

chronic insomnia symptoms: A randomized controlled trial. *Nutrients, 8*, 751. doi:10.3390/nu8110751

Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika, 38*, 1-10. doi:10.1007/BF02291170

Uhmann, S., Beesdo-Baum, K., Becker, E. S., & Hoyer, J. (2010). Specificity of interpersonal problems in generalized anxiety disorder versus other anxiety disorders and depression. *Journal of Nervous and Mental Disease, 198*, 846-851. doi:10.1097/NMD.0b013e3181f98063

van der Zwan, J. E., de Vente, W., Tolvanen, M., Karlsson, H., Buil, J. M., Koot, H. M., . . . Karlsson, L. (2017). Longitudinal associations between sleep and anxiety during pregnancy, and the moderating effect of resilience, using parallel process latent growth curve models. *Sleep Medicine, 40*, 63-68. doi:10.1016/j.sleep.2017.08.023

Waller, K. L., Mortensen, E. L., Avlund, K., Osler, M., Fagerlund, B., Lauritzen, M., & Jennum, P. (2016). Subjective sleep quality and daytime sleepiness in late midlife and their association with age-related changes in cognition. *Sleep Medicine, 17*, 165-173. doi:10.1016/j.sleep.2015.01.004

Wang, C. F., Sun, Y. L., & Zang, H. X. (2014). Music therapy improves sleep quality in acute and chronic sleep disorders: A meta-analysis of 10 randomized studies. *Int J Nurs Stud, 51*, 51-62. doi:10.1016/j.ijnurstu.2013.03.008

Wang, W. C., & Cunningham, E. G. (2005). Comparison of alternative estimation methods in confirmatory factor analyses of the general health questionnaire. *Psychological Reports, 97*, 3-10. doi:10.2466/pr0.97.1.3-10

Wen, Z., & Fan, X. (2015). Monotonicity of effect sizes: Questioning kappa-squared as mediation effect size measure. *Psychological Methods, 20*, 193-203. doi:10.1037/met0000029

Wittchen, H.-U. (1994). Reliability and validity studies of the WHO-Composite International Diagnostic Interview (CIDI): A critical review. *Journal of Psychiatric Research, 28*, 57-84. doi:10.1016/0022-3956(94)90036-1

Yu, Y., Li, M., Pu, L., Wang, S., Wu, J., Ruan, L., . . . Jiang, W. (2017). Sleep was associated with depression and anxiety status during pregnancy: A prospective longitudinal study. *Archives of Women's Mental Health, 20*, 695-701. doi:10.1007/s00737-017-0754-5

Zainal, N. H., & Newman, M. G. (in press-a). Within-person increase in pathological worry predicts future depletion of unique executive functioning domains. *Psychological Medicine*. doi:10.1017/S0033291720000422

Zainal, N. H., & Newman, M. G. (in press-b). Larger increase in trait negative affect is associated with greater future cognitive decline and vice versa across 23 years. *Depression and Anxiety*. doi:10.1002/da.23093

Zhang, W.-j., Yan, C., Shum, D., & Deng, C.-p. (2020). Responses to academic stress mediate the association between sleep difficulties and depressive/anxiety symptoms in Chinese adolescents. *Journal of Affective Disorders, 263*, 89-98. doi:10.1016/j.jad.2019.11.157

Zhu, D.-m., Zhang, C., Yang, Y., Zhang, Y., Zhao, W., Zhang, B., . . . Yu, Y. (2020). The relationship between sleep efficiency and clinical symptoms is mediated by brain function in major depressive disorder. *Journal of Affective Disorders, 266*, 327-337. doi:10.1016/j.jad.2020.01.155

Zou, P., Wang, X., Sun, L., Liu, K., Hou, G., Yang, W., . . . Chen, Q. (2020). Poorer sleep quality correlated with mental health problems in college students: A longitudinal observational study among 686 males. *Journal of Psychosomatic Research, 136*, 110177. doi:10.1016/j.jpsychores.2020.110177

ACADEMIC VITA

EDUCATION

The Pennsylvania State University **University Park, PA**
Schreyer Honors College *Class of 2021*
B.S. Psychology, Neuroscience Option
Honors Thesis: *Why sleep is key: Poor sleep quality is a mechanism for the bidirectional relationship between Major Depressive Disorder and Generalized Anxiety Disorder across 18 years*

RELATED EXPERIENCE

Penn State University, College of the Liberal Arts **University Park, PA**
Michelle Newman, PhD, and Nur Hani Zainal, MS *Aug 2019 – Dec 2020*
Research Assistant

- Collected data for a randomized controlled trial of a mindfulness-based treatment administered via smartphone app for persons with generalized anxiety disorder.
- Conducted a battery of neuropsychological tests on participants, including the Bell-Lysaker Emotion Recognition Task, Delis Kaplan Executive Function System (D-KEFS) Color-Word Interference Test, Trail Making Test, and Verbal Fluency Test, and Wechsler Adult Intelligence Scale–Fourth Edition (WAIS-IV) Digit Span and Letter Span subtests.
- Conducted the Mini International Neuropsychiatric Interview (MINI) (aligned with the Diagnostic and Statistical Manual–Fifth Edition; DSM-5) on participants and determining diagnoses of affective, anxiety, and psychotic disorders.
- Organized multiple datasets via Microsoft Excel as part of the data management process.
- Independently wrote a research thesis testing coping theories to explain the longitudinal associations between major depressive disorder and generalized anxiety disorder.
- Participated in weekly journal clubs pertaining to treatments for depression and anxiety disorders.

Penn State University, Eberly College of Science **University Park, PA**
Katherine Masters, PhD *Aug 2019 – Present*
Learning Assistant, CHEM 202 (Fundamentals of Organic Chemistry) & CHEM 213M (Laboratory in Organic Chemistry - Honors, Writing Intensive)

- Assisted students in the CHEM 202 classroom during graded group problems
- Facilitated learning organic chemistry laboratory techniques and writing skills in and outside the CHEM 213M laboratory
- Hosted weekly, two-hour office hours for students to ask questions regarding course material
- Corresponded with faculty and teaching assistants to create an optimal learning experience for students

Penn State University, College of the Liberal Arts **University Park, PA**
Michael Hallquist, PhD *Nov 2018 – May 2019*
Research Assistant

- Administered neurocognitive tasks to human subjects to investigate personality development in adolescents
- Quality checked fMRI data to ensure that images were co-registered with each other
- Created flyers and brochures to be used in recruiting research participants
- Wrote R code for scoring of various NIH Toolbox® neuro-behavioral measurements

LEADERSHIP

ServeState **University Park, PA**

President *Aug 2020 – Present*

- Headed weekly executive board meetings to plan intra- and extra-organization events, such as American Red Cross Blood Drives, food drives, movie nights, and merchandise orders
- Managed officer and general body membership to maintain recognition as a registered student organization

Fellowship Chair *Aug 2019 – Dec 2019*

- Cultivated positive relationships by planning intra-organization events, such as tie-dying, lunch dates, and board game nights
- Regulated a points system for tracking members' involvement in fellowship events

Treasurer *Jan 2019 – May 2019*

- Created a budget for executive board members and the organization to adhere to for the semester
- Collaborated with Associated Student Activities to request checks and purchase orders, deposit money, and transfer funds to other student organizations

Fundraising Chair *May 2018 – Jan 2019*

- Organized volunteer cleanup of Beaver Stadium the morning after home football games
- Partnered with Tailgate Ambassadors to aid in the execution of recycling efforts during football tailgates
- Coordinated fundraising partnerships with local businesses, such as Chipotle and Kiwi Yogurt

Scholar Ambassador Team **University Park, PA**

Member *Aug 2018 – Present*

- Informed prospective students about the values and history of the Schreyer Honors College through student panels and tours
- Helped to organize Founders Day, a celebration for current scholars to learn about the founding of the Schreyer Honors College
- Assisted in coordinating and checking in guests to various events hosted by the Schreyer Honors College, such as the Homecoming Day Parade Watch, New Student Orientation, and Accepted Students Programs
- Served as a point of contact for prospective students of the Schreyer Honors College