THE ROLE OF NEUROTICISM IN DAILY EXPERIENCES, AFFECT, AND NIGHTLY SLEEP QUALITY

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Abstract

Neuroticism is one of the personality characteristics most strongly implicated in impaired health outcomes, yet the pathways by which this impairment occurs in daily life are not well understood. The differential exposure-reactivity model provides support for the role of neuroticism in increased reactivity to daily stress, and the cognitive-hyperarousal model of insomnia provides support for the role of neuroticism in impaired sleep. However, these models have not been integrated, nor tested longitudinally in naturalistic settings with comprehensive measures of both daily experiences and sleep.

To address these gaps, the current research investigated the role of neuroticism in the associations between daily experiences and sleep quality using a 14-day daily diary design. The study included the investigation of three aims: 1) examining the role of neuroticism in predicting exposure to daily positive and negative experiences (DPEs and DNEs, respectively); 2) examining the moderating role of neuroticism in the within-person associations between exposure to DPEs and DNEs and daily positive and negative affect (PA and NA, respectively); and 3) examining the mediating role of average daily PA and NA in the association between neuroticism and subjective and objective sleep quality. Participants (N = 60 young adults; M_age = 25.33 ± 3.98 years; 53% female) completed a baseline questionnaire and a 14-day daily diary protocol, during which they completed two surveys per day and wore an actigraphy device for determination of objective sleep quality.

Results revealed that neuroticism was not associated with a higher frequency of DNEs, but it was associated with a lower frequency of DPEs. Compared to those lower in neuroticism, for those higher in neuroticism, days with more DNEs than usual were associated with greater differences in NA, and days with more DPEs than usual were associated with greater differences in PA and NA. Neuroticism was associated with greater average daily NA, lower average daily PA, and worse subjective but not objective sleep quality. Average daily NA, but not PA, mediated the associations between neuroticism and subjective as well as some objective measures of sleep quality.

These findings underscore the importance of assessing both DPEs and DNEs, as well as the contention that those higher in neuroticism are more labile in their positive and negative affect in daily life. Daily negative affect may be an important intervening pathway between neuroticism and impaired sleep quality. Findings help identify those individuals at greater risk for affective lability and sleep-related disturbance and help better characterize the daily psychological processes by which neuroticism may contribute to impaired health.
# Table of Contents

List of Tables .................................................................................................................. v  
List of Figures ..................................................................................................................... vi  
Acknowledgements ........................................................................................................... vii  

## CHAPTER 1: Introduction .............................................................................................. 1  
Defining Neuroticism and Its Biological Correlates ......................................................... 1  
Types of Relationships between Neuroticism and Health ............................................. 2  
Overview of the Aims of the Current Study ................................................................. 4  

## CHAPTER 2: Literature Review .................................................................................... 6  
Theoretical and Empirical Models Linking Neuroticism and Daily Experiences ...... 6  
Theoretical and Empirical Models Linking Neuroticism and Sleep Quality .......... 10  
Methodological Advantages of Repeated Daily Measures ........................................ 14  
The Current Study .......................................................................................................... 16  

## CHAPTER 3: Method .................................................................................................... 20  
Overview ......................................................................................................................... 20  
Participants .................................................................................................................... 20  
Procedure ....................................................................................................................... 21  
Measures ........................................................................................................................ 23  
Statistical Analysis Plan ................................................................................................. 29  

## CHAPTER 4: Results .................................................................................................... 44  
Preliminary Results ......................................................................................................... 44  
Aim 1 Results .................................................................................................................. 46  
Aim 2 Results .................................................................................................................. 47  
Aim 3 Results .................................................................................................................. 49  

## CHAPTER 5: Discussion ............................................................................................... 66  
Aim 1 Discussion of Findings ......................................................................................... 66  
Aim 2 Discussion of Findings ......................................................................................... 67  
Aim 3 Discussion of Findings ......................................................................................... 72  
Implications and Future Directions ............................................................................. 77  
Limitations ..................................................................................................................... 80  
Conclusion ...................................................................................................................... 84  

References ....................................................................................................................... 85  

Appendix A: Inclusion/Exclusion Criteria and Neuroticism Measures ..................... 108  
Appendix B: Waking Survey Measures ......................................................................... 110  
Appendix C: Bedtime Survey Measures ......................................................................... 112  
Appendix D: Sample SAS and R Code ........................................................................... 116  
Appendix E: Sample Scored Actigraphy Record using a Graphical User Interface.... 119  

iv
List of Tables

Table 1. Summary of Measures.............................................................................................41
Table 2. Participant Characteristics....................................................................................42
Table 3. Descriptive Statistics for Primary Measures........................................................53
Table 4. Between-Person and Within-Person Correlations for Primary Measures and Covariates..................................................................................................................54
Table 5. Effects of Neuroticism on Average Frequency of DNEs (Hypotheses 1.1.) and DPEs (Hypothesis 1.2.)..................................................................................................................55
Table 6. Moderated Effects of Neuroticism on the Associations between DNEs and NA (Hypothesis 2.1.), DNEs and PA (Hypothesis 2.2.), DPEs and PA (Hypothesis 2.3.), and DPES and NA (Hypothesis 2.4.)..................................................................................................................56
Table 7. Effects of Neuroticism on Average Daily NA (Hypothesis 3.1.) and Average Daily PA (Hypothesis 3.2.)..................................................................................................................58
Table 8. Effects of Neuroticism on Subjective and Objective Sleep Quality (Hypothesis 3.3.)...59
Table 9. Mediated Effects of Average Daily NA in the Association between Neuroticism and Sleep Quality (Hypothesis 3.4.)..................................................................................................60
Table 10. Mediated Effects of Average Daily PA in the Association between Neuroticism and Sleep Quality (Hypothesis 3.5.)........................................................................................61
List of Figures

Figure 1. Conceptual model.................................................................19
Figure 2. Flow diagram of study protocol and participants...........................................43
Figure 3. Primary neuroticism measure distribution.........................................................62
Figure 4. The moderated effect of neuroticism on the relationship between daily negative
experience frequency (DNE) and negative affect (NA; Hypothesis 2.1.)........................63
Figure 5. The moderated effect of neuroticism on the relationship between daily positive
experience frequency (DPE) and negative affect (NA; Hypothesis 2.3.)......................64
Figure 6. The moderated effect of neuroticism on the relationship between daily positive
experience frequency (DPE) and positive affect (PA; Hypothesis 2.4.).........................65
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CHAPTER 1: Introduction

The study of individual differences, or understanding variation between individuals and its underlying causes, is of rising interest in the fields of health psychology and behavioral medicine. Psychologists now concur that health behaviors interact in complex ways with individual predispositions and situational influences (Fleeson, 2004). With the rise of mobile technologies and advanced analytic techniques, it is now possible to non-invasively measure transactions between individuals and their environment over time in everyday life. Harnessing these techniques, behavioral scientists can integrate the assessment of individual- and daily-level risk factors to better characterize health behaviors and improve health trajectories.

Defining Neuroticism and Its Biological Correlates

One individual-level characteristic, neuroticism, is increasingly being acknowledged as a public health concern (Lahey, 2009). Neuroticism can be defined as the tendency to perceive one’s environment as threatening and difficult to manage and to possess the “behavioral and cognitive traits [such as] fearfulness, irritability, low self-esteem, social anxiety, poor inhibition of impulses, and helplessness” (Costa & McCrae, 1987, p. 301). These multiple facets of neuroticism are often related but partially distinct; therefore, neuroticism typically is conceptualized a heterogeneous trait (Lahey, 2009). Neuroticism is considered part of the Five-Factor Model (FFM) of personality (Costa & McCrae, 1985), and a distinct construct from the other four facets of the FFM: extraversion, openness to experience, agreeableness, and conscientiousness.

According to Eysenck (1967), neuroticism stems from a physiological predisposition toward an overreactive limbic system, or the areas of the brain that govern emotion regulation, memory formation, and the fear response. Individuals high in neuroticism tend to exhibit a low
threshold, high intensity, and long-lasting activation of the autonomic nervous system (ANS) in response to psychological stimuli (Eysenck, 1967). Neuroimaging studies have confirmed that neuroticism is correlated with brain regions involved in threat sensitivity and the fear response, such as the left amygdaloid complex (Koelsch, Skouras, & Jentschke, 2013), as well as resting-state regional cerebral blood-flow activity in the hippocampus, midbrain, and middle temporal gyrus (Sutin, Beason-Held, Dotson et al., 2010). Neuroticism also has been shown to be highly heritable (~15%; Smith, Escott-Price, Davies et al., 2016), and there is strong evidence it correlates with the 5-HTTLPR (serotonin-transporter-linked polymorphic region) and CRHR1 (corticotropin-releasing hormone receptor 1) genetic polymorphisms (Lahey, 2009; Smith et al., 2016). Together, these findings provide some evidence that neuroticism is a heritable trait-like phenotype with a corresponding profile that may predispose individuals to states of hyperarousal and hyperreactivity in the face of perceived threat.

**Types of Relationships between Neuroticism and Health**

Neuroticism is of great public health concern given its strong linkage to a constellation of poor health outcomes, including risk for depression (Boyce, Parker, Barnett et al., 1991; Duggan, Lee, & Murray, 1990; Muris, Roelofs, Rassin et al., 2005; Ormel, Oldehinkel, & Brilman, 2001; Roelofs, Huibers, Peeters et al., 2008a; Roelofs, Huibers, Peeters et al., 2008b; Surtees & Wainwright, 1996), cardiovascular disease (Čukić & Bates, 2015; Eysenck, 1985; Shipley, Weiss, Der et al., 2007), and sleep impairment as well as sleep disorders such as insomnia (Fernandez-Mendoza, Vela-Bueno, Vgontzas et al., 2010; Gurtman, McNicol, & McGillivray, 2014). Sleep may be a particularly important biobehavioral correlate of neuroticism considering the high prevalence of sleep-related problems reported in the United States population (Centers for Disease Control and Prevention, 2011; Institute of Medicine, 2006). Recent nationally
representative studies show approximately 35% of U.S. adults are not receiving sufficient amounts of sleep (i.e., at least 7 hours per night), and approximately 38% of U.S. adults report sleep-related impairment at least once in the past month (Centers for Disease Control and Prevention, 2011). Impaired sleep is linked robustly to variety of impaired cardiometabolic outcomes (Newman, Spiekerman, Lefkowitz et al., 2000; Stamatakis & Punjabi, 2010; Tasali, Leproult, Ehrmann et al., 2008) and premature all-cause mortality (Dew, Hoch, Buysse et al., 2003). Furthermore, there is a large degree of variation in sleep quality from individual to individual (Van Dongen, Vitellaro, & Dinges, 2005), warranting further investigation of potential individual-level characteristics, such as neuroticism, that may help explain some of this variation.

Multiple theoretical and empirical perspectives suggest that not only is neuroticism important for health, but that a variety of routes may exist by which neuroticism interacts with proximal health-relevant variables to predict distal health outcomes. For example, neuroticism may affect health through altering both physiological and behavioral pathways (e.g., poor health behavioral decision making and/or neuroendocrine-immune dysregulation), which over time can lead to disease inception and progression (Chapman, Roberts, & Duberstein, 2011). Individuals higher in neuroticism also may be more likely to perceive and complain about negative psychological states; such reports simply may reflect response biases or may represent processes that can lead to greater physiological impairment (Costa et al., 1987). Importantly, these two possibilities are not necessarily mutually exclusive: Those higher in neuroticism may both complain about greater health-related impairment, as well as actually experience greater health-impairment as measured through more objective means.
Together, theories investigating links between neuroticism and health imply that neuroticism can be investigated as a direct predictor (i.e., a vulnerability factor that directly predicts differential outcomes) or as a moderator of health-related variables (i.e., a vulnerability factor that modifies associations between other variables). However, these perspectives are often seen as mutually exclusive and do not harness an interdisciplinary perspective to understand the connections between neuroticism and health. Furthermore, most studies do not use both subjective and objective markers of health to untangle potential response biases of neuroticism. Finally, it is not well understood how the complex influence of neuroticism on health unfolds in daily life, as most previous studies of neuroticism and health have been cross-sectional or laboratory-based. To better understand how neuroticism may predict important health behaviors such as sleep in everyday life, daily processes, such as exposure to daily experiences and patterns of affect, must be explored using naturalistic, repeated measures. Using these methods, both differences between people, as well as differences within people from day to day in daily experiences, affect, and sleep can be assessed. Such types of investigations would allow for a more nuanced understanding of the specific individual-level and daily-level modifiable targets for potential intervention and prevention efforts to mitigate the detrimental effects of neuroticism on health.

**Overview of the Aims of the Current Study**

The overarching purpose of this dissertation is to examine the relationships between neuroticism and exposure to daily experiences, patterns of affect, and sleep quality. Specifically, new data were collected to enable the investigation of three aims: 1) examining the role of neuroticism in predicting exposure to daily positive and negative experiences, and 2) the moderating role of neuroticism in the association between exposure to daily positive and
negative experiences and daily positive and negative affect; and 3) the mediating role of daily positive and negative affect in the associations between neuroticism and objectively- and subjectively-assessed sleep quality. The overarching aims of this study are represented schematically in Figure 1 and then are discussed later in detail, with specific hypotheses. Drawing from diverse theoretical perspectives and empirical findings that speak to how neuroticism may interact with both daily experiences and sleep, a review of the literature is provided in the subsequent sections on: 1) theoretical and empirical models linking neuroticism and daily experiences 2) theoretical and empirical models linking neuroticism and sleep quality. Together, these two perspectives help identify the important gaps in the current literature that are addressed in the present study.
CHAPTER 2: Literature Review

Theoretical and Empirical Models Linking Neuroticism and Daily Experiences

With the appreciation for understanding “life as it is lived” and the rise of technology to capture such psychological processes in individuals’ everyday life, the assessment of daily experiences has become increasingly popular in personality-health research. It is well established that there is a great deal of individual, as well as day-to-day variation in the extent to which people are exposed to, respond to, and recover from these daily experiences (Williams, 2011). Across studies, the personality characteristic of neuroticism has been shown robustly to predict increased exposure and heightened negative affect (NA) in the face of daily negative experiences (Bolger, DeLongis, Kessler et al., 1989; Bolger & Zuckerman, 1995; Chapman et al., 2011; Gunthert, Cohen, & Armeli, 1999). Daily negative experiences (DNEs; often also referred to as daily stressors or hassles) are defined as the everyday irritating or undesirable demands, such as interpersonal conflicts, financial concerns, traffic and commuting, childcare coordination, or household chores, that have the potential to create adverse outcomes for an individual (Kanner, Coyne, Schaefer et al., 1981). Previous studies have found DNEs to occur fairly frequently, i.e., approximately 10-45% of all days in healthy adults (Almeida, McGonagle, & King, 2009; Piazza, Charles, Sliwinski et al., 2013; Sin, Almeida, Crain et al., 2017; Stawski, Sliwinski, Almeida et al., 2008), and an average of approximately once per day in both clinical and healthy adult samples (David, Green, Martin et al., 1997; Zautra, Affleck, Tennen et al., 2005).

**Differential exposure-reactivity model.** Bolger and Zuckerman’s (1995) differential exposure-reactivity model provides a theoretical framework for how neuroticism may be explained in the context of DNEs. Their model asserts that neuroticism can predict differential exposure to DNEs and/or differential reactivity to DNEs (Bolger et al., 1995). In other words,
individuals higher in neuroticism may *expose* themselves to (or be exposed to) more DNEs and/or *react* more strongly to DNEs (i.e., exhibit greater increases in NA) as compared to individuals lower in neuroticism. In a daily diary study, Bolger and Zuckerman (1995) found support for their theoretical model: Individuals higher in neuroticism reported both greater exposure to DNEs, as well as heightened NA reactivity compared to individuals lower in neuroticism. Other studies similarly have shown that, compared to those lower in neuroticism, individuals higher in neuroticism exhibit not only greater NA on days with more DNEs than their individual average (Bolger & Schilling, 1991; Bolger et al., 1995; Marco & Suls, 1993), but also more daily NA even in the absence of a DNE (Marco et al., 1993). Across studies, these results suggest both a direct role of neuroticism predicting more frequent exposure to DNEs, as well as a moderating role of neuroticism in increasing the strength of the positive association between frequency of DNE exposure and daily NA.

**Integrating daily positive experiences.** Although Bolger and Zuckerman’s (1995) model provides a useful framework for studying the influence of neuroticism on daily *negative* experiences, it does not acknowledge the influence of neuroticism in daily *positive* experiences. Daily positive experiences (DPEs) are everyday uplifting occurrences, such as task accomplishment or positive social interactions, that create adaptive outcomes for an individual (Kanner et al., 1981). Previous research in clinical and healthy adult samples has shown that DPEs occur an average of approximately one to five times per day (David et al., 1997; Sin, Graham-Engeland, & Almeida, 2015a; Zautra et al., 2005). DPEs tend to occur about two to five times more frequently than DNEs, on average (Zautra et al., 2005) and approximately 25-70% of all days (Sin et al., 2017; Sin et al., 2015a).
To account for the gap in Bolger and Zuckerman’s (1995) model, Zautra and colleagues (2005) developed a complementary theory, which states that personality also may influence one’s daily engagement in (similar to Bolger and Zuckerman’s concept of “exposure” to DNEs) and responsiveness to DPEs (similar to Bolger and Zuckerman’s concept of “reactivity”). Zautra and colleagues define daily uplift engagement as the degree to which one actively seeks out and participates in DPEs, and uplift responsiveness as the active benefit one acquires from engagement in these DPEs. Similar to Bolger and Zuckerman’s reactivity component, uplift responsiveness may relate to increases in positive affect (PA) and/or decreases in NA.

Because DPEs tend to occur more frequently, they tend to have a different meaning than DNEs (Taylor, 1991; Zautra et al., 2005). One rarely seeks out DNEs; in contrast, individuals tend to more actively seek out DPEs due to their rewarding nature. However, unlike DNEs, which are by definition undesirable, DPEs may vary in their level of desirability. It is therefore plausible DPEs may have a weaker relationship to daily well-being than DNEs do to daily NA. Although DNEs have been linked more strongly to health outcomes than DPEs, DPEs (or the lack thereof) may provide information about one’s daily life that is not captured by assessing DNEs alone (Sin et al., 2015a; Zautra et al., 2005). In fact, some studies have shown the occurrence of DPEs and DNEs to be uncorrelated (Zautra et al., 2005). Together, these findings support the necessity of investigating both DNEs and DPEs in the context of neuroticism.

The influence of neuroticism on the associations between DPE exposure and daily PA has not been as thoroughly investigated as the influence of neuroticism on the associations between DNE exposure and daily NA, nor has it been assessed in conjunction with other important health behaviors such as sleep. In one study, neuroticism was marginally inversely associated with some types of DPEs (work and leisure) averaged across an 8-day period, but not
with other types of DPEs (family/friends, financial, nor the total sum of all the types; David et al., 1997). A dyadic daily diary study of spousal neuroticism found that higher neuroticism was associated with lower average PA, higher average NA, as well as a greater number of DNEs (DPEs were not assessed; Lay & Hoppmann, 2014). In a test of their theoretical model described above, Zautra and colleagues (2005) found that neuroticism was associated with average DNE exposure but not DPE exposure in a sample of rheumatoid arthritis patients. In the same study, neuroticism did not moderate connections between DPE exposure and PA, but it did moderate connections between DNE exposure and NA: as a person's level of neuroticism increased, so did their tendency to report NA on days with more negative experiences than their typical amount. However, the Zautra and colleagues (2005) study is the only research that has examined the influence of neuroticism on the association between DPEs and PA. Given the unique characteristics of the Zautra and colleagues (2005) sample (i.e., all had rheumatoid arthritis), it is unknown if these findings replicate in healthier samples.

Together, previous research suggests that there may be between-person differences in the within-person associations between exposure to daily experiences and affect. However, the role of neuroticism across positive-negative domains (i.e., the links between DNEs and PA, and between DPEs and NA) has yet to be investigated. For example, it is unknown if days with more negative experiences than usual result in only higher NA, or also lower PA for those high in neuroticism (as compared to those lower in neuroticism). Because of the known distinct impact of NA and PA on health (e.g., Fredrickson, 2000; Kiecolt-Glaser, McGuire, Robles et al., 2002; Pressman & Cohen, 2005; Watson, 1988), these cross-domain effects represent important gaps in the literature that need to be better understood. Given the attentional bias toward negative stimuli that is characteristic of neuroticism, it is plausible that those higher in neuroticism may exhibit
more NA in the context of DNEs and less PA in the context of DPEs, as compared to those lower in neuroticism. Therefore, assessment of both DPE and DNEs in relation to neuroticism and other biobehavioral correlates would allow for better characterization of the distinct processes by which neuroticism may influence health.

**Theoretical and Empirical Models Linking Neuroticism and Sleep Quality**

In addition to exposure to daily experiences and patterns of affect, sleep is another important biobehavioral process with which neuroticism is strongly associated. Impaired sleep quality and also has strong clinical relevance, given impaired and short sleep’s (i.e., typically defined as <6 hours per night) robust linkage to a variety of detrimental cardiometabolic outcomes, including incident cardiovascular disease morbidity and mortality, myocardial infarction, hypertension, diabetes, and congestive heart failure (Knutson, 2010; Knutson, Spiegel, Penev et al., 2007; Newman et al., 2000), as well as reduced insulin sensitivity and glucose effectiveness (Spiegel, Knutson, Leproult et al., 2005; Stamatakis et al., 2010; Tasali et al., 2008). It is well established that sleep requires the downregulation of the autonomic nervous system (ANS; Covassin, Zambotti, Cellini et al., 2013; Holmes, Burgess, & Dawson, 2002), a system which also controls the stress response. Neuroticism’s strong hyperreactivity and hyperarousal profile (Bonnet & Arand, 1997; van de Laar, Verbeek, Pevernagie et al., 2010) and influence on stress processes (which are regulated by the sympathetic and parasympathetic branches of the ANS) suggests it is an important individual difference to examine in relation to sleep quality.

A number of cross-sectional studies have demonstrated that poor subjective sleep quality is associated with higher neuroticism (Calkins, Hearon, Capozzoli et al., 2013; Cheng, Shih, Lee et al., 2012; Duggan, Friedman, McDevitt et al., 2014). A prospective study has shown that
neuroticism predicts self-reported sleep quality approximately two months later (Williams & Moroz, 2009), and an experimental laboratory-based study has shown that following experimental sleep deprivation, higher neuroticism is associated with participants feeling sleepier (Mastin, Peszka, Poling et al., 2005). However, no naturalistic studies utilizing repeated measures of both objective and subjective measures of sleep quality in daily life and linking these measures to neuroticism have been conducted. As such, it is unknown how neuroticism relates to different facets of sleep quality in everyday contexts.

**The hyperarousal and cognitive models of insomnia.** The hyperarousal and cognitive models of insomnia (Harvey, 2002; Riemann, Spiegelhalder, Feige et al., 2010) help provide a theoretical framework for how neuroticism may be related to impaired sleep quality, and how affect may be an intervening link in this relationship. According to these models, neurobiological hyperarousal (e.g., corticol activation) and cognitive-affective hyperarousal make the act of falling asleep, maintaining sleep, or obtaining restorative sleep more difficult (Harvey, 2002; Riemann et al., 2010). The hyperarousal profile typical of individuals high in neuroticism may predispose them to dysfunctional cognitions (e.g., rumination, worry over sleep loss), negative psychological consequences (e.g., mood disturbance, fatigue, impairment of performance), and maladaptive habits (e.g., excessive time in bed, irregular sleep cycle; Fernandez-Mendoza et al., 2010; Harvey, 2002; Riemann et al., 2010). In turn, these processes may lead to or exacerbate sleep quality disturbances, such as increased sleep latency (time it takes to fall asleep after getting into bed with the intention of sleeping), more time awake after sleep onset, or decreased sleep efficiency (ratio of time actually asleep to time in bed with the intention of sleeping). Indeed, across a number of studies, individuals with sleep quality-related issues report more excessive negatively-toned psychological states during both the pre-sleep period and throughout
the day in comparison to individuals without such issues (for a review, see Harvey, 2002). If this state of hyperarousal and sleep disturbance persists over time, escalation to clinical levels of insomnia may occur in some individuals. Therefore, it is important to identify premorbid vulnerabilities for poor sleep before these vulnerabilities may translate into actual sleep disturbances.

**Daily negative affect as a link between neuroticism and sleep quality.** Although there are some empirically demonstrated associations between neuroticism and sleep quality, mediators of this relationship have been largely unexplored. Investigation of potential daily psychological pathways in the link between neuroticism and sleep quality would enable better characterization of how this process unfolds in daily life and identify potential clinical targets. Although it has not been systematically tested, affect may be a potential mediating factor in this link between neuroticism and sleep quality, given its robust linkage to arousal of the ANS (for a review, see Kreibig, 2010), as well as to both neuroticism (as reviewed in previous sections) and sleep quality (as reviewed below).

Cross-sectionally, greater NA has been associated with poorer subjective sleep quality (Brummett, Babyak, Siegler et al., 2006; Norlander, Johansson, & Bood, 2005). However, in a daily study of healthy control participants and participants with major or minor depression, sleep quality assessed cross-sectionally was not associated significantly with ambulatory NA in the full sample (Bower, Bylsma, Morris et al., 2010). In a study of healthy young women, pre-sleep arousal (cognitive and somatic) mediated the relationship between daily stress and self-reported sleep quality (Winzeler, Voellmin, Schäfer et al., 2014), and a similar study found that increased daily NA mediated the relationship between interpersonal conflict and subjective sleep quality (Brissette & Cohen, 2002). Days with greater NA than average also have been associated with
poorer subjective but not objective sleep quality in older adults (McCrae, McNamara, Rowe et al., 2008). Together, these studies show some evidence of associations between NA and subjective sleep quality. However, the role of daily NA as a potential link between neuroticism and sleep quality has not been examined. Further, most studies of neuroticism and sleep, or of affect and sleep, have not included confirmation of sleep quality through more direct, objective measures such as actigraphy, which would help clarify potential response biases.

**Integrating daily positive affect.** An even less understood component of the neuroticism-sleep connection is the role of daily PA. Although the hyperarousal and cognitive models of insomnia provide theoretical support for the premise that NA impairs sleep quality, many fewer studies have investigated PA in relation to sleep quality. Days with lower PA than one’s individual average have been associated with poorer self-reported sleep quality in a few daily diary studies (McCrae et al., 2008; Ong, Exner-Cortens, Riffin et al., 2013; Sin et al., 2017). Another study found that daily aggregated PA was inversely associated with cross-sectionally assessed self-reported sleep problems (Steptoe, O'Donnell, Marmot et al., 2008). A review of sleep quality and emotions in the context of insomnia found that poor sleep quality was related to lower PA in both clinical and sub-clinical insomnia populations; however, these studies also were primarily cross-sectional (Baglioni, Spiegelhalder, Lombardo et al., 2010). Further, none of these studies examined how daily PA may mediate the relationship between neuroticism and sleep quality in daily life. Therefore, little is known about whether neuroticism is associated with only greater daily NA, or also lower daily PA, and how NA and PA may be differentially influential for sleep quality measured through subjective and more objective means. Although PA and NA are not wholly independent of each other, evidence suggests they are at least partially distinct concepts and appear to operate independently at the biological level.
with divergent physiological effects (Cacioppo & Berntson, 1994; Cacioppo, Gardner, & Berntson, 1999; David et al., 1997; Fredrickson, 2001; Watson, Clark, & Tellegen, 1988). Therefore, consideration of not only NA but also PA when investigating links between neuroticism and sleep quality is essential for informing potential prevention and intervention efforts.

**Methodological Advantages of Repeated Daily Measures**

Although some is known about these associations between neuroticism, exposure to DPEs and DNEs, affect, and sleep quality, most of this work has been cross-sectional; therefore, little is about how these relationships unfold in daily life. The information obtained from cross-sectional designs of personality and health can be valuable for informing longitudinal or intervention studies, but it is limited in its reliability and external validity. Studies using repeated daily measures (RDM) allow for two primary methodological features that are not possible in cross-sectional or laboratory-based designs: 1) assessment in closer to real-time in real-world contexts, and 2) assessment over time to investigate temporal dynamics (Ebner-Priemer & Trull, 2012; Shiffman, Stone, & Hufford, 2008).

**Assessment in closer to real-time in real-world contexts.** Foremost, by taking measurements over the course of multiple days in participants’ daily lives, researchers can prevent the memory bias that is present in longer-term retrospective reports, more clearly categorizing the “experiencing self,” opposed to the “remembering self” (Conner & Barrett, 2012). End-of-week or end-of-month reports tend to be heavily biased toward reporting of the most recent or intense experience, whereas daily reports can minimize the use of heuristics to recall information. Importantly, daily assessments also have stronger associations with bodily
experiences and physiological measurements than do generalized or longer-term retrospective reports obtained in cross-sectional studies (Conner et al., 2012).

Furthermore, RDM allow for enhanced ecological validity, or generalizability to real-world phenomena (Ebner-Priemer & Trull, 2009; Ebner-Priemer et al., 2012; Scollon, Prieto, & Diener, 2009; Shiffman et al., 2008). Many of the most popular stressor paradigms employed in behavioral research are not stressors one would commonly or ever face in everyday life (e.g., serial subtraction in front of an audience), and therefore, they may not characterize accurately individuals’ “typical” responses to real-world situations. Other constructs like health behaviors cannot be measured with validity in a laboratory setting, as they are dependent on one’s transaction with environmental influences (Ebner-Priemer et al., 2012). For example, laboratory-based sleep studies may not accurately characterize an individual’s typical sleep patterns and actually may be disruptive for sleep. In contrast, measurements taken in everyday using ambulatory devices may be less invasive and disruptive for sleep.

Assessment of temporal dynamics. The other primary advantage of RDM is the ability to determine relationships over time. Not all individuals respond to stimuli in the same manner on average, nor does one individual typically respond in the same manner across time (Wilhelm, Perrez, & Pawlik, 2012). From a statistical standpoint, the dense sampling that is characteristic of RDM can enable examination of both between- and within-person effects (i.e., the investigation of how people differ from each other and how people differ from themselves from day-to-day). Such studies also can aggregate daily measures to the between-person level to capture individual’s “typical” responses as they are lived, presumably with more accuracy than one-time retrospective reports (Shiffman et al., 2008; Stone & Shiffman, 1994).
The Current Study

In summary, previous studies have demonstrated some associations between neuroticism, daily experiences, affect, and sleep quality. Yet, the mechanisms and specific types of relationships between these variables are still relatively untested in daily settings using comprehensive repeated measures of both daily experiences and sleep. No studies have been conducted investigating *a priori* the relationships between neuroticism, exposure to both DPEs and DNEs, daily PA and NA, and subjectively- and objectively-assessed sleep quality with an ecological daily diary design. Therefore, a theoretical and methodological disconnect remains between the literature linking neuroticism to daily experiences and the literature linking neuroticism to sleep quality. Integration of the differential exposure-reactivity model with the hyperarousal and cognitive models of insomnia would facilitate a more complete understanding of how neuroticism, daily experiences, and sleep quality interact in everyday life. This approach would provide a better characterization of who may be at a greater risk for adverse outcomes, as well as the daily affective patterns that might be important for health.

Given these gaps in the literature, the overall goal of the proposed study was to investigate: 1) the role of neuroticism in predicting reported exposure to DPEs and DNEs, 2) the moderating role of neuroticism in the associations between exposure to DPEs and DNEs with reported daily PA and NA, and 3) the mediating role of daily PA and NA in the association between neuroticism and subjective and more objective measures of sleep quality. To investigate these aims, a 14-day daily diary design was employed, using a combination of cross-sectional, fixed interval daily self-reports, and ambulatory actigraphy measurements. The study was conducted in a sample of relatively healthy young adults to avoid the known confounds that increased age and chronic conditions contribute to affect regulation and sleep-related impairment.
(e.g., see Foley, Ancoli-Israel, Britz et al., 2004; Smith & Haythornthwaite, 2004). The three aims and corresponding hypotheses of the current study are listed below.

**Aim 1:** To investigate the degree to which neuroticism predicts average frequencies of reported DPEs and DNEs assessed across a 14-day daily diary study.

*Hypothesis 1.1.* Neuroticism will be associated positively with average DNE frequency.

*Hypothesis 1.2.* Neuroticism will be associated negatively with average DPE frequency.

**Aim 2:** To examine the cross-level moderation effects of neuroticism in the links between reported DPE and DNE frequency and daily positive and negative affect assessed across a 14-day daily diary study.

*Hypothesis 2.1.* Neuroticism will moderate the association between DNE frequency and daily NA; specifically, days with a greater number of DNEs than one’s individual average will be more strongly positively associated with daily NA for those with higher neuroticism than for those with lower neuroticism.

*Hypothesis 2.2.* Neuroticism will moderate the association between DNE frequency and daily PA; specifically, days with a greater number of DNEs than one’s individual average will be more strongly negatively associated with daily PA for those with higher neuroticism than for those with lower neuroticism.

*Hypothesis 2.3.* Neuroticism will moderate the association between DPE frequency and daily NA; specifically, days with a greater number of DPEs than one’s individual average will be less strongly negatively associated with daily NA for those with higher neuroticism than for those with lower neuroticism.

*Hypothesis 2.4.* Neuroticism will moderate the association between DPE frequency and daily PA; specifically, days with a greater number of DPEs than one’s individual average will be
less strongly positively associated with daily PA for those with higher neuroticism than for those with lower neuroticism.

**Aim 3:** To investigate the mediating role of average daily PA and NA in the association between neuroticism and average nightly sleep quality assessed across a 14-day daily diary study.

*Hypothesis 3.1.* Neuroticism will be associated positively with average daily NA.

*Hypothesis 3.2.* Neuroticism will be associated negatively with average daily PA.

*Hypothesis 3.3.* Neuroticism will be associated negatively with nightly subjective and objective sleep quality.

*Hypothesis 3.4.* Average daily NA will mediate the association between neuroticism and nightly subjective and objective sleep quality. Specifically, neuroticism will be associated positively with average daily NA (Hypothesis 3.1.), and average daily NA will be associated negatively with nightly subjective and objective sleep quality after controlling for neuroticism. The product term of these two effects will be significant. Together, these expected results would support the hypothesis that average daily NA will mediate the relationship between neuroticism and nightly subjective and objective sleep quality.

*Hypothesis 3.5.* Average daily PA will mediate the association between neuroticism and nightly subjective and objective sleep quality. Specifically, neuroticism will be associated negatively with average daily PA (Hypothesis 3.2.), and average daily PA will be associated positively with nightly subjective and objective sleep quality after controlling for neuroticism. The product term of these two effects will be significant. Together, these expected results would support the hypothesis that average daily PA will mediate the relationship between neuroticism and nightly subjective and objective sleep quality.
Figure 1. Conceptual model.
CHAPTER 3: Method

Overview

This study consisted of three components: 1) an online survey to determine eligibility and baseline characteristics, 2) a brief in-person training session for step three, and 3) a 14-day daily diary component that consisted of ambulatory actigraphy assessment and smartphone-assisted twice-daily surveys. Additional details regarding the participants, procedure, measures, and statistical analyses are provided in subsequent sections.

Participants

Participants were 60 healthy young adults ($M_{age} = 25.33 \pm 3.98$ years old, 53% female; for additional demographic characteristics, see Table 2.) To be included in the study, participants were required to be between the ages of 21 and 35, fluent in English, and have a personal smartphone with a cellular data and unlimited texting plan for completion of the daily surveys. A relatively young age range was chosen due to confounds that increased age creates with increased likelihood for chronic disease and dysregulated sleep (Foley et al., 2004), but a minimum age of 21 was chosen to avoid having younger college students in the sample (who often exhibit more atypical sleep cycles; Hershner & Chervin, 2014; Lund, Reider, Whiting et al., 2010). English fluency and owning a personal smartphone with a cellular data and unlimited texting plan were necessary criteria for completion of all survey materials and to eliminate cost burden for survey completion. Individuals were excluded if they had a chronic inflammatory disease or autoimmune problems (e.g., cancer, HIV/AIDS, rheumatoid arthritis, multiple sclerosis, lupus), or were pregnant or nursing, all of which are major confounds for sleep (Lashley, 2003; Sahota, Jain, & Dhand, 2003).
Procedure

Recruitment and study timeline. (See Figure 2 for a flow diagram of the study protocol and participants.) Participants were recruited through convenience sampling methods on a rolling basis via campus and community posters, campus list-services, laboratory websites, and online research volunteer forums. Interested individuals \( n = 163 \) emailed or called the laboratory, upon which they were emailed a link to the screening survey. One hundred and fifty-one individuals (93%) completed the initial screening, and 119 of the 143 eligible individuals were emailed a link to complete the online baseline survey to answer additional questions on health status, personality (including the neuroticism assessment), and demographics. The 119 individuals who were sent the baseline survey (83% of eligible individuals) were selected based on timing of initial contact and based on their gender, as there were more eligible women than men, and a more balanced gender breakdown of participants was sought. Of the 119 participants sent the baseline survey, 89 (75%) completed the survey and were invited to sign up for an in-person training session to participate in the daily portion of the study. Sixty-seven participants (75%) signed up for the daily diary training session, and 60 participants (90%) attended the training and completed the daily diary portion of the study. Based on the order in which they initially contacted the lab, participants were divided into seven cohorts to ensure more effective study management and due to the limited number of actigraphy devices available. At the end of the daily portion, participants returned the actigraphy device and collected compensation. All study procedures were approved by The Pennsylvania State University Institutional Review Board prior to implementation.

Training sessions. In-person training sessions for instruction on the daily diary and actigraphy protocol were conducted with participants after they completed the baseline survey.
Informed consent was obtained at this time. A standardized presentation (for instruction delivery consistency) provided participants with details on smartphone survey terminology, timing, interface, as well as actigraphy device instructions, study compensation, and technology troubleshooting. Participants registered their personal smartphones using the SurveySignal program, practiced using their smartphone to take a sample daily survey, and practiced wearing the actigraphy device. All training sessions were led by the same experimenter to avoid confounds with interrater reliability. At the end of the training session all participants completed a short quiz, and answers were reviewed to ensure comprehension of the training instructions.

**Daily diary and actigraphy protocol.** The daily diary and actigraphy protocol began either one or two days after the training session and involved 14 days of mobile smartphone-assisted survey completion, using a combination of fixed interval signals (i.e., waking and bedtime surveys) and passive ambulatory data collection (i.e., using the actigraphy device). At 6:00 a.m. each day, a text message was sent via SurveySignal (SurveySignal, Chicago, IL) containing an embedded Qualtrics link (Qualtrics, Provo, UT) to complete the waking survey. Participants were instructed to complete the waking survey within 30 minutes after waking, but the link expired at 12:00 p.m. to allow those who awakened in late morning to report. This survey involved self-report of the previous night’s sleep duration, time in bed, sleep timing, sleep latency, sleep-related disturbances, and past 24-hour alcohol and caffeine use. Similarly, at 9:00 p.m. each night, a text message was sent via Survey Signal containing an embedded Qualtrics link to complete the bedtime survey. Participants were instructed to complete the bedtime survey within 30 minutes prior to going to sleep, but the link expired at 3:00 a.m. the next day to allow those who went to sleep late to report. This survey involved self-report of DPEs and DNEs, PA,
NA, somatic symptoms, sleep-related impairment, physical activity, rumination, and mindfulness.

For determination of objective sleep quality measures, participants were instructed to wear a Respironics Actiwatch Spectrum Device™ (Philips/Respironics; Murrysville, PA) for the entirety of the 14-day daily diary portion of the study. The Actiwatch™ tracked periods of sleep and wake with an actigraphy sensor and with light sensors that recorded red, green, blue, and photopic light (i.e., the combination of the three light spectrums). Off-wrist detection capabilities determined when the device was not in use. All devices were calibrated by Philips prior to usage and independently validated before distribution to participants. Devices were configured in Philips Actiware Sleep Scoring Program™ Version 6.0.7. (Philips/Respironics; Murrysville, PA) to collect data at 30-second epochs over the 14-day daily diary data collection period.

Compensation. For completion of the baseline survey, participants were entered into a raffle for a chance to win one of 20 prizes of $10 each. Odds of the raffle were approximately 1 in 5, as 89 participants completed the baseline survey. Participants were compensated $1.00 for each of the 28 daily surveys they completed, and a $10 bonus for greater than 90% completion (i.e., at least 25 of the 28 daily surveys); therefore, participants had the chance to receive up to $38 for the daily diary portion. Thus, daily diary-eligible participants who completed the entire study (baseline survey and 14-day daily diary period) with good compliance had a chance to earn up to $48. As an additional incentive, participants were given the option to receive a summarized report of their sleep quality data collected via actigraphy, distributed after study completion.

Measures

For a full list of all measures used in each portion of the study, see Appendixes A-C. For a summary of each construct and related measures, see Table 1.
Neuroticism. Neuroticism was assessed at baseline using the widely implemented and validated 12-item neuroticism subscale from the 24-item Eysenck Personality Questionnaire-Brief Version (EPQ-BV; Sato, 2005), taken from the longer 48-item Eysenck Personality Questionnaire–Revised (EPQR-S; Eysenck, 1992). Each item in the 12-item scale is rated on a scale of 1 (not at all) to 5 (extremely), with possible scores ranging from 12 to 60. This scale has good internal consistency ($\alpha = .90$), test-retest reliability ($r = .92$), and convergent validity with the full-length Eysenck Personality Questionnaire Revised measure from which it was derived ($r = .89$; Sato, 2005). Examples of questions include: “Would you call yourself a nervous person?” “Does your mood go up and down?” and “Are you an irritable person?” (See Appendix A for a full list of questions.)

For additional confirmation and validation of the neuroticism construct (as well as the other five-factor personality characteristics), the Mini-International Personality Item Pool (IPIP; Donnellan, Oswald, Baird et al., 2006) also was completed at baseline. The Mini-IPIP is a 20-item scale that measures neuroticism, in addition to extraversion, agreeableness, conscientiousness, and intellect/imagination, on a scale of 1 (very inaccurate) to 5 (very accurate), with total possible scores ranging from 20 to 100. The 5-item neuroticism subscale has acceptable internal consistency ($\alpha = .70$) and convergent validity ($r = .93$) with the original full-length 50-item International Personality Item Pool-Five-Factor Model measure from which it was derived (Goldberg, 1999).

Daily positive and negative experiences. DPEs and DNEs were measured once daily at the bedtime survey using an abbreviated version of the Hassles and Uplifts Scale Short-Form (DeLongis, Folkman, & Lazarus, 1988). The original short form of the scale contains 53 items, but for the current study, items with similar topics were condensed into one item for brevity, and
items that are likely irrelevant to younger adult populations were removed (e.g., childrearing-related experiences; see Appendix C for a full list of measures), resulting in a total of 19 items. Participants were instructed to rate how much of an uplift (i.e., DPE) and how much of a hassle (i.e., DNE) each item was for them that day, on a scale of 0 (none or not applicable) to 3 (a great deal). This approach provides flexibility and discriminate validity of daily positive and negative experience measurement; items can be rated as either or both positive and negative daily experiences with varying intensities. Example items include “amount of free time,” “your supervisor or employer,” and “social commitments.” Frequency of both DPEs and DNEs was calculated by summing the items rated as greater than 0 for each day.

**Daily positive and negative affect.** In the bedtime survey, daily positive affect (PA) and negative affect (NA) were assessed. A combination of scales was used to represent fully both high arousal and low arousal PA and NA, which is an important distinction according to the circumplex model of affect (Diener, Larsen, Levine et al., 1985; Russell, 1980). High arousal PA (“Please indicate to what extent you have felt each emotion today—determined, happy/joyful, inspired, attentive/alert”) and high arousal NA (“Please indicate to what extent you have felt each emotion today—fearful/afraid, nervous/anxious, angry/annoyed, ashamed”) were each assessed on a scale of 0 (“not at all”) to 100 (“extremely”) using slightly revised versions of the internationally-validated five-item scale versions (original PA scale: determined, inspired, alert, active, attentive; original NA scale: afraid, nervous, upset, ashamed, hostile; Thompson, 2007) derived from the 10-item PA and NA subscales from the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). The original five-item PA and NA subscales each have good internal consistency (α = 0.80 and α = .74, respectively), and have been shown to be significantly negatively correlated with each other (r = −0.32, p < .01; Thompson, 2007). Low arousal PA
(“Please indicate to what extent you have felt each emotion today—relaxed/calm, grateful/appreciative, content/satisfied”) and low arousal NA (“Please indicate to what extent you have felt each emotion today—lonely, sad, guilty”) were each assessed on a scale of 0 (“not at all”) to 100 (“extremely”) using slightly revised wording of items from the original Profile of Mood States (POMS; McNair, Lorr, & Dropelman, 1981; Nezlek, 2005). Items from the high arousal and low arousal PA and NA scales were averaged together within each day for each participant to form a daily measure of both PA and NA.

**Objective sleep quality.** To derive measures of objective sleep quality, actigraphy data were utilized. Actigraphy-derived measures of sleep quality have been shown to be valid and correlate strongly with polysomnography-derived measures \( r = 0.86 \) (Marino, Li, Rueschman et al., 2013). For the current study, actigraphy data were collected, downloaded, and exported from the Respironics Actiwatch Spectrum Device™ using Actiware software (Philips/Respironics, Murrysville, PA). A customized SAS macro algorithm and a graphical user interface (GUI; for an example of the GUI, see Appendix E) were used to score the actigraphy data (using scoring criteria described in more detail below). The macro estimated cutpoints (i.e., the “start” and “end” of a day), rest intervals (i.e., time in bed), sleep intervals (i.e., time elapsed from sleep to wake), and valid days. All computer-scored records then were reviewed and updated manually by two independent, trained scorers using the GUI, and were adjudicated by an independent third scorer for determination of finalized records. (Given that the SAS macro and GUI were being updated continuously at the time of the actigraphy scoring, there were many differences in the cutpoints and valid days between the two trained scorers. All discrepancies were adjudicated by an experienced third scorer using a more updated version of the SAS macro and the GUI.)
Therefore, measures of interrater reliability between scorers are likely invalid and are not reported.)

Cutpoints were determined for each participant based on a hierarchy of criteria: 1) selection of a time that intersected the minimum number of rest intervals, 2) selection of a time that intersected the minimum number of off-wrist periods, and 3) selection of a time that maximized the number of valid days of the recording (described in more detail below). The start of a rest interval was determined by examining the last epoch (i.e., 30-second recording interval) with an activity count of >10 prior to 5 consecutive epochs with activity counts of ≤10, and the end of a rest interval was determined by identifying the first epoch with an activity count of >10 following 5 consecutive epochs with activity counts of ≤10. Light levels were utilized in difficult recordings as a sufficient but not necessary additional criterion for confirmation of rest interval start and end. The start of the sleep interval was determined as the first epoch followed by 10 consecutive immobile (i.e., epochs with an activity count of 0 or 1) minutes after the start of the rest interval. The end of a sleep interval was determined as the last epoch preceded by 10 consecutive immobile minutes before the end of the rest interval. Naps were considered sleep intervals ≥30 minutes outside of the main rest interval for the day. Days were considered invalid if: 1) the participant had the device off-wrist for a total of ≥4 hours over the course of one day, 2) there was a total of ≥4 hours of invalid data (i.e., periods of device error, communication error, or data corruption) over the course of one day, or 3) there was an off-wrist period of ≥60 minutes ending within 10 minutes of a main rest interval start time or beginning within 10 minutes of a main rest interval end time. Exceptions to the invalid day criteria were made for the first day of recording (i.e., the day participants received the device) and the last day of recording (i.e., the day participants returned the device).
To determine main rest interval and sleep duration, the SAS macro (and each scorer if the macro was incorrect) estimated time elapsed during the main rest and sleep interval each day (based on the above criteria). To determine WASO, the SAS macro calculated the total number of minutes determined as wake (i.e., activity counts > 20 per 30-second epoch) between the start time and the end time of the main sleep interval. To determine objective sleep latency, the SAS macro calculated the time between the start of a given rest interval and the start of a given corresponding sleep interval in minutes. To determine objective sleep efficiency, the SAS macro calculated the scored total sleep time (i.e., sleep interval duration minus WASO) divided by the total rest interval duration (i.e., time in bed), multiplied by 100.

**Subjective sleep quality.** As measures of subjective sleep quality, participants reported on subjective sleep disturbances and sleep-related impairment. In the waking survey, participants reported on previous night sleep disturbances. This measure was derived from the PROMIS™ Sleep Disturbance short form (Yu, Buysse, Germain et al., 2012), adapted for daily measurement. This scale contains eight items, such as “I had difficulty falling asleep,” and “My sleep was restless,” each rated on a scale of 1 (not at all) to 5 (very much), with possible total scores ranging from 8-40. In addition to these questions, in the waking survey, participants reported on the time they got into bed with the intention of sleeping, the time they got out of bed in the morning, the amount of time it took them to fall asleep, and the number of hours of actual sleep they received. In the bedtime survey, participants reported on that day’s sleep-related impairment, as measured by the PROMIS™ Sleep-Related Impairment short form (Yu et al., 2012), adapted for daily measurement. This scale contains eight items, such as “I had trouble staying awake during the day,” and “I had a hard time getting things done because I was sleepy,” each rated on a scale of 1 (not at all) to 5 (very much), with possible scores ranging from 8-40.
Each of these scales correlates robustly with the longer forms, and demonstrates larger test information values than the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale, suggesting greater measurement precision with fewer total items (Yu et al., 2012). Items from the Sleep-Related Impairment (SI) and the Sleep Disturbance (SD) scales were averaged together within each day for each participant to form daily measures of SI and SD.

**Covariates.** For all analyses, baseline age, gender, race, ethnicity, education, and annual household income were used as covariates. These variables were chosen to ensure neuroticism, and not demographic characteristics with which it has been shown to overlap (Costa, Terracciano, & McCrae, 2001; Donnellan & Lucas, 2008; Sutin, Costa, Miech et al., 2009), were explaining variation in the outcomes. Due to low variability in the race, education, and annual household income variables, race was dichotomized into “white” and “not white,” education was dichotomized into “no college” and “at least some college,” and annual household income was dichotomized into “below $59,999” and “above $60,000. Although they were not included as covariates, to further characterize baseline health characteristics of the sample, the Center for Epidemiological Studies-Depression Scale (Radloff, 1977) was used to assess recent depressed mood, and body mass index (BMI) was calculated by taking each participant’s weight in pounds divided by their squared height in inches, multiplied by 703.

**Statistical Analysis Plan**

**General analyses types.** All analyses were conducted using SAS software, version 9.4. (SAS Institute Inc., 2008, Cary, NC, USA) and R software, version 3.2.4. (R Core Team, 2013, Vienna, Austria) . Preliminary descriptive analyses on means, standard deviations, frequencies, outliers, missing data, and bivariate between- and within-person (i.e., person-mean centered) correlations were conducted on all primary study variables. Multilevel modeling (MLM) and
ordinary least squares (OLS) regression techniques were applied to examine Aims 1-3 using the SAS program PROC MIXED for MLM and PROC REG for OLS regression. (For sample code for all aims, see Appendix D.)

MLM is designed to analyze data that are nested, or when one level of data is dependent on another level of data (e.g., days of a study nested within people). Because these types of data violate assumptions of independence of errors, it is necessary to account for the variance at each level of analysis. Unlike OLS regression, MLM can simultaneously account for this variance at multiple levels and also robustly can handle data that are unbalanced, or data where participants do not all provide the same amount of information, or in this case, the number of days on which participants valid data (Nezlek, 2012). For all MLMs, unconditional means models (i.e., model with no predictors) first were run for each variable. Intraclass correlation coefficients (ICCs), or the ratio of the between-person variance to the total variance (i.e., between-person plus within-person variance) then were calculated to determine the proportion of variance at each level of analysis (i.e., level 1 days and level 2 people). OLS regression was used for Hypothesis 3.1. and 3.2. in the context of level 2-2-1 mediation modeling (see pg. 37-38 for more details).

**Missing data.** When missing data are not handled properly, a loss of statistical power can result. Therefore, missing data were handled both proactively and retroactively. Proactively, efforts were made to minimize non-responsiveness through monetary compensation, by assuring anonymity of the data, by minimizing response burden, by thoroughly explaining the purpose of the study and the assessment timing in the training sessions, and by providing reminder emails and staying in frequent contract with participants (Black, Harel, & Matthews, 2012). Data collection itself can result in three types of missing data: data missing completely at random (MCAR; i.e., when the probability that a value is missing does not depend on the observed data.
values or on the missing data values), data missing at random (MAR; i.e., when the probability a value is missing depends partly on the data observed but not any of the values missing), or missing not at random (MNAR; i.e., when the probability a value is missing depends on the missing data values themselves; Black et al., 2012; Newman, 2009). Full information maximum likelihood (FIML) estimate was applied to account for unbalanced data assumed to be MCAR or MAR (i.e., when individuals in the sample have varying numbers of observations; Black et al., 2012). FIML uses all complete and partially observed cases in maximum likelihood estimation, with missing data values considered as random variables that can be “averaged over” (Black et al., 2012). FIML reduces bias due to data that are MNAR even though they assume data are MAR (Schafer & Graham, 2002).

**Statistical power and effect sizes.** To reject the null hypothesis (which states that there is no relationship between the variables of interest), power should be at least 0.80. For MLM analyses, power depends on the total sample size and the statistical model used. The total sample size is the number of statistically independent observations available for the study, or in the case of daily diary research (i.e., when the levels of data are not statistically independent), the total number of observations (i.e., the number of participants multiplied by the number of data collection time points) adjusted for within-person correlations. Statistical power estimations for daily diary studies can be complicated by the multiple sources of randomness, such as the level of assessment and the type of effect being estimated (e.g., fixed, random, cross-level interaction; Snijders, 2005). The variance in lowest level (e.g., level 1; in this case, daily assessments) residual errors often is accurately estimated, but the variance in higher group level (e.g., level 2; in this case, participants) errors often is underestimated (Maas & Hox, 2005). Simulations show
that a small level-2 sample size (<50) can lead to biased level-2 standard error estimates (Maas et al., 2005).

Therefore, previous multilevel power simulation studies suggest following the “50/20 rule,” particularly when cross-level interactions are of interest (Hox, 2010). Assuming a standard .05 alpha, 50 participants with at least 20 observations each provides sufficient power to detect small and medium-sized effects. The current sample size of 60 participants with 14 days of assessment, suggested nearly sufficient power to meet the study’s aims. Linear mixed model sample size calculations (using methods from Donohue & Edland, 2013; Lu, Luo, & Chen, 2008) confirmed that a sample size of $N_{persons} = 60$ elicited sufficient power (i.e., 0.80) to detect significant direct effects and cross-level interaction effects at the $p < .05$ level.

For an approximation of total effect sizes for each model using MLM, pseudo-$R^2$ values for level 1 (when applicable) and level 2 effects were calculated by subtracting the error variance after all predictors were added from the level 1 and level 2 error variances in the unconditional model, divided by the error variance of the unconditional model (Holden, Kelley, & Agarwal, 2008). Negative pseudo $R^2$ values (i.e., when the inclusion of additional predictors increased the variance components) were considered uninterpretable, according to recommendations from Holden et al. (2008). Adjusted $R^2$ values, or the percentage of variance explained by only the predictor variables affecting the outcome, were used as an approximation of total model effect size for OLS regression.

**Centering.** An additional major concern within MLM is appropriate centering, as this determines the interpretation of the intercept (Nezlek, 2012). Binary level 2 variables (e.g., gender coded as 0 = male, 1 = female) were left uncentered given that 0 represented a meaningful value, in accordance with recommendations from Nezlek (2012). By leaving these
variables uncentered, the intercept represents the expected value of the outcome when predictors are 0 (e.g., for men). Continuous level 2 variables (e.g., neuroticism and age) were grand-mean-centered (i.e., mean across all participants subtracted from an individual’s score) according to recommendations from Nezlek (2012). With this type of centering, the intercept represents the expected value of the outcome for a “typical” person, i.e., someone who is at the grand mean of the predictor. For Aim 2, continuous level 1 variables (e.g., DPE and DNE frequency) were person-mean centered (i.e., mean across all days for each person subtracted from each person’s daily score). With this type of centering, the intercept represents the expected value of the outcome on each person’s “typical” day, i.e., days with average levels of the predictor.

**Within-person reliability.** For daily repeated measures that contained only one item per scale (i.e., WASO, SE, SL), ICCs were calculated as measures of reliability (see above under “General analysis types”). For daily repeated measures that contained more than one item per scale (i.e., DPE, DNE, NA, PA, SI, and SD), according to recommendations from Bonito, Ruppel, and Keyton (2012), three-level unconditional models were run to estimate variances at item-, day-, and person-levels of analysis. The below equation was used for calculating day-level (i.e., level 1) reliability estimates:

\[ \alpha_{\text{day-level}} = \left( \frac{\sigma^2_{\text{day-level}}}{\sigma^2_{\text{day-level}} + \left( \frac{\sigma^2_{\text{item-level}}}{p} \right)} \right) \]

and the below equation was used for calculating person-level (i.e., level 2) reliability estimates:

\[ \alpha_{\text{person-level}} = \left( \frac{\sigma^2_{\text{person-level}}}{\sigma^2_{\text{person-level}} + \left( \frac{\sigma^2_{\text{day-level}}}{n} \right) + \left( \frac{\sigma^2_{\text{item-level}}}{p*n} \right)} \right) \]

where \( \sigma^2 \) is the variance at each level of analysis, \( p \) is the number of items in each scale, and \( n \) is the number of days of the study.

**Covariance structures.** Although limited criteria exist for how to select the best covariance structure in MLM, it is important to acknowledge that the covariance structure can
affect interpretation of the random effects. Because the unstructured covariance matrix is the most liberal of structures (in that it does not impose any constraints on the model, allowing every variance and covariance term to be different), it was used as the default structure in all MLM analyses (Kincaid, 2005).

**Example equations by study aim.** Example equations and statistical methods for each aim and corresponding hypotheses are presented below. (For simplification of presentation, simplified equations are presented; specific measures of each construct and covariates are not included. For the specific measures of the constructs in the below equation, refer to the “Measures” section and Table 1.)

**Aim 1.** To investigate the degree to which neuroticism predicts average frequency of DPEs and DNEs assessed across a 14-day daily diary study.

*Hypothesis 1.1.* Neuroticism will be associated positively with average frequency of DNEs, using the below example random intercept equation:

\[
\text{Level 1: DNE}_{ij} = \beta_0j + \epsilon_{ij} \\
\text{Level 2: } \beta_0j = \gamma_{00} + \gamma_{01}(\text{Neuroticism}_j) + \epsilon_{0j}
\]

At Level 1, DNE frequency (DNE\(_{ij}\)) for person \(j\) on day \(i\) was modeled as a function of a person-specific intercept (\(\beta_0j\)) and a person-day-specific residual (\(\epsilon_{ij}\)), or the difference between DNE frequency for person \(j\) on day \(i\) and their person-specific mean across days. At Level 2, the DNE intercept (\(\beta_0j\)) was modeled as a function of the average frequency of DNEs across people and days (\(\gamma_{00}\); i.e., the grand mean), the between-person association between neuroticism and average DNE frequency (\(\gamma_{01}\)), and between-person residuals (\(\epsilon_{0j}\)), or the difference between average DNE frequency for person \(j\) and the grand mean.
Hypothesis 1.2. Neuroticism will be associated negatively with average frequency of DPEs (see Hypothesis 1.1. for a similar random intercept equation).

**Aim 2.** To examine the cross-level moderation effects of neuroticism on the associations between DPEs and DNEs and daily PA and NA assessed across a 14-day daily diary study. Both random intercepts and random slopes were specified due to the assumption that both the average levels of the outcome (i.e., intercept) and the associations between daily experiences and affect (i.e., slopes) would vary across people. Previous work suggests that all coefficients (i.e., both intercepts and slopes) should be specified as random if random effects can be estimated reliably (Nezlek, 2012). Although including only random intercepts is more parsimonious than including both random intercepts and random slopes, because all random slopes in Aim 2 were significant, they were retained in order to accurately model variation in associations between daily experiences and affect across people.

Significant interaction effects were examined by comparing the simple slope estimates at higher (i.e., +1 SD) and lower (i.e., -1 SD) levels of neuroticism, in accordance with methods for estimating cross-level interactions from Alguinis and colleagues (2013) and Preacher and colleagues (2006). Simple slopes were plotted at each conditional value of the moderator using an R utility (see Preacher, Curran, & Bauer, 2003; Preacher et al., 2006). Region of significance testing was used to determine the specific values of the moderator at which the slope between the predictor and the outcome transitioned from non-significance to significance.

Hypothesis 2.1. Neuroticism will moderate the association between DNE frequency and daily NA; specifically, days with a greater number of DNEs than one’s individual average will be more strongly positively associated with daily NA for those with higher neuroticism than for those with lower neuroticism, using the below random intercept and random slope equations:
Level 1: Negative affect$_{ij}$ = $\beta_{0j} + \beta_{1j}(\text{DNE}) + r_{ij}$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Neuroticism}_j) + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Neuroticism}_j) + u_{1j}$

At Level 1, negative affect (Negative affect$_{ij}$) for person $j$ on day $i$ was modeled as a function of a person-specific intercept ($\beta_{0j}$), a person-day-specific slope ($\beta_{1j}$), or the association between DNEs and negative affect on days when an individual reports more or fewer DNEs relative to their average across days, and a person-specific residual ($r_{ij}$), or the difference between negative affect for person $j$ on day $i$ and their person-specific mean across days. At Level 2, the negative affect intercept ($\beta_{0j}$) was modeled as a function of average negative affect across people and days ($\gamma_{00}$; i.e., the grand mean), the between-person association between neuroticism and average frequency of DNEs ($\gamma_{01}$), and between-person residuals ($u_{0j}$), or the difference between average negative affect for person $j$ and the grand mean (i.e., $\gamma_{00}$). The within-person DNE slope ($\beta_{1j}$) was modeled as a function of the average within-person association between DNE and negative affect across persons ($\gamma_{10}$), the cross-level interaction of within-person centered DNE and between-person centered neuroticism ($\gamma_{11}$), and between-person residuals ($u_{1j}$), or the difference between person $j$’s slope (between DNE and negative affect) and the average slope across people and days (i.e., $\gamma_{10}$).

**Hypothesis 2.2.** Neuroticism will moderate the association between DNE frequency and daily PA; specifically, days with a greater number of DNEs than one’s individual average will be more strongly negatively associated with daily PA for those with higher neuroticism than for those with lower neuroticism (see Hypothesis 2.1. for a similar random intercept and random slope equation).
Hypothesis 2.3. Neuroticism will moderate the association between DPE frequency and daily NA; specifically, days with a greater number of DPEs than one’s individual average will be less strongly negatively associated with daily NA for those with higher neuroticism than for those with lower neuroticism (see Hypothesis 2.1. for a similar random intercept and random slope equation).

Hypothesis 2.4. Neuroticism will moderate the association between DPE frequency and daily PA; specifically, days with a greater number of DPEs than one’s individual average will be less strongly positively associated with daily PA for those with higher neuroticism than for those with lower neuroticism (see Hypothesis 2.1. for a similar random intercept and random slope equation).

Aim 3. To investigate the mediating role of both average daily PA and NA in the association between neuroticism and nightly sleep quality assessed across a 14-day daily diary study. To examine MLM mediation effects, one can employ the same basic framework for testing mediation effects in non-MLM models (Baron & Kenny, 1986; Card, 2012; Fairchild & MacKinnon, 2009; MacKinnon, 2008; Zhang, Zyphur, & Preacher, 2009): Step 1) estimate the effect of the predictor on the outcome, Step 2) estimate the effect of the predictor on the mediator, Step 3) estimate the effect of the predictor on the outcome, controlling for the mediator, and Step 4) examine the joint significance (i.e., the product term, or indirect effect) of the effects in Step 2 and Step 3. If the predictor is at level 2, but the mediator and outcome are at level 1, the mediator must be aggregated to the level 2 unit to avoid conflation of the level 1 and level 2 effects when examining the indirect effect (Zhang et al., 2009). Therefore, Step 1 (i.e., Hypothesis 3.3.) and Step 3 (i.e., Hypotheses 3.4. and 3.5.) utilized intercept-only MLMs and
Step 2 (i.e., Hypotheses 3.1. and 3.2.) utilized single-level OLS regression with neuroticism predicting the person-mean of the mediator.

Most contemporary mediation experts agree that Step 1 (often called the “total” or “direct” effect) does not need to be significant and that generating confidence intervals around the joint significance of the effects in Steps 2 and 3 is sufficient to demonstrate statistical mediation (MacKinnon, Lockwood, Hoffman et al., 2002; Rucker, Preacher, Tormala et al., 2011). Therefore, if Step 2 and Step 3 were significant, in accordance with recommendations from Bauer and colleagues (2006), the product term (i.e., the “indirect” effect) was computed using the Monte Carlo method for assessing mediation (MCMAM) via an R utility (see Bauer et al., 2006; Selig & Preacher, 2008). MCMAM repeatedly randomly samples from the joint distributions of the estimates and variances in Step 2 and Step 3 to produce 95% confidence intervals (CIs) of the mediated (i.e., indirect) effect. Studies have shown MCMAM to perform equally as well as comparable techniques such as bootstrapping (Selig et al., 2008). Random samples were generated 20,000 times, and 95% CIs were examined for each mediation effect; CIs that did not include 0 were considered significant mediation effects.

Hypothesis 3.1. Neuroticism will be associated positively with average daily NA using the below single-level OLS regression equation:

\[
\text{Level 2: Negative affect}_j = \gamma_{00} + \gamma_{01}(\text{Neuroticism}_j) + u_{0j}
\]

At Level 2, negative affect (Negative affect\(_j\)) was modeled as a function of average negative affect for the sample across people and days (\(\gamma_{00}\)), the between-person association between neuroticism and negative affect (\(\gamma_{01}\)), and between-person residuals (\(u_{0j}\)), or the difference between neuroticism for person \(j\) and the grand mean.
Hypothesis 3.2. Neuroticism will be associated negatively with average daily PA (see Hypothesis 3.1. for a similar single-level OLS regression equation).

Hypothesis 3.3. Neuroticism will be associated negatively with nightly sleep quality (see Hypothesis 1.1. for a similar random intercept equation).

Hypothesis 3.4. Daily negative affect will mediate the association between neuroticism and nightly sleep quality. Specifically, neuroticism will be associated positively with average daily NA (Hypothesis 3.1), and daily NA will be associated negatively with nightly sleep quality after controlling for neuroticism (see below random intercept equation). The product term of $\gamma_{01}$ from Hypothesis 3.1 and $\gamma_{02}$ from Hypothesis 3.4. will be significant. Together, these expected results would support the hypothesis that average daily NA will mediate the relationship between neuroticism and nightly sleep quality:

Level 1: $\text{Sleep quality}_{ij} = \beta_{0j} + r_{ij}$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Neuroticism}_j) + \gamma_{02}(\text{Negative affect}_i) + u_{0j}$

At Level 1, Sleep quality ($\text{Sleep quality}_{ij}$) for person $j$ on day $i$ was modeled as a function of a person-specific intercept ($\beta_{0j}$) and a within-person residual ($r_{ij}$), or the difference between sleep quality for person $j$ on day $i$ and their person-specific mean across days. At Level 2, the person-specific mean ($\beta_{0j}$) was modeled as a function of an intercept (the coefficient $\gamma_{00}$ represents the average sleep quality for the sample across people and days, i.e., the grand mean), neuroticism ($\gamma_{01}$), average (i.e., person-mean) negative affect ($\gamma_{02}$), and between-person residuals ($u_{0j}$), or the difference between average sleep quality for person $j$ and the grand mean.

Hypothesis 3.5. Daily PA will mediate the association between neuroticism and nightly sleep quality. Specifically, neuroticism will be associated negatively with average PA (Hypothesis 3.2.), and average daily PA will be associated positively with nightly sleep quality.
after controlling for neuroticism (see Hypothesis 3.4. for a similar equation). The product term of \( \gamma_{01} \) from Hypothesis 3.2. and \( \gamma_{02} \) from Hypothesis 3.5. will be significant (see Hypotheses 3.1. and 3.4. for similar equations). Together, these expected results would support the hypothesis that average daily PA will mediate the relationship between neuroticism and nightly sleep quality.
<table>
<thead>
<tr>
<th>Construct</th>
<th>Domain(s)</th>
<th>Specific measures</th>
<th>Abbreviation</th>
<th>Measurement Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroticism</td>
<td>--</td>
<td>Neuroticism subscale of the Eysenck Personality Questionnaire-Brief Version (Sato, 2005) and Mini-International Personality Item Pool (Donnellan et al., 2006)</td>
<td>--</td>
<td>Cross-sectional; at baseline questionnaire</td>
</tr>
<tr>
<td>Daily positive and negative experiences</td>
<td>Frequency</td>
<td>Adapted from the Uplifts and Hassles Scale (DeLongis et al., 1988)</td>
<td>DPE; DNE</td>
<td>Daily; at the bedtime survey for 14 days</td>
</tr>
<tr>
<td>Daily positive and negative affect</td>
<td>Frequency</td>
<td>Selected items from the Positive and Negative Affect Schedule (Watson et al., 1988) and the Profile of Mood States (McNair et al., 1981)</td>
<td>NA; PA</td>
<td>Daily; at the bedtime survey for 14 days</td>
</tr>
<tr>
<td>Nightly sleep quality</td>
<td>Objective</td>
<td>Actiwatch Spectrum™ actigraphy device for assessment of time awake after sleep onset, sleep efficiency, and sleep latency</td>
<td>WASO; SE; SL</td>
<td>Daily; continuously for 14 days</td>
</tr>
<tr>
<td></td>
<td>Subjective</td>
<td>PROMIS™ Sleep Disturbances and Sleep-Related Impairment Scales (Yu et al., 2012)</td>
<td>SD; SI</td>
<td>Daily; at the waking (SD) and bedtime (SI) surveys for 14 days</td>
</tr>
</tbody>
</table>
Table 2. Participant Characteristics

<table>
<thead>
<tr>
<th></th>
<th>M or n</th>
<th>SD or %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>25.33</td>
<td>3.98</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>47%</td>
</tr>
<tr>
<td>Female</td>
<td>32</td>
<td>53%</td>
</tr>
<tr>
<td><strong>Race</strong></td>
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<td></td>
</tr>
<tr>
<td>White</td>
<td>50</td>
<td>83%</td>
</tr>
<tr>
<td>Black or African-American</td>
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<td>2%</td>
</tr>
<tr>
<td>Asian or Indian</td>
<td>6</td>
<td>10%</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>5%</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
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<td></td>
</tr>
<tr>
<td>Hispanic or Latino</td>
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<td>8%</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>55</td>
<td>92%</td>
</tr>
<tr>
<td><strong>Annual Household Income</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than $4,999</td>
<td>4</td>
<td>7%</td>
</tr>
<tr>
<td>$5,000-$19,999</td>
<td>12</td>
<td>20%</td>
</tr>
<tr>
<td>$20,000-$39,999</td>
<td>14</td>
<td>23%</td>
</tr>
<tr>
<td>$40,000-$59,999</td>
<td>14</td>
<td>23%</td>
</tr>
<tr>
<td>$60,000-$79,999</td>
<td>4</td>
<td>7%</td>
</tr>
<tr>
<td>$80,000-$99,999</td>
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<td>2%</td>
</tr>
<tr>
<td>$100,000-$149,999</td>
<td>2</td>
<td>3%</td>
</tr>
<tr>
<td>$150,000 or above</td>
<td>3</td>
<td>5%</td>
</tr>
<tr>
<td>Chose not to answer</td>
<td>6</td>
<td>10%</td>
</tr>
<tr>
<td><strong>Phone Type</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I-phone</td>
<td>39</td>
<td>65%</td>
</tr>
<tr>
<td>Android</td>
<td>20</td>
<td>33%</td>
</tr>
<tr>
<td>Windows</td>
<td>1</td>
<td>2%</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school diploma/GED (General Education Diploma)</td>
<td>3</td>
<td>5%</td>
</tr>
<tr>
<td>Some college or 2-year associate degree</td>
<td>12</td>
<td>20%</td>
</tr>
<tr>
<td>College graduate (4- or 5-year program)</td>
<td>30</td>
<td>50%</td>
</tr>
<tr>
<td>Master’s degree (or other post-graduate training)</td>
<td>14</td>
<td>23%</td>
</tr>
<tr>
<td>Doctoral degree (PhD., MD, EdD, DVM, DDS, JD, etc.)</td>
<td>1</td>
<td>2%</td>
</tr>
<tr>
<td><strong>Body Mass Index</strong></td>
<td>25.91</td>
<td>7.05</td>
</tr>
<tr>
<td><strong>Depressed Mood</strong></td>
<td>10.91</td>
<td>8.85</td>
</tr>
</tbody>
</table>

Note. Npersons = 60.
Figure 2. Flow diagram of study protocol and participants.
CHAPTER 4: Results

Preliminary Results

Compliance rates and missing data. Participants completed an average of 13.63 (SD = 1.87) out of a possible 14 waking surveys and 13.22 (SD = 1.44) out of a possible 14 bedtime surveys. In sum, participants completed an average of 26.85 out of a possible 28 total daily surveys, for a daily diary compliance rate of approximately 96%. Data collection resulted in between 775 and 780 person-days (out of a possible 840; i.e., 60 people x 14 days) for bedtime survey measures, 812 person-days for waking measures, and 790 valid person-days for actigraphy measures (see Table 3). No data were missing at the item- or person-level for the neuroticism measure. Approximately 3-8% of data were missing at the person-day-level for the daily negative experiences (DNE), daily positive experiences (DPE), negative affect (NA), positive affect (PA), subjective sleep disturbances (SD), and subjective sleep impairment (SI) measures, and 6% of data were missing for objective time awake after sleep onset (WASO), objective sleep efficiency (SE), and objective sleep latency (SL) at the person-day-level (see Table 3).

Cohort effects. Analysis of variance tests (ANOVA) tests for neuroticism and average scores (i.e., scores aggregated across the 14-day study period) of primary study variables revealed no significant differences between the seven cohorts in neuroticism scores, average DNE or DPE frequency, or average NA, PA, SD, SI, or WASO, SE, or SL scores. Dummy-coded cohort, (0 = belongs to that cohort, 1 = does not belong to that cohort) was added as a covariate in all analyses to account for any other potential differences in cohort characteristics; Cohort 7 was removed from all analyses to avoid collinearity, or overestimation due to inclusion of all dummy coding possibilities. Cohort was included as a covariate because of a priori
theoretical considerations (i.e., that those in earlier cohorts may exhibit greater enthusiasm to participate or other response biases, as compared to later cohorts) and to be conservative. However, cohort was not consistently associated with any primary study outcome. Thus, results were also run without covarying for cohort; the general pattern of results did not change when cohort was excluded as a covariate.

**Scale reliabilities.** Chronbach’s alpha ($\alpha$) calculations revealed that the primary neuroticism measure (Eysenck Personality Questionnaire-Brief Version; Sato, 2005) demonstrated high reliability ($\alpha = 0.94$) and that the secondary neuroticism measure (Mini-International Personality Item Pool; Donnellan et al., 2006) demonstrated acceptable reliability ($\alpha = 0.65$). The two measures of neuroticism were highly correlated ($r = 0.81, p < .0001$). Only the primary neuroticism measure was used in analyses, due to its higher levels of internal consistency. All daily measures with multiple items (i.e., DPE, DNE, NA, PA, SI, and SD) demonstrated high (i.e., $\alpha > 0.90$) level 2 (i.e., person-level) reliability, with lower levels of level 1 (i.e., day-level) reliability (see Table 3). Low level 1 reliability is expected when measures are meant to capture daily fluctuations in states.

**Intraclass correlation coefficients.** The daily diary design captured significant ($ps < .001$) variation in DPEs, DNEs, PA, NA, SI, SD, WASO, SE at both the between- and the within-person levels (see Table 3). For example, the ICC for SI was 0.30, indicating 30% of the total variation in SI could be attributed to between-person variation (i.e., variation from person to person) and 70% could be attributed to within-person variation (i.e., variation from day to day within each person).
Aim 1 Results

**Descriptive results.** The primary neuroticism measure demonstrated a fairly normal distribution; participants reported average neuroticism scores of 27.33 ± 11.42 (skewness = 0.63, kurtosis = -0.50; see Figure 3) with all scores ranging from 12 to 54, and 68% of scores (i.e., scores ± 1 SD from the mean) ranging from 16 to 39. Across people and days, participants reported at least one DPE 97.82% of days, with an average of 7.60 ± 3.52 DPEs per day (skewness = 0.20, kurtosis = -0.28). The two most commonly reported DPEs were “friends” (n = 560) and “amount of free time” (n = 507). Participants reported at least one DNE 89.63% of days, with an average of 3.90 ± 3.02 DNEs per day (skewness = 1.32, kurtosis = 2.68). The two most commonly reported DNEs were “work, work load (school or other type of work), or job security” (n = 359) and “meeting deadlines or goals” (n = 336). DPE and DNE frequency were positively correlated at the between-person level (r = 0.41, p < .01), but not at the within-person level (r = -0.04, p = 0.32; see Table 4). At the between-person level, neuroticism was significantly correlated with average frequency of DPEs and was marginally significantly correlated with average frequency of DNEs (see Table 3).

**Hypothesis 1.1. Neuroticism will be associated positively with frequency of DNEs.** Neuroticism was not associated with frequency of DNEs, controlling for age, gender, race, ethnicity, education, income, and cohort (β = 0.03, SE = 0.02, p = 0.26; see Table 5, which also lists all covariates included).

**Hypothesis 1.2. Neuroticism will be associated negatively with frequency of DPEs.** Neuroticism was associated negatively with frequency of DPEs, controlling for the same covariates (β = -0.08, SE = 0.03, p = 0.01; see Table 5). For every one-unit increase in neuroticism, the frequency of DPEs decreased by 0.08 units.
Aim 2 Results

**Descriptive results.** Across people and days, participants reported average daily PA scores of 55.57 ± 22.68 (skewness = -0.16, kurtosis = -0.74), and average daily NA scores of 14.70 ± 14.72 (skewness = 1.73, kurtosis = 3.08). PA and NA were negatively correlated at the between-person level \( r = -0.26, p < .05 \) and at the within-person level \( r = -0.47, p < .0001 \); see Table 4.

**Hypothesis 2.1. Neuroticism will moderate the association between DNEs and daily negative affect.** Neuroticism moderated the association between DNE frequency and daily NA, controlling for age, gender, race, ethnicity, education, income, and cohort (\( \beta = 0.04, SE = 0.02, p < 0.01 \); see Table 6, which also lists all covariates included). At neuroticism scores 1 SD above the mean, on days with average frequencies of DNEs, participants reported daily NA levels of 26.51 (simple intercept: \( \beta = 26.51, SE = 7.34, z = 3.61, p = 0.0003; \) see Figure 4), and at neuroticism scores 1 SD below the mean, on days with average frequencies of DNEs, participants reported daily NA levels of 14.61 (simple intercept: \( \beta = 14.61, SE = 7.39, z = 1.98, p = 0.05; \) see Figure 4). At neuroticism scores 1 SD above the mean, days with one more DNE than an individual’s average were associated with a 1.89-unit increase in daily NA (simple slope: \( \beta = 1.89, SE = 0.25, z = 7.45, p < .0001; \) see Figure 4), and at neuroticism scores 1 SD below the mean, days with one more DNE than an individual’s average were associated with a 0.92-unit increase in daily NA (simple slope: \( \beta = 0.92, SE = 0.27, z = 3.44, p < .001; \) see Figure 4).

**Hypothesis 2.2. Neuroticism will moderate the association between DNEs and daily positive affect.** Neuroticism did not moderate the association between DNE frequency and daily PA, controlling for the same covariates (\( \beta = 0.01, SE = 0.03, p = 0.79; \) see Table 6).
Hypothesis 2.3. Neuroticism will moderate the association between DPEs and negative affect. Neuroticism moderated the association between DPE frequency and daily NA, controlling for the same covariates ($\beta = -0.03, SE = 0.01, p < 0.05$; see Table 6). At neuroticism scores 1 SD above the mean, on days with average frequencies of DPEs, participants reported daily NA levels of 25.66 (simple intercept: $\beta = 25.66, SE = 7.22, z = 3.55, p = 0.0004$; see Figure 5), and at neuroticism scores 1 SD below the mean, on days with average frequencies of DPEs, participants reported daily NA levels of 13.11 (simple intercept: $\beta = 13.11, SE = 7.27, z = 3.55, 1.80, p = 0.07$; see Figure 5). At neuroticism scores 1 SD above the mean, days with one more DPE than an individual’s average were associated with a 1.24-unit decrease in daily NA (simple slope: $\beta = -1.24, SE = 0.24, z = -5.08, p < 0.0001$; see Figure 5), and at neuroticism scores 1 SD below the mean, days with one more DPE than an individual’s average were associated with a 0.55-unit decrease in daily NA (simple slope: $\beta = -0.55, SE = 0.24, z = -2.30, p = 0.02$; see Figure 5).

Hypothesis 2.4. Neuroticism will moderate the association between DPEs and daily positive affect. Neuroticism moderated the association between DPE frequency and daily PA, controlling for the same covariates ($\beta = 0.05, SE = 0.03, p < 0.05$; see Table 6). At neuroticism scores 1 SD above the mean, on days with average frequencies of DPEs, participants reported daily PA levels of 30.28 (simple intercept: $\beta = 30.28, SE = 12.66, z = 2.39, p = 0.02$; see Figure 6), and at neuroticism scores 1 SD below the mean, on days with average frequencies of DPEs, participants reported daily PA levels of 43.37 (simple intercept: $\beta = 43.37, SE = 12.75, z = 3.40, p = 0.0007$; see Figure 6). At neuroticism scores 1 SD above the mean, days with one more DPE than an individual’s average were associated with a 2.53-unit increase in daily PA (simple slope: $\beta = 2.53, SE = 0.42, z = 6.10, p < 0.0001$; see Figure 6), and at neuroticism scores 1 SD below the
mean, days with one more DPE than an individual’s average were associated with a 1.40-unit increase in daily PA (simple slope: $\beta = 1.40$, $SE = 0.41$, $z = 3.45$, $p = .0006$; see Figure 6).

**Aim 3 Results**

**Descriptive results.** Across all people and days, participants reported average subjective SI scores of $1.76 \pm 0.67$ (skewness = 1.24, kurtosis = 1.17) and average subjective SD scores of $2.26 \pm 0.81$ (skewness = 0.65, kurtosis = -0.09). SI and SD were positively correlated at the between-person level ($r = 0.77$, $p < .0001$) and at the within-person level ($r = 0.35$, $p < .001$; See Table 4). Across all people and days, participants reported being in bed for an average of $8.03 \pm 1.63$ hours. Participants reported sleep durations of <5 hours 2.46% of nights, 5-6 hours 16.63% of nights, 6-7 hours 27.46% of nights, 7-8 hours 34.98% of nights, 8-9 hours 15.64% of nights, 9-10 hours 2.34% of nights, and >10 hours 0.49% of nights. Participants reported SL’s of 0-5 minutes 20.07% of nights, 6-10 minutes 27.34% of nights, 11-15 minutes 22.41% of nights, 16-30 minutes 15.89% of nights, 31-59 minutes 8% of nights, 1-2 hours 5.17% of nights, and greater than 2 hours 1.11% of nights. Participants reported napping 13.11% ($n = 102$) of all days.

Across all people and days, actigraphy revealed that participants had average rest intervals (i.e., time in bed) of $7.94 \pm 0.59$ hours (476.25 ± 35.54 minutes; skewness = -0.21, kurtosis = -0.02), and average sleep durations (i.e., time actually asleep) of $6.36 \pm 2.24$ hours (381.59 ± 134.69 minutes; skewness = -1.19, kurtosis = 1.36). Participants napped at least once 11.77% ($n = 93$) of all days, for an average of $86.83 \pm 36.93$ minutes per nap. Participants spent an average of $37.35 \pm 12.00$ minutes awake after sleep onset (WASO; skewness = 0.46, kurtosis = -0.09), had average SL’s of $4.99 \pm 4.89$ minutes (skewness = 3.16, kurtosis = 14.65), and had average SE’s of $88.06 \pm 3.73$ (skewness = -0.63, kurtosis = 0.02).
At the between-person level, WASO was correlated with SE ($r = -0.83, p < .0001$) but not SL, and SE was correlated with SL ($r = -0.28, p < .05$; see Table 4). At the within-person level, WASO was correlated with SE ($r = -0.27, p < .0001$) and marginally correlated with SL ($r = 0.07, p < .07$; see Table 4). SE was correlated with SL ($r = -0.36, p < .001$; see Table 4). At the between-person level, subjective SI was correlated with objective WASO ($r = 0.31, p < .05$), SE ($r = -0.27, p < .05$), and SL ($r = 0.26, p < .05$), and subjective SD was correlated with objective WASO ($r = 0.34, p < .01$) and SE ($r = -0.25, p < .05$), but not SL (see Table 4). At the within-person level, subjective SI and SD were not correlated with objective WASO, SE, or SL (see Table 4).

On an exploratory post-hoc basis, the baseline characteristics of BMI and depressed mood were examined in relation to neuroticism and the sleep quality variables. Neuroticism was significantly correlated with depressed mood ($r = 0.81, p < .0001$) but not with BMI. At the between-person level, subjective SI and SD were correlated with depressed mood ($r = 0.61, p < .0001$; $r = 0.49, p < .0001$, respectively), but objective WASO, SE, and SL were not. At the between-person level, only objective SL, and not objective SE or WASO or subjective SD or SI, was correlated with BMI ($r = 0.26, p < .05$).

**Hypothesis 3.1. Neuroticism will be associated positively with average daily negative affect.** Neuroticism was associated positively with average daily NA, controlling for age, gender, race, ethnicity, education, income, and cohort ($\beta = 0.52, SE = 0.14, p < .0001$; see Table 7, which also lists all covariates included). For every one-unit increase in neuroticism, average daily NA increased by 0.52 units.

**Hypothesis 3.2. Neuroticism will be associated negatively with average daily positive affect.** Neuroticism was associated negatively with average daily PA, controlling for the same
covariates ($\beta = -0.56, SE = 0.23, p < .01$; see Table 7). For every one-unit increase in neuroticism, average daily PA decreased by 0.56 units.

**Hypothesis 3.3. Neuroticism will be associated negatively with (better) sleep quality.** Neuroticism was associated positively with subjective SI ($\beta = 0.02, SE = 0.004, p < .0001$) and subjective SD ($\beta = 0.02, SE = 0.005, p < .001$), but not with objective SL ($\beta = 0.04, SE = 0.05, p = 0.49$), objective WASO ($\beta = 0.13, SE = 0.15, p = 0.37$), or objective SE ($\beta = -0.03, SE = 0.04, p = 0.42$; see Table 8), controlling for the same covariates. For every one-unit increase in neuroticism, subjective SI and SD each increased by 0.02 units.

**Hypothesis 3.4. Daily negative affect will help explain the association between neuroticism and sleep quality.** Average daily NA was associated positively with subjective SI ($\beta = 0.01, SE = 0.004, p = .007$), subjective SD ($\beta = 0.02, SE = 0.005, p = 0.002$), objective SL ($\beta = 0.13, SE = 0.06, p = 0.05$), and was associated negatively with objective SE ($\beta = -0.09, SE = 0.05, p = 0.05$), but not with objective WASO ($\beta = 0.22, SE = 0.16, p = 0.17$; see Table 9), controlling for the same covariates (in addition to neuroticism). For every one-unit increase in NA, subjective SI increased by 0.01 units, subjective SD increased by 0.02 units, objective SL increased by 0.13 units, and objective SE decreased by 0.09 units. Using the Monte Carlo Method for Assessing Mediation (MCMAM) to assess the joint significance of the indirect effect, NA mediated the effects of neuroticism on subjective SD, 95% CI [0.003, 0.02], subjective SI, 95% CI [0.003, 0.01], objective SL, 95% CI [0.001, 0.15], and objective SE, 95% CI [-0.11, -0.001], but not on objective WASO.

**Hypothesis 3.5. Daily positive affect will help explain the association between neuroticism and nightly sleep quality.** Average daily PA was marginally negatively associated with subjective SI ($\beta = -0.004, SE = 0.002, p = 0.06$) and significantly negatively associated with
subjective SD ($\beta = -0.006, SE = 0.003, p = 0.04$), but not with objective SL ($\beta = 0.02, SE = 0.04$, $p = 0.62$), objective WASO ($\beta = 0.08, SE = 0.09, p = 0.39$), or objective SE ($\beta = -0.0004, SE = 0.03, p = 0.99$; see Table 10), controlling for the same covariates (in addition to neuroticism). For every one-unit increase in PA, subjective SI decreased by 0.004 units and subjective SD decreased by 0.006 units. PA did not mediate the effects of neuroticism on subjective SD or SI, or on objective SL, WASO, or SE.
Table 3. Descriptive Statistics for Primary Measures

<table>
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<tr>
<th></th>
<th>N</th>
<th># of miss. obs.</th>
<th>% of miss. data</th>
<th>M</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
<th>Person-level reliability</th>
<th>Day-level reliability</th>
<th>Between-person variance</th>
<th>Within-person variance</th>
<th>ICC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroticism</td>
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</tr>
<tr>
<td>NA</td>
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<td>65</td>
<td>7.7%</td>
<td>14.70</td>
<td>14.72</td>
<td>1.73</td>
<td>3.08</td>
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<td>141.81 (27.16)***</td>
<td>74.72 (3.95)***</td>
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<tr>
<td>PA</td>
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<td>7.6%</td>
<td>55.57</td>
<td>22.68</td>
<td>-0.16</td>
<td>-0.74</td>
<td>0.92</td>
<td>0.47</td>
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<td>186.07 (9.83)***</td>
<td>0.64</td>
</tr>
<tr>
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<td>50</td>
<td>6.0%</td>
<td>3.90</td>
<td>3.02</td>
<td>1.32</td>
<td>2.68</td>
<td>0.93</td>
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<td>4.02 (0.21)***</td>
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</tr>
<tr>
<td>DPE</td>
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<td>7.60</td>
<td>3.52</td>
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<td>0.93</td>
<td>0.18</td>
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<td>5.60 (0.30)***</td>
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<tr>
<td>SI</td>
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<td>7.4%</td>
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<td>1.24</td>
<td>1.17</td>
<td>0.93</td>
<td>0.02</td>
<td>0.14 (0.03)***</td>
<td>0.31 (0.02)***</td>
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<tr>
<td>SD</td>
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<td>28</td>
<td>3.3%</td>
<td>2.26</td>
<td>0.81</td>
<td>0.65</td>
<td>-0.09</td>
<td>0.92</td>
<td>0.02</td>
<td>0.18 (0.04)***</td>
<td>0.47 (0.02)***</td>
<td>0.27</td>
</tr>
<tr>
<td>WASO</td>
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<td>50</td>
<td>6.0%</td>
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<td>18.96</td>
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<td>2.59</td>
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<td>249.75 (13.03)***</td>
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</tr>
<tr>
<td>SE</td>
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<td>0.68</td>
<td>11.76 (2.52)***</td>
<td>25.55 (1.33)***</td>
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<tr>
<td>SL</td>
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<td>6.0%</td>
<td>4.99</td>
<td>4.89</td>
<td>3.16</td>
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<td>0.85</td>
<td>16.67 (4.34)***</td>
<td>92.27 (4.81)***</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Note. Missing data for daily measures (i.e., number of missing observations and percentage of missing data) were calculated out of a possible 840 observations (i.e., 14 days x 60 participants). WASO, SE, and SL consisted of only one item each; therefore, person-level reliability reflects the proportion of between-person variance to total variance and day-level reliability reflects the proportion of within-person variance to total variance. NA = negative affect; PA = positive affect; DNE = daily negative experience frequency; DPE = daily negative experience frequency; SI = subjective sleep impairment; SD = subjective sleep disturbance; WASO = objective time awake after sleep onset; SE = objective sleep efficiency; SL = objective sleep latency. ICC = intraclass correlation coefficient (i.e., proportion of between-person variance to total variance).

***p < .001.
Table 4. Between-Person and Within-Person Correlations for Primary Measures and Covariates

<table>
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<tbody>
<tr>
<td>1. Neuroticism</td>
<td>1</td>
<td>0.23†</td>
<td>-0.36**</td>
<td>0.58***</td>
<td>0.59***</td>
<td>0.48***</td>
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<td>0.08</td>
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<td>-0.10</td>
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<td>2. DPE</td>
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<td>0.41**</td>
<td>0.57***</td>
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<td>-0.25*</td>
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<td>3. DNE</td>
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<td>0.43***</td>
<td>0.32**</td>
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<td>0.16</td>
<td>0.06</td>
<td>-0.13</td>
<td>-0.04</td>
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<td>4. PA</td>
<td>0.32***</td>
<td>-0.26***</td>
<td>1</td>
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<td>-0.42***</td>
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<td>5. NA</td>
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<td>0.53***</td>
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<td>6. SI</td>
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<td>0.77***</td>
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<td>0.31*</td>
<td>0.26*</td>
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<td>0.04</td>
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<td>0.11***</td>
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<td>8. WASO</td>
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<td>-0.02</td>
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<td>0.03</td>
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<td>0.02</td>
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<td>0.01</td>
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<td>0.27*</td>
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<td>0.04</td>
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<td>13. Race</td>
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<td>--</td>
</tr>
<tr>
<td>17. Cohort</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
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<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

Note. N_{persons} = 60 for between-person correlations, N_{person-days} = 834 for within-person (i.e., person-mean centered) correlations. Within-person correlations are in the lower diagonal; between-person correlations are in the upper diagonal. NA = negative affect; PA = positive affect; DNE frequency = daily negative experience frequency; DPE frequency = daily negative experience frequency; SI = subjective sleep impairment; SD = subjective sleep disturbance; WASO = objective time awake after sleep onset; SE = objective sleep efficiency; SL = objective sleep latency; Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 1 (first cohort) to 7 (last cohort).

†p < .07. *p < .05. **p < .01. ***p < .001.
Table 5. Effects of Neuroticism on Average Frequency of DNEs (Hypotheses 1.1.) and DPEs (Hypothesis 1.2.)

<table>
<thead>
<tr>
<th></th>
<th>H. 1.1.</th>
<th>H. 1.2.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary variables (fixed effects)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>2.49 (1.52)</td>
<td>2.80 (1.72)</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.03 (0.02)</td>
<td>-0.08 (0.03)**</td>
</tr>
<tr>
<td><strong>Covariates (fixed effects)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.17 (0.08)*</td>
<td>0.13 (0.09)</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.46 (0.54)</td>
<td>-0.02 (0.62)</td>
</tr>
<tr>
<td>Race</td>
<td>1.44 (0.86)</td>
<td>2.89 (0.98)**</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>2.15 (1.04)*</td>
<td>3.00 (1.18)**</td>
</tr>
<tr>
<td>Education</td>
<td>-0.43 (1.42)</td>
<td>1.64 (1.60)</td>
</tr>
<tr>
<td>Income</td>
<td>-0.44 (0.63)</td>
<td>0.11 (0.71)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.76 (1.07)</td>
<td>0.48 (1.21)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>1.36 (1.19)</td>
<td>1.23 (1.34)</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>2.51 (1.17)</td>
<td>1.35 (1.32)</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>0.31 (1.15)</td>
<td>0.08 (1.30)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>0.83 (1.18)</td>
<td>0.69 (1.33)</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>1.35 (1.09)</td>
<td>0.15 (1.23)</td>
</tr>
<tr>
<td><strong>Variance components</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 1 residual variance</td>
<td>4.02 (0.21)***</td>
<td>5.60 (0.29)***</td>
</tr>
<tr>
<td>Level 2 variance in intercept</td>
<td>3.26 (0.66)***</td>
<td>4.14 (0.83)***</td>
</tr>
<tr>
<td><strong>Model statistics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 2 pseudo $R^2$</td>
<td>0.34</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Note. $N_{\text{persons}} = 60$, $N_{\text{days}} = 780$. Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort.

*p < .05. **p < .01. ***p < .001.
<table>
<thead>
<tr>
<th>Primary variables (fixed effects)</th>
<th>$H. \ 2.1.$</th>
<th>$H. \ 2.2.$</th>
<th>$H. \ 2.3.$</th>
<th>$H. \ 2.4.$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>20.56 (7.24)**</td>
<td>36.52 (12.64)**</td>
<td>19.38 (7.12)**</td>
<td>36.83 (12.49)**</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.52 (0.12)***</td>
<td>-0.57 (0.20)***</td>
<td>0.55 (0.12)***</td>
<td>-0.57 (0.20)***</td>
</tr>
<tr>
<td>DPE, $\gamma_{10}$</td>
<td>--</td>
<td>--</td>
<td>-0.89 (0.17)***</td>
<td>1.97 (0.29)***</td>
</tr>
<tr>
<td>Neuroticism x DPE, $\gamma_{11}$</td>
<td>1.41 (0.19)***</td>
<td>-2.06 (0.34)***</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Neuroticism x DNE, $\gamma_{11}$</td>
<td>0.04 (0.02)***</td>
<td>0.01 (0.03)</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Covariates (fixed effects)</th>
<th>$H. \ 2.1.$</th>
<th>$H. \ 2.2.$</th>
<th>$H. \ 2.3.$</th>
<th>$H. \ 2.4.$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.33 (0.37)</td>
<td>-0.05 (0.64)</td>
<td>-0.29 (0.36)</td>
<td>-0.05 (0.64)</td>
</tr>
<tr>
<td>Gender</td>
<td>0.19 (2.60)</td>
<td>-3.26 (4.48)</td>
<td>-0.43 (2.54)</td>
<td>-3.63 (4.48)</td>
</tr>
<tr>
<td>Race</td>
<td>4.24 (4.13)</td>
<td>21.17 (7.11)**</td>
<td>3.11 (4.05)</td>
<td>21.00 (7.12)**</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-0.84 (4.98)</td>
<td>23.90 (8.58)**</td>
<td>-1.20 (4.87)</td>
<td>23.88 (8.58)**</td>
</tr>
<tr>
<td>Education</td>
<td>-14.72 (6.76)*</td>
<td>-6.98 (11.66)</td>
<td>-11.29 (6.65)</td>
<td>-6.97 (11.66)</td>
</tr>
<tr>
<td>Income</td>
<td>2.34 (3.00)</td>
<td>4.51 (5.17)</td>
<td>2.54 (2.93)</td>
<td>4.59 (5.17)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.19 (5.10)</td>
<td>11.69 (8.79)</td>
<td>0.39 (4.98)</td>
<td>11.25 (8.79)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>6.23 (5.66)</td>
<td>12.20 (9.77)</td>
<td>5.16 (5.57)</td>
<td>11.42 (9.77)</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>9.02 (5.67)</td>
<td>9.53 (9.60)</td>
<td>7.16 (5.47)</td>
<td>10.53 (9.60)</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>5.19 (5.48)</td>
<td>4.88 (9.46)</td>
<td>4.74 (5.39)</td>
<td>4.77 (9.46)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>2.03 (5.63)</td>
<td>4.08 (9.72)</td>
<td>1.06 (5.53)</td>
<td>4.11 (9.71)</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>5.09 (5.19)</td>
<td>7.20 (8.95)</td>
<td>2.95 (5.11)</td>
<td>7.58 (8.95)</td>
</tr>
</tbody>
</table>

**Variance components**

| Level 1 residual variance                | 64.04 (3.46)***| 161.48 (8.80)***| 66.54 (3.66)***| 152.82 (8.40)***|
| Level 2 variance in intercept            | 76.49 (14.99)***| 230.89 (44.91)***| 77.67 (15.46)***| 231.44 (44.86)***|
| Level 2 variance in DPE slope            | --          | --          | 0.63 (0.31)*| 2.36 (0.90)***|
| Level 2 variance in DNE slope            | 0.61 (0.30)*| 2.66 (1.08)**| --          | --          |

**Model statistics**

| Level 1 pseudo $R^2$                     | 0.14          | 0.14          | 0.10          | 0.18          |
| Level 2 pseudo $R^2$                     | 0.45          | 0.30          | 0.44          | 0.30          |
Note. $N_{\text{persons}} = 60$, $N_{\text{days}} = 780$. Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort.

*p < .05. **p < .01. ***p < .001.
Table 7. Effects of Neuroticism on Average Daily NA (Hypothesis 3.1.) and Average Daily PA (Hypothesis 3.2.)

<table>
<thead>
<tr>
<th></th>
<th>$H_{3.1.}$ NA</th>
<th>$H_{3.2.}$ PA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>20.53 (8.33)*</td>
<td>35.84 (14.33)***</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.52 (0.14)***</td>
<td>-0.56 (0.23)**</td>
</tr>
<tr>
<td><strong>Covariates</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.31 (0.42)</td>
<td>-0.12 (0.73)</td>
</tr>
<tr>
<td>Gender</td>
<td>0.18 (2.99)</td>
<td>-3.77 (5.14)</td>
</tr>
<tr>
<td>Race</td>
<td>4.26 (4.75)</td>
<td>21.02 (8.17)*</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-0.86 (5.73)</td>
<td>24.09 (9.86)*</td>
</tr>
<tr>
<td>Education</td>
<td>-14.66 (7.78)</td>
<td>-6.19 (13.38)</td>
</tr>
<tr>
<td>Income</td>
<td>2.39 (3.45)</td>
<td>4.73 (5.93)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.23 (5.87)</td>
<td>12.25 (10.09)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>6.15 (6.52)</td>
<td>11.71 (11.21)</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>9.03 (6.41)</td>
<td>10.40 (11.02)</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>5.21 (6.31)</td>
<td>5.17 (10.85)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>1.84 (6.48)</td>
<td>4.34 (11.15)</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>5.19 (5.96)</td>
<td>7.62 (10.25)</td>
</tr>
<tr>
<td><strong>Model statistics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.28</td>
<td>0.11</td>
</tr>
</tbody>
</table>

Note. $N_{persons} = 60$. Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort.

*p < .05. **p < .01. ***p < .001.
### Table 8. Effects of Neuroticism on Subjective and Objective Sleep Quality (Hypothesis 3.3.)

<table>
<thead>
<tr>
<th></th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary variables (fixed effects)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>2.11 (0.23)***</td>
<td>2.49 (0.30)***</td>
<td>36.23 (9.04)***</td>
<td>87.39 (2.65)***</td>
<td>8.37 (3.40)*</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.02 (0.004)***</td>
<td>0.02 (0.005)***</td>
<td>0.13 (0.15)</td>
<td>-0.03 (0.04)</td>
<td>0.04 (0.05)</td>
</tr>
<tr>
<td><strong>Covariates (fixed effects)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.01 (0.01)</td>
<td>-0.005 (0.02)</td>
<td>0.24 (0.46)</td>
<td>-0.16 (0.13)</td>
<td>0.44 (0.17)**</td>
</tr>
<tr>
<td>Gender</td>
<td>0.01 (0.08)</td>
<td>0.09 (0.11)</td>
<td>-5.57 (3.23)</td>
<td>2.17 (0.95)*</td>
<td>-0.08 (1.21)</td>
</tr>
<tr>
<td>Race</td>
<td>-0.06 (0.13)</td>
<td>0.01 (0.17)</td>
<td>0.11 (5.12)</td>
<td>0.75 (1.50)</td>
<td>-2.19 (1.91)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-0.41 (0.16)*</td>
<td>-0.43 (0.20)*</td>
<td>-1.08 (6.18)</td>
<td>-0.04 (1.81)</td>
<td>-3.78 (2.30)</td>
</tr>
<tr>
<td>Education</td>
<td>-0.20 (0.22)</td>
<td>-0.06 (0.28)</td>
<td>2.56 (8.45)</td>
<td>-1.71 (2.45)</td>
<td>2.45 (3.19)</td>
</tr>
<tr>
<td>Income</td>
<td>-0.02 (0.10)</td>
<td>-0.07 (0.12)</td>
<td>-1.29 (3.73)</td>
<td>-0.57 (1.09)</td>
<td>-0.71 (1.39)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.12 (0.16)</td>
<td>-0.13 (0.21)</td>
<td>1.27 (6.33)</td>
<td>1.92 (1.86)</td>
<td>-3.86 (2.36)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>-0.25 (0.18)</td>
<td>-0.16 (0.23)</td>
<td>4.15 (7.03)</td>
<td>1.02 (2.06)</td>
<td>-6.37 (2.62)</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>-0.003 (0.18)</td>
<td>-0.25 (0.23)</td>
<td>2.36 (6.91)</td>
<td>0.93 (2.03)</td>
<td>-5.26 (2.58)</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>-0.05 (0.18)</td>
<td>-0.11 (0.22)</td>
<td>-0.06 (6.81)</td>
<td>0.96 (1.99)</td>
<td>-1.22 (2.54)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>0.003 (0.18)</td>
<td>-0.42 (0.23)</td>
<td>-1.11 (6.99)</td>
<td>2.03 (2.05)</td>
<td>-4.19 (2.60)</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>-0.13 (0.17)</td>
<td>-0.27 (0.21)</td>
<td>7.66 (6.43)</td>
<td>-2.19 (1.88)</td>
<td>-3.41 (2.40)</td>
</tr>
<tr>
<td><strong>Variance components</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 1 residual variance</td>
<td>0.31 (0.02)***</td>
<td>0.47 (0.02)***</td>
<td>249.76 (13.03)***</td>
<td>25.56 (1.33)***</td>
<td>92.25 (4.81)***</td>
</tr>
<tr>
<td>Level 2 variance in intercept</td>
<td>0.06 (0.02)***</td>
<td>0.10 (0.03)***</td>
<td>107.08 (23.18)***</td>
<td>8.89 (1.99)***</td>
<td>10.65 (3.23)***</td>
</tr>
<tr>
<td><strong>Model statistics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 2 pseudo R²</td>
<td>0.56</td>
<td>0.43</td>
<td>0.12</td>
<td>0.24</td>
<td>0.36</td>
</tr>
</tbody>
</table>

Note. $N_{\text{persons}} = 60$, $N_{\text{days}} = 794$. Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort; SI = subjective sleep impairment; SD = subjective sleep disturbance; WASO = objective time awake after sleep onset; SE = objective sleep efficiency; SL = objective sleep latency.

$\dagger p < .07$. *$p < .05$. **$p < .01$. ***$p < .001$. 

---

59
Table 9. Mediated Effects of Average Daily NA in the Association between Neuroticism and Sleep Quality (Hypothesis 3.4.)

<table>
<thead>
<tr>
<th>Fixed effects</th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>2.02 (0.21)**</td>
<td>2.41 (0.27)**</td>
<td>34.89 (9.11)**</td>
<td>87.94 (2.64)**</td>
<td>8.20 (3.60)*</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.01 (0.004)**</td>
<td>0.01 (0.01)$\dagger$</td>
<td>-0.0001 (0.17)</td>
<td>0.02 (0.05)</td>
<td>-0.03 (0.07)</td>
</tr>
<tr>
<td>NA person-mean, $\gamma_{02}$</td>
<td><strong>0.01 (0.004)</strong></td>
<td><strong>0.02 (0.005)</strong></td>
<td>0.22 (0.16)</td>
<td><strong>-0.09 (0.05)</strong></td>
<td><strong>0.13 (0.06)</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Covariates (fixed effects)</th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.004 (0.01)</td>
<td>-0.002 (0.01)</td>
<td>0.34 (0.46)</td>
<td>-0.18 (0.13)</td>
<td>0.50 (0.18)**</td>
</tr>
<tr>
<td>Gender</td>
<td>0.01 (0.07)</td>
<td>0.09 (0.10)</td>
<td>-6.00 (3.22)$\dagger$</td>
<td>2.57 (0.93)**</td>
<td>-0.40 (1.26)</td>
</tr>
<tr>
<td>Race</td>
<td>-0.13 (0.12)</td>
<td>-0.09 (0.16)</td>
<td>-0.34 (5.17)</td>
<td>0.71 (1.50)</td>
<td>-2.81 (2.02)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-0.40 (0.14)**</td>
<td>-0.43 (0.19)$*$</td>
<td>-0.93 (6.17)</td>
<td>-0.43 (1.79)</td>
<td>-3.90 (2.40)</td>
</tr>
<tr>
<td>Education</td>
<td>0.02 (0.20)</td>
<td>0.22 (0.26)</td>
<td>6.10 (8.78)</td>
<td>-2.84 (2.55)</td>
<td>4.67 (3.47)</td>
</tr>
<tr>
<td>Income</td>
<td>-0.05 (0.09)</td>
<td>-0.13 (0.11)</td>
<td>-2.31 (3.75)</td>
<td>-0.38 (1.09)</td>
<td>-1.11 (1.42)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.11 (0.14)</td>
<td>-0.14 (0.19)</td>
<td>1.67 (6.33)</td>
<td>1.86 (1.83)</td>
<td>-4.31 (2.47)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>-0.34 (0.16)$*$</td>
<td>-0.29 (0.21)</td>
<td>2.57 (7.09)</td>
<td>1.30 (2.06)</td>
<td>-7.97 (2.76)**</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>-0.14 (0.16)</td>
<td>-0.42 (0.21)</td>
<td>0.53 (7.05)</td>
<td>1.43 (2.04)</td>
<td>-6.85 (2.75)$*$</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>-0.12 (0.16)</td>
<td>-0.21 (0.21)</td>
<td>-2.03 (6.87)</td>
<td>1.30 (1.99)</td>
<td>-2.27 (2.69)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>-0.02 (0.16)</td>
<td>-0.47 (0.21)$*$</td>
<td>-2.40 (6.99)</td>
<td>2.29 (2.62)</td>
<td>-5.32 (2.72)$\dagger$</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>-0.20 (0.15)</td>
<td>-0.43 (0.20)</td>
<td>7.22 (6.53)</td>
<td>-2.19 (1.89)</td>
<td>-4.30 (2.57)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variance components</th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1 residual variance</td>
<td>0.31 (0.02)**</td>
<td>0.48 (0.02)**</td>
<td>228.38 (12.32)**</td>
<td>24.61 (1.33)**</td>
<td>93.57 (3.52)**</td>
</tr>
<tr>
<td>Level 2 variance in intercept</td>
<td>0.04 (0.01)**</td>
<td>0.08 (0.02)**</td>
<td>107.06 (23.33)**</td>
<td>8.55 (1.96)**</td>
<td>11.57 (3.52)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model statistics</th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 2 pseudo $R^2$</td>
<td>0.70</td>
<td>0.54</td>
<td>0.12</td>
<td>0.27</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Note. $N_{\text{persons}} = 60$, $N_{\text{days}} = 794$. **Bold** values represent significant mediation effects (based on examination of joint significance test 95% confidence intervals). Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort; SI = subjective sleep impairment; SD = subjective sleep disturbance; WASO = objective time awake after sleep onset; SE = objective sleep efficiency; SL = objective sleep latency.

$\dagger p < .07$. $* p < .05$. **$p < .01$. ***$p < .001$. 

60
Table 10. Mediated Effects of Average Daily PA in the Association between Neuroticism and Sleep Quality (Hypothesis 3.5.)

<table>
<thead>
<tr>
<th></th>
<th>SI</th>
<th>SD</th>
<th>WASO</th>
<th>SE</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept, $\gamma_{00}$</td>
<td>2.03 (0.23)**</td>
<td>2.40 (0.29)**</td>
<td>37.81 (9.32)**</td>
<td>87.36 (2.77)**</td>
<td>9.36 (3.75)*</td>
</tr>
<tr>
<td>Neuroticism, $\gamma_{01}$</td>
<td>0.02 (0.004)**</td>
<td>0.01 (0.005)**</td>
<td>0.16 (0.16)</td>
<td>-0.03 (0.05)</td>
<td>0.04 (0.06)</td>
</tr>
<tr>
<td>PA person-mean, $\gamma_{02}$</td>
<td>-0.004 (0.002)†</td>
<td>-0.01 (0.003)*</td>
<td>0.08 (0.09)</td>
<td>-0.0004 (0.03)</td>
<td>0.02 (0.04)</td>
</tr>
<tr>
<td>Covariates (fixed effects)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Age</td>
<td>-0.01 (0.01)</td>
<td>-0.01 (0.01)</td>
<td>0.27 (0.46)</td>
<td>-0.15 (0.14)</td>
<td>0.46 (0.18)*</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.003 (0.08)</td>
<td>0.07 (0.10)</td>
<td>-5.65 (3.28)</td>
<td>2.55 (0.97)†</td>
<td>-0.29 (1.31)</td>
</tr>
<tr>
<td>Race</td>
<td>0.03 (0.14)</td>
<td>0.10 (0.17)</td>
<td>-1.10 (5.53)</td>
<td>0.34 (1.64)</td>
<td>-2.70 (2.20)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-0.30 (0.16)</td>
<td>-0.30 (0.21)</td>
<td>-3.06 (6.63)</td>
<td>-0.35 (1.97)</td>
<td>-4.42 (2.64)</td>
</tr>
<tr>
<td>Education</td>
<td>-0.23 (0.21)</td>
<td>-0.07 (0.27)</td>
<td>3.31 (8.56)</td>
<td>-1.46 (2.54)</td>
<td>2.91 (3.46)</td>
</tr>
<tr>
<td>Income</td>
<td>0.003 (0.09)</td>
<td>-0.07 (0.12)</td>
<td>-2.15 (3.79)</td>
<td>-0.60 (1.13)</td>
<td>-0.90 (1.52)</td>
</tr>
<tr>
<td>Cohort 1</td>
<td>-0.06 (0.16)</td>
<td>-0.07 (0.21)</td>
<td>-0.89 (6.49)</td>
<td>1.90 (1.93)</td>
<td>-4.60 (2.59)</td>
</tr>
<tr>
<td>Cohort 2</td>
<td>-0.20 (0.18)</td>
<td>-0.12 (0.22)</td>
<td>2.99 (7.18)</td>
<td>0.75 (2.13)</td>
<td>-7.44 (2.86)**</td>
</tr>
<tr>
<td>Cohort 3</td>
<td>0.04 (0.17)</td>
<td>-0.22 (0.22)</td>
<td>1.71 (7.04)</td>
<td>0.61 (2.09)</td>
<td>-5.93 (2.81)*</td>
</tr>
<tr>
<td>Cohort 4</td>
<td>-0.03 (0.17)</td>
<td>-0.10 (0.22)</td>
<td>-1.25 (6.90)</td>
<td>0.82 (2.05)</td>
<td>-1.68 (2.76)</td>
</tr>
<tr>
<td>Cohort 5</td>
<td>0.02 (0.18)</td>
<td>-0.42 (0.22)†</td>
<td>-2.31 (7.06)</td>
<td>2.12 (2.09)</td>
<td>-5.15 (2.81)†</td>
</tr>
<tr>
<td>Cohort 6</td>
<td>-0.10 (0.16)</td>
<td>-0.32 (0.21)</td>
<td>7.79 (6.57)</td>
<td>-2.66 (1.95)</td>
<td>-3.81 (2.65)</td>
</tr>
<tr>
<td>Variance components</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 1 residual</td>
<td>0.31 (0.02)**</td>
<td>0.47 (0.03)**</td>
<td>228.37 (12.32)**</td>
<td>24.61 (1.33)**</td>
<td>93.54 (5.04)**</td>
</tr>
<tr>
<td>variance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 2 variance in</td>
<td>0.05 (0.01)**</td>
<td>0.09 (0.02)**</td>
<td>109.62 (23.80)**</td>
<td>9.28 (2.10)**</td>
<td>12.90 (3.75)**</td>
</tr>
<tr>
<td>intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model statistics</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Level 2 pseudo R²</td>
<td>0.60</td>
<td>0.47</td>
<td>0.10</td>
<td>0.21</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Note. $N_{persons} = 60$, $N_{days} = 794$. Gender: 0 = male, 1 = female; Race: 0 = not white, 1 = white; Ethnicity: 0 = not Hispanic or Latino, 1 = Hispanic or Latino; Education: 0 = no college, 1 = at least some college; Income: 0 = below $59,999, 1 = above $60,000; Cohort: 0 = does not belong to that cohort, 1 = belongs to that cohort; SI = subjective sleep impairment; SD = subjective sleep disturbance; WASO = objective time awake after sleep onset; SE = objective sleep efficiency; SL = objective sleep latency.

†$p < .07$. *$p < .05$. **$p < .01$. ***$p < .001$. 
Figure 3. Primary neuroticism measure distribution. Possible scores ranged from 12 to 60.

$N_{\text{persons}} = 60$. 
Figure 4. The moderated effect of neuroticism on the association between daily negative experience frequency (DNE) and negative affect (NA; Hypothesis 2.1.). Equations represent the simple intercept and the simple slope for each group (i.e., ±1 SD of the mean of neuroticism). Daily negative experience frequency is person-mean centered.

\[ y = 26.51^{***} + 1.89^{***}x \]

\[ y = 14.61^{*} + 0.92^{***}x \]

† \( p < .07 \). * \( p < .05 \). ** \( p < .01 \). *** \( p < .001 \).
Figure 5. The moderated effect of neuroticism on the association between daily positive experience frequency (DPE) and negative affect (NA; Hypothesis 2.3.). Equations represent the simple intercept and the simple slope for each group (i.e., ±1 SD of the mean of neuroticism). Daily positive experience frequency is person-mean centered.

\( y = 25.66^{***} - 1.24^{***}x \)

\( y = 13.11^{†} - 0.55^{*}x \)

\( ^{†}p < .07. \ *p < .05. \ **p < .01. \ ***p < .001. \)
Figure 6. The moderated effect of neuroticism on the association between daily positive experience frequency (DPE) and positive affect (PA; Hypothesis 2.4.). Equations represent the simple intercept and the simple slope for each group (i.e., ±1 SD of the mean of neuroticism). Daily positive experience frequency is person-mean centered.

\[ y = 43.37^{***} + 1.40^{***}x \]

\[ y = 30.28^* + 2.53^{***}x \]

\[ ^\dagger p < .07. * p < .05. ** p < .01. *** p < .001. \]
CHAPTER 5: Discussion

This study was the first to examine the relationships between neuroticism, daily positive and negative experiences, and subjective and objective sleep quality using an ecological, repeated measures design. In Aim 1, it was hypothesized that neuroticism would predict average frequencies of daily negative experiences (DNEs) and daily positive experiences (DPEs). In Aim 2, it was hypothesized that neuroticism would moderate associations between frequencies of DNEs and DPEs and daily negative affect (NA) and positive affect (PA). Finally, in Aim 3, it was hypothesized that average daily NA and PA would mediate associations between neuroticism and subjective sleep impairment (SI), subjective sleep disturbances (SD), objective time awake after sleep onset (WASO), objective sleep efficiency (SE), and objective sleep latency (SL). The findings from each aim, as well as overall study implications, directions for future research, and limitations are discussed below.

Aim 1 Discussion of Findings

Contrary to the expected hypothesis, higher neuroticism was not associated with a greater frequency of DNEs; however, as expected, it was associated with a lower frequency of DPEs. Results from the current investigation suggest that neuroticism may be more impactful for decreasing reported exposure to DPEs than it is for increasing reported exposure to DNEs. These findings are in contrast with previous studies demonstrating that neuroticism predicts a greater frequency of exposure to DNEs (Bolger et al., 1991; Bolger et al., 1995; David et al., 1997; Suls & Martin, 2005), and not a lower frequency of exposure to DPEs (David et al., 1997; Zautra et al., 2005). In the current study, both DPEs and DNEs were reported more frequently than in previous studies (e.g., David et al., 1997; Zautra et al., 2005). It is possible that differences in measurement techniques of DNEs and DPEs or in sample characteristics (e.g., age differences)
may explain the discrepancy of findings between past research and the current study. For example, previous work consistently has shown that older adults are more effective at coping with stress and report more DPEs and fewer DNEs on average (Almeida, 2005; Charles, Luong, Almeida et al., 2010; Stawski et al., 2008; Whitehead & Bergeman, 2014); therefore, it is plausible that interactions exist between age and neuroticism on reported average frequency of DPEs and DNEs. Because the current sample was relatively young, future research should examine if the same results hold across more diverse samples, in order to understand for whom and under what circumstances neuroticism is associated with DPEs and DNEs.

It is also important to note that in the current study, average frequency of reported DPEs and DNEs were only moderately correlated across people and days. This finding is consistent with other studies (e.g., Zautra et al., 2005) and suggests that DNEs and DPEs reflect at least partially distinct concepts that both warrant measurement. This is a contention supported by a growing body of literature seeking to acknowledge the distinct importance of DPEs (e.g., Sin et al., 2015a; Zautra et al., 2005). The majority of previous studies of neuroticism and daily experiences have failed to integrate the assessment of DPEs into their methodology (e.g., Bolger et al., 1991; Bolger et al., 1995; Gunthert et al., 1999). Although further replication of the observed association between neuroticism and decreased DPE frequency is necessary, results from the current study suggest that the integration of DPEs adds a distinct and important contribution to the understanding of how neuroticism influences exposure to different types of daily experiences.

**Aim 2 Discussion of Findings**

In Aim 2, results indicated that neuroticism significantly moderated within-person associations between DNEs and NA (Hypothesis 2.1.), DPEs and NA (Hypothesis 2.3.), and
DPEs and PA (Hypothesis 2.4.), but not the association between DNEs and PA (Hypothesis 2.2.). However, not all of the significant moderation findings were of the hypothesized strength or direction at conditional values of neuroticism (i.e., ±1 SD from the mean). Possible explanations and implications for each of these findings in Aim 2 are discussed below.

**Hypotheses 2.1. and 2.2.** Contrary to the expected hypothesis, days with more DNEs than one’s individual average were not associated with lower PA for those higher (vs. lower) in neuroticism: Both those higher and lower in neuroticism experienced comparable differences in PA on days with more DNEs than their individual average. However, as hypothesized, days with more DNEs than one’s individual average were associated with greater differences in NA for those higher (vs. lower) in neuroticism: Days with one more negative experience than an individual’s average were associated with approximately double the difference in NA for those higher (vs. lower) in neuroticism.

These findings support a large body of literature demonstrating that higher neuroticism predicts greater NA on days with more DNEs than average (Bolger et al., 1991; Bolger et al., 1995; Gunaydin, Selcuk, & Ong, 2016; Mroczek & Almeida, 2004). Interestingly, compared to those lower in neuroticism, not only did those higher in neuroticism report greater NA on “atypical” days (i.e., days with more DNEs than their individual average), but they also reported greater NA on their “typical” days (i.e., days with average frequencies of DNEs). Together, these findings reinforce the idea that those higher in neuroticism tend to persist in more heightened negative emotional states, regardless of external stimuli, as well as exhibit larger differences in NA on particularly stressful days, relative to those lower in neuroticism (see Suls et al., 2005).

Bolger and Zuckerman (1995) propose that greater NA on days with more DNEs than average may be due to poorer coping choices (e.g., rumination, worry, or learned helplessness).
and/or reduced coping effectiveness (i.e., failure of the chosen coping strategy to reduce NA).

The heightened NA on days with more DNEs than average of those higher in neuroticism also may be explained by the “contrast avoidance model of worry” (Newman & Llera, 2011). Among those with generalized anxiety disorder (GAD; a disorder that shows some overlap with neuroticism), Newman and Llera (2011) propose that a trigger for heightened NA may be a detection of a mismatch between one’s expectation of the situation and the actual experience of the situation (i.e., “a contrast,” such as days with more DNEs than usual). As a defense mechanism, those with GAD may persist in states of elevated worry and NA to avoid these contrasts. This theory may help explain why, compared to those lower in neuroticism, those higher in neuroticism experience more NA even on days with average frequencies of DNEs, as well as greater differences in NA on days when they experience more DNEs than their typical amount.

**Hypotheses 2.3. and 2.4.** For both those higher and lower in neuroticism, days with more DPEs than one’s individual average were significantly associated with lower NA and higher PA. However, contrary to expectations, both of these associations were stronger for those higher in neuroticism: Days with one more positive experience than an individual’s average were associated with approximately twice the difference in reported NA and PA for those higher (vs. lower) in neuroticism. In addition, even on days with average frequencies of DPEs, compared to those lower in neuroticism, those higher in neuroticism reported approximately twice the levels of NA and half the levels of PA. Although neuroticism still had a significant effect on the association between DPEs and PA at lower values of neuroticism, this effect was weaker than at higher values of neuroticism. These findings further support the contention that individuals lower in neuroticism may be more effective at regulating affect, regardless of external stimuli.
Together with the results from Hypothesis 2.1., findings demonstrate that even on their “typical” days, those high in neuroticism report more NA and less PA, as compared to those lower in neuroticism on their “typical” days. Days with more DPEs than average appear to disrupt both the high NA and low PA set point of individuals higher in neuroticism.

The significant moderation effect of neuroticism on the within-person association between DPEs and PA is in contrast with findings from Zautra and colleagues (2005), who found that neuroticism did not moderate this same association in their sample. The lack of concordance in findings between the present study and Zautra and colleagues (2005) may be due to differences in sample characteristics, however. Participants in the latter study were mostly midlife to older adults all with rheumatoid arthritis, and both age and chronic pain are factors that have been shown to alter daily experiences and affect regulation (as reviewed in Hamilton, Karoly, & Kitzman, 2004; Kessler & Staudinger, 2009; Lumley, Cohen, Borszcz et al., 2011). Although no study has examined the role of neuroticism in NA in the context of DPEs, some work has shown that those with unipolar major or minor depression (both of which show some overlap with neuroticism) report greater reductions in NA following exposure to DPEs compared to healthy controls (Bylsma, Taylor-Clift, & Rottenberg, 2011; Peeters, Nicolson, Berkhof et al., 2003). Together with the current study, these findings imply that greater affective lability in the context of DPEs actually may reflect maladaptive mental health.

Previous work has shown that greater NA lability predicts adverse outcomes, including increases in depressive symptoms (Whitehead et al., 2014) and inflammation (Sin, Graham-Engeland, Ong et al., 2015b). However, much less work has examined the role of neuroticism in associations between DPEs and NA or PA, or links between PA lability and health. One study showed that greater PA variability across time (irrespective of DPEs) was associated with lower
well-being and life satisfaction and greater depression and anxiety (Gruber, Kogan, Quoidbach et al., 2013); however, little work has investigated how changes in affect in the context of DPEs may confer heightened risk for adverse outcomes. A major strength of the current study was the ability to demonstrate that not only does higher neuroticism relate to greater differences in NA on days with more DNEs than average, but it also relates to greater differences in both PA and NA on days with more DPEs than average. Replication of these findings is needed in other samples, but PA lability may represent a distinct facet of neuroticism that is important for health.

It is important to note that the results in Aim 2 cannot definitively demonstrate affective “reactivity,” as no external stimuli were experimentally manipulated. By choosing to examine daily experiences in situ, the present research gained ecological validity and perspective on real-life associations but sacrificed experimental manipulation and inferences of causality. Therefore, it is possible that the observed associations between daily experiences and differences in affect were caused by some unmeasured variable, or that heightened affective lability simply reflects a trait-like characteristic that is correlated with neuroticism. Indeed, previous work has shown that those higher in neuroticism are more labile in their NA across time (Suls et al., 2005). However, it is also plausible that days with more DPEs and DNEs than average may causally lead to greater increases or decreases in PA and NA for those higher in neuroticism. Unfortunately, without experimental manipulation of external stimuli, it is impossible to untangle the true causal nature of these relationships. Because manipulation of common daily experiences is likely difficult in naturalistic settings, to strengthen the current research, future work should examine whether laboratory-based manipulation of common daily experiences leads to greater affective reactivity for those higher in neuroticism.
**Aim 3 Discussion of Findings**

In Aim 3, results demonstrated that neuroticism was associated with greater average NA and lower average PA (Hypotheses 3.1. and 3.2., respectively), as well as poorer subjective but not objective sleep quality (Hypothesis 3.3.). NA mediated the relationships between neuroticism and subjective sleep quality, as well as some measures of objective sleep quality (Hypothesis 3.4.). PA did not mediate the relationships between neuroticism and subjective or objective sleep quality (Hypothesis 3.5.). Possible explanations and implications for each of these results are discussed below.

**Hypotheses 3.1., 3.2., and 3.3.** As expected, higher neuroticism was associated with greater average daily NA and lower average daily PA, but contrary to expectations, neuroticism only was associated with poorer subjective and not objective sleep quality. These results suggest that neuroticism plays an important role in the reported experience of both positive and negative affect, as well as the perception of sleep quality in daily life.

These findings are in alignment with cross-sectional and daily diary studies showing that neuroticism is associated with greater NA (Bolger et al., 1991; Bolger et al., 1995; Rusting & Larsen, 1997; Suls et al., 2005), lower PA (Shiota, Keltner, & John, 2006; Zautra et al., 2005), as well more reported sleep problems (Calkins et al., 2013; Cheng et al., 2012; Duggan et al., 2014; Gurtman et al., 2014). However, previous research only has examined associations between neuroticism and affect, affect and sleep, or neuroticism and sleep, whereas the current research examined all of these constructs to elucidate potential pathways between these variables. Furthermore, measures of affect and sleep were assessed in daily life and were aggregated across time, increasing external validity and reliability in comparison to more global self-report measures or measures taken in laboratory settings (see Shiffman et al., 2008). Finally, by
incorporating more objective measures of sleep quality, the current study was able to bolster findings of the majority of previous work that included solely self-report measures. There are several possible explanations for why neuroticism predicted subjective but not objective sleep quality in the current study, which are discussed in more detail below.

First, previous work has shown evidence of response biases in the reporting of sleep among those individuals with insomnia and depression; individuals with these mental health conditions tend to have an attentional bias toward negative stimuli and often overestimate actual sleep problems (Argyropoulos, Hicks, Nash et al., 2003; Matousek, Cervena, Zavesicka et al., 2004; Vgontzas, Fernandez-Mendoza, Liao et al., 2013). In the current sample, among those individuals with neuroticism scores greater than or equal to the mean \( (n = 25) \), 68% met clinical criteria for subclinical or moderate to severe insomnia (as measured by the Insomnia Severity Index; Bastien, Vallieres, & Morin, 2001), and 52% met criteria clinical for depression (as measured by the Center for Epidemiological Studies Depression Scale Revised; Radloff, 1977). By comparison, in those individuals with neuroticism scores below the mean \( (n = 35) \), only 17% met criteria for subclinical or moderate to severe insomnia and 3% met criteria for depression. This comorbidity with depressive, and particularly, with insomnia symptoms, may help explain why individuals higher in neuroticism in this sample tended to report impaired subjective sleep quality without experiencing corresponding impairments in objective sleep quality.

Second, previous work has shown that insomnia may exist with or without objective sleep disturbances (Fernandez-Mendoza et al., 2010; Vgontzas et al., 2013). Those with insomnia with objective sleep disturbances often exhibit increased cognitive-emotional and cortical arousal, heightened activation of the HPA and SAM-axes, elevated risk for impaired health outcomes, and premature mortality (Fernandez-Mendoza et al., 2010; Vgontzas et al.,...
In contrast, those with insomnia *without* objective sleep disturbances often exhibit only increased cognitive-emotional and cortical arousal and sleep misperception, but not adverse health outcomes or premature mortality (Fernandez-Mendoza et al., 2010; Vgontzas et al., 2013). It is possible that those individuals higher in neuroticism may be more likely to exhibit this latter phenotype of reported sleep disturbances without corresponding objective disturbances in sleep. However, these individuals may nonetheless be important to identify and treat, as they tend to demonstrate heightened risk for mental health comorbidities (as appears to be the case in the current sample).

Third, it is plausible that neuroticism only predicted subjective and not objective sleep quality because subjective and objective measures of sleep quality may reflect partially distinct constructs. In the current study, the subjective and objective measures of sleep quality were correlated only moderately at the between-person level. There is some evidence suggesting that subjective and objective sleep have different biological correlates (Jackowska, Ronaldson, Brown et al., 2016), and many studies have shown partial discrepancies between subjective and objective measures of sleep across diverse clinical and healthy samples (e.g., Argyropoulos et al., 2003; Chen, McHugh, Campbell et al., 2014; Jackowska, Dockray, Hendrickx et al., 2011; Jackowska et al., 2016; Matousek et al., 2004; McCrae et al., 2008). Furthermore, in the current study, the subjective sleep quality scales each included a wide breadth and depth of facets of sleep quality (e.g., sleep initiation, sleep maintenance, sleep-related daytime dysfunction, sleep quality), whereas the objective measures each assessed one specific facet of sleep. By doing so, the objective measures may have excluded the critical component of sleep quality important for neuroticism. Together, these observations raise the possibility that the subjective and objective measures of sleep quality used in this study may be partially discrete constructs and/or each
differentially impacted by personality and daily affect. Future work examining personality and sleep in daily life should continue to include both objective and subjective measures of sleep, as each may provide unique information about phenotypes of risk.

**Hypothesis 3.4. and Hypothesis 3.5.** As expected, both higher average daily NA and lower average daily PA were associated with poorer subjective sleep quality; however, contrary to expectations, only average daily NA was associated with poorer objective sleep quality (SE and SL, but not WASO). Although neuroticism was not directly associated with objective sleep quality (i.e., the “direct” effect; Hypothesis 3.3.), neuroticism was associated with higher average daily NA (Hypotheses 2.2.), and higher average daily NA predicted both poorer subjective sleep quality and objective sleep quality (SE and SL), controlling for neuroticism. Therefore, consistent with contemporary mediation frameworks which emphasize the joint significance of these latter two pathways and deemphasize the significance of direct effect (see MacKinnon et al., 2002; Rucker et al., 2011), average daily NA mediated the relationship between neuroticism and subjective sleep quality and some objective measures of sleep quality (SE and SL). These findings suggest that the tendency to report greater NA in daily life may be an important intervening variable explaining how neuroticism may lead to subjective and objective sleep impairment.

Other studies similarly have shown that both greater NA and lower PA are correlated with poorer sleep quality in healthy and clinical samples (Baglioni et al., 2010; McCrae et al., 2008; Steptoe et al., 2008). NA also has been shown to predict reduced objective sleep efficiency (Jackowska et al., 2016), and one study demonstrated that NA mediated the relationship between interpersonal conflict and self-reported sleep quality (Brissette et al., 2002). However, the current study was the first to examine both daily NA and PA as mediators of the link between
neuroticism and both subjective and objective sleep quality using a repeated measures daily diary design. As previously discussed, this approach enhances ecological validity, elucidating how transactions between neuroticism and responses to one’s everyday environment may trigger sleep disturbances. Measures of neuroticism, affect, and sleep also were assessed at different time points: Neuroticism was assessed at baseline, and for 14 days NA and PA were assessed nightly (reflecting back on the previous day), subjective sleep quality measures were assessed nightly or the next morning, and objective sleep quality was recorded continuously overnight via actigraphy. Although the study design is still only correlational and cannot determine that heightened NA caused impaired sleep, this measurement approach allows for partial temporal ordering of the psychological phenomena assessed, strengthening inferences from mediation analyses.

Overall, results from these mediation analyses are in alignment with the cognitive and hyperarousal models of insomnia, which assert that excessive negatively-toned cognitive activity during the day can trigger autonomic arousal that makes the act of falling asleep, staying asleep, and obtaining restorative sleep more difficult. Previous studies have shown that heightened average NA (as well as heightened average PA) is a risk factor for insomnia (as reviewed in Baglioni et al., 2010 and Kahn et al., 2013). In fact, emotional arousability has been suggested as one of the most defining characteristics of those vulnerable to insomnia (Fernandez-Mendoza et al., 2010). It is important to acknowledge that the observed relationship between NA and impaired sleep may be further mediated by other variables, such as health behaviors (such as substance use or physical activity), decreased connectivity between the pre-frontal cortex and amygdala (Gruber & Cassoff, 2014) and/or by components of immune system that have been shown to be important for both affect and sleep (e.g., pro-inflammatory cytokines interleukin 1-
beta and tumor-necrosis factor alpha; Kiecolt-Glaser et al., 2002; Riemann et al., 2010). Future work should explore these and other potential variables to better understand the psychophysiological processes by which NA may impair sleep.

**Implications and Future Directions**

In summary, the findings from the present research provide support for the differential exposure-reactivity theory as well as the hyperarousal and cognitive models of insomnia. Those higher in neuroticism reported fewer DPEs in daily life, as well as larger differences in NA on days with more DNEs than average and larger differences in PA and NA on days with more DPEs than average. Higher neuroticism also predicted greater average NA and lower average PA, as well as poorer average subjective but not objective sleep quality. The tendency to report greater average NA in daily life mediated the associations between neuroticism and impaired subjective and objective sleep quality. These results suggest that those higher in neuroticism may experience fewer positive events in daily life, may be less able to regulate affect in the face of changing daily events, and that their tendency to experience and/or report greater NA in daily life may lead to subjective and objective sleep impairment.

As previously discussed, given that there was no experimental manipulation of variables, it is imperative to recognize that the results of this study cannot determine that neuroticism or exposure to daily events caused changes in affect, or that affect caused an attenuation in the association between neuroticism and sleep quality. Therefore, future experimental work is needed to confirm the observed associations. However, results from the current investigation at least raise the possibility that increasing exposure to DPEs, improving affect regulation strategies, and decreasing average levels of NA may be potential strategies for improving mental health and sleep in those individuals higher in neuroticism. Previous work has shown that
frequent, minor, daily positive events, or “micro-moments” of positivity, can broaden attention and flexibility, increase PA, increase social resources, and be protective for mental health and physiological markers of health (Fredrickson, 1998, 2001; Fredrickson & Branigan, 2005; Sin et al., 2015a). There is some further evidence to suggest that across situations, relatively stable and low levels of NA, and relatively stable PA (regardless of mean levels) are both indicative of better psychological health (Gruber et al., 2013; Suls, Green, & Hillis, 1998).

Again, although experimental work is needed, results in Aim 3 suggest the possibility that efforts to decrease average NA also may help improve sleep quality for those high in neuroticism. In concordance with results from this study, evidence across studies suggests that lower levels of NA are associated with better sleep quality (as reviewed in Baglioni et al., 2010). More work is needed using rigorous randomized clinical trials, but cognitive-behavioral therapies acknowledging the interrelated nature of emotions and sleep may be more effective for addressing the specific concerns of individuals higher in neuroticism. For example, cognitive behavioral therapy for insomnia (CBT-I) has been shown to lead to both improved emotion regulation and sleep in some samples (as reviewed in Baglioni et al., 2010), and many CBT-I protocols already involve techniques to address intrusive emotions (e.g., through cognitive control strategies; Baglioni et al., 2010).

Furthermore, the observed associations between neuroticism and poorer subjective but not objective sleep quality could reflect an increased risk for co-morbid mental health complications (e.g., depression, anxiety, and insomnia). Over time, impaired health and premature mortality may result if worry over perceived sleep disturbances translates into actual chronic sleep disturbances. Individuals with sleep misperception may be important to identify and treat to help challenge maladaptive sleep-related beliefs, reduce hyperarousal through
relaxation techniques, and change maladaptive emotional patterns that may be driving perceived sleep impairment.

In the future, studies examining neuroticism in daily life should continue to incorporate measures of both DNE and DPE exposure and PA and NA, as the effects of neuroticism in this sample appear to more reliably predict associations between affect and DPEs than between affect and DNEs. It is unclear if this link between affect and exposure to DPEs may confer a differential risk for adverse health outcomes than does affect and exposure to DNEs, as little work to date has been done on this topic. Therefore, it would be ideal to examine DPE and DNE exposure and patterns of daily affect longitudinally in relation to neuroticism and more objective health outcomes (e.g., cardiometabolic disease incidence) or health-related biomarkers (e.g., heart rate variability, cortisol, or pro-inflammatory cytokines). This type of work would enable a better understanding of both who is most at risk for particular adverse health outcomes, as well as the distinct daily patterns of affect that may be driving particular health trajectories over time.

When examining neuroticism and sleep in daily life, future research should integrate the assessment of both objective and subjective measures of sleep quality to clarify phenotypes of risk and potential reporting biases in neuroticism (discussed in further detail in the Limitations section below). Self-reported psychological measures may be related to objective health markers, but because those higher in neuroticism also are more likely to report negative states (Costa et al., 1987), it cannot be assumed that self-reported measures are a proxy for objective health status. As was done in Aim 3 of the current study, integration of subjective and more objective measures of psychophysiological constructs is needed to help understand these potential response biases. Though it was beyond the scope of this dissertation, the examination of other variables likely related to personality, affect, and sleep quality – such as depression, relationship
quality, other health behaviors such as substance use and physical activity, or biomarkers of neuroendocrine-immune function – also may be useful areas for future research to inform more targeted intervention efforts. Longer term-prospective longitudinal or measurement burst study designs also could help determine if the observed links between neuroticism, affect, and impaired sleep in daily life predict later risk for adverse health outcomes. Finally, the examination of within-person associations between NA, PA, and subjective and objective measures of sleep, and cross-level interactions with neuroticism would help identify if days characterized by particular emotional patterns are more or less detrimental for sleep, and how neuroticism may modify these associations in daily life.

Overall, findings from the present research highlight the importance of studying individual differences in the role of daily experiences, affect, and sleep quality. Using daily diary methods, this study demonstrated how neuroticism manifests its influence in daily experiences, affect, and sleep quality more reliably than previous cross-sectional work. Results from the current study suggest that both daily experiences and individual predispositions, and the interactions between them, are important predictors of daily affect and sleep. Future work on personality and health should continue to include repeated, comprehensive measures of experiences and health behaviors in everyday life. Such research will continue to help identify those individuals who may be at risk for poor health outcomes, as well as identify the daily processes that have the most impact for health.

**Limitations**

**Sampling bias.** Some important methodological limitations must be acknowledged when drawing conclusions from the current study. Foremost, due to time and budget constraints, convenience sampling was utilized; therefore, participants in the current study were not
randomly selected to participate. This leads to sampling bias: Those individuals who participated
in the study may possess characteristics different than those who did not participate (e.g., higher
levels of conscientiousness). Furthermore, participants all were relatively young and
homogenous in terms of high levels of education. Both of these issues limit generalizability of
results, warranting replication of these findings using random sampling techniques to obtain a
more representative sample.

**Response bias in neuroticism.** Second, as previously mentioned, it is important to
acknowledge that there is a known response bias in neuroticism: Individuals high in neuroticism
are more likely to report negative states (Costa et al., 1987). It is plausible that those higher in
neuroticism are reporting more impaired states but not actually suffering negative
psychophysiological consequences, confounding the interpretation of the observed results and
threatening the internal validity of the study. However, as was demonstrated in Aim 3, subjective
reports (e.g., heightened reported NA) may still be important predictors of more objective markers
of health (e.g., actigraphy-determined sleep quality). Those higher in neuroticism actually may
be experiencing more impaired psychophysiological states and impaired health, particularly if
their tendency to report and notice problems leads to chronic worry and rumination over
perceived problems. Without more objective measures of health and environmental
characteristics, response biases cannot be fully remediated. Future research would benefit from
supplementing the current methodology by assessing more objective psychosocial and contextual
characteristics (e.g., by using ambient auditory assessment or actor-partner methodologies). The
assessment of additional biomarkers of health-related function and clinical health outcomes over
time also would help clarify how neuroticism may affect reporting of behaviors in daily life
and/or more objective health processes or health outcomes.
Actigraphy. Third, it is possible that the observed discrepancy between the objective and subjective measures of sleep and/or null findings with neuroticism and objective measures of sleep may have been due to overly conservative actigraphy settings and/or poor detection capabilities of actigraphy. For example, the Actiware algorithm used 10 immobile minutes as the basis for identifying sleep start and end times. Actiware identified the first and last group of 10 minutes during a rest interval for which all but one epoch were scored as immobile. Sleep start then was set to the first epoch satisfying these requirements, and sleep end was set to the last epoch satisfying these requirements. However, Actiware has a somewhat difficult time distinguishing between time in bed not moving and time in bed asleep; therefore, participants may have been immobile but not actually asleep.

To help ameliorate this issue, objective measures (as determined solely by Actiware and the SAS macro algorithm) ostensibly could have been used in conjunction with the sleep diary (where participants reported on the time they went to bed with the intention of sleeping and time they got out of bed each day). Sleep latency then could have been calculated by taking the time elapsed from the reported sleep diary bedtime and the actigraphy sleep start time, and sleep efficiency could have been calculated by taking the total sleep time from actigraphy divided by the time in bed from the sleep diary. However, inaccurate participant reporting of “in bed” start and end times resulted in a significant amount of negative sleep latencies (i.e., participants reported getting into bed after they fell asleep as determined by actigraphy) and sleep efficiencies greater than 100% (i.e., participants reported being in bed less time than they were asleep as determined by actigraphy). Therefore, it was decided that the solely algorithm-determined parameters would be less biased that using these measures in conjunction with the times participants reported in the sleep diary.
Due to these methodological limitations noted above, the decision was made to use the conservative, solely algorithm-determined measures. However, the use of these measures may have resulted in shorter sleep latencies and higher sleep efficiencies than were actually experienced, possible resulting in Type II error (i.e., incorrectly accepting the null hypothesis that neuroticism had no direct effect on objective sleep quality parameters). One study of patients with mild to moderate Parkinson’s disease showed that using this Actiware setting of 10 immobile minutes yielded sleep latencies that were within one minute of laboratory-based polysomnography-assessed sleep latency; however, this same study also showed that actigraphy- and polysomnography-assessed sleep latency were uncorrelated (Maglione, Liu, Neikrug et al., 2013). Therefore, future research should aim to enhance wakefulness detection capabilities of actigraphy devices, and researchers should aim to supplement actigraphy with ambulatory polysomnography techniques for triangulation of sleep measures whenever possible. With proper training of participants on how to report their “in bed” start and end times, it may be possible to use sleep diaries in conjunction with actigraphy-determined parameters to determine sleep latency and sleep efficiency.

**Correlational study design.** Finally, as previously mentioned, because this study design was correlational and not experimental, the causality of the relationships assessed cannot be determined. It is likely that bidirectional pathways exist between some of the variables assessed (e.g., sleep influences affect and affect influences sleep), or that a related third variable (e.g., depression, as previously discussed) could be influencing the observed relationships. However, the study design does allow at least partial consideration of the three requirements for causal inference: 1) temporal precedence (based on the intentional temporal ordering of the assessments of the constructs in the conceptual model), 2) covariation between predictors and outcomes, and
3) elimination of plausible alternatives (through controlling for potential confounding variables). Future research would benefit from testing lagged and bidirectional pathways between daily experiences, affect, and sleep, as well as by supplementing daily diary designs with laboratory-based studies, where common daily experiences could be manipulated in more controlled settings and assessed in relation to sleep.

**Conclusion**

The current study was the first to examine neuroticism as a predictor of daily positive and negative experiences, daily positive and negative affect, and subjective and objective measures of nightly sleep quality using an ecological, longitudinal design. Together, findings indicate that those higher in neuroticism report fewer positive experiences in daily life, as well as increased affective lability in the context of daily experiences, and poorer subjective sleep quality. Results also support the premise that heightened negative affect may be an important pathway between neuroticism and impaired subjective and objective sleep quality. Future experimental research is needed to better understand if daily experiences and affect causally impact sleep quality, and how neuroticism may modify these associations. However, this study extended the applications of the differential exposure-reactivity model and the cognitive and hyperarousal models of insomnia, providing valuable insight into the influence of neuroticism on daily experiences, daily affect, and nightly sleep quality. Together, results underscore the importance of examining the degree to which neuroticism may function not only as a single predisposing factor, but also in interaction with daily psychophysiological factors that may be related to health processes over time. Findings in the present study have the potential to make a public health impact by identifying both daily- and individual-level factors relevant for affect regulation and sleep health in everyday life that can be examined in future research.
References


Cacioppo, J. T., & Berntson, G. G. (1994). Relationship between attitudes and evaluative space: A critical review, with emphasis on the separability of positive and negative substrates.


Čukić, I., & Bates, T. C. (2015). The association between neuroticism and heart rate variability is not fully explained by cardiovascular disease and depression. *PLoS ONE, 10*, e0125882. doi: [http://dx.doi.org/10.1371/journal.pone.0125882](http://dx.doi.org/10.1371/journal.pone.0125882)


Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., Houck, P. R., Hall, M., Kupfer, D. J., & Reynolds, C. F., 3rd. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine, 65*, 63-73. doi:[http://dx.doi.org/10.1097/01.PSY.0000039756.23250.7C](http://dx.doi.org/10.1097/01.PSY.0000039756.23250.7C)


Psychosomatic Medicine, 72, 397-403.

doip: http://dx.doi.org/10.1097/PSY.0b013e3181d75319


Fredrickson, B. L. (2000). Cultivating positive emotions to optimize health and well-being. Prevention and Treatment, 3. doi: http://dx.doi.org/10.1037/1522-3736.3.1.31a


Goldberg, L. R. (1999). A broad-bandwidth, public domain, personality inventory measuring the lower-level facets of several five-factor models. Personality Psychology in Europe, 7, 7-28.


doi: [http://dx.doi.org/10.1037/hea0000042](http://dx.doi.org/10.1037/hea0000042)


doi: [http://dx.doi.org/10.2202/1557-4679.1098](http://dx.doi.org/10.2202/1557-4679.1098)


doi: [http://dx.doi.org/10.1002/jclp.20816](http://dx.doi.org/10.1002/jclp.20816)


doi: [http://dx.doi.org/10.1016/j.jadohealth.2009.06.016](http://dx.doi.org/10.1016/j.jadohealth.2009.06.016)


doi: [http://dx.doi.org/10.1027/1614-1881.1.3.86](http://dx.doi.org/10.1027/1614-1881.1.3.86)


doi: [http://dx.doi.org/10.1037//1082-989X.7.1.83](http://dx.doi.org/10.1037//1082-989X.7.1.83)


97


doi: [http://dx.doi.org/10.1016/j.cpr.2011.01.008](http://dx.doi.org/10.1016/j.cpr.2011.01.008)


doi: [http://dx.doi.org/10.3102/10769986031004437](http://dx.doi.org/10.3102/10769986031004437)


doi: [http://dx.doi.org/10.13072/midss.120](http://dx.doi.org/10.13072/midss.120)


positive as well as negative affects. *Journal of Personality*, 73, 1511-1538.


Appendix A: Inclusion/Exclusion Criteria and Neuroticism Measures

Inclusion/exclusion screening

<table>
<thead>
<tr>
<th>Variable</th>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>gender</td>
<td>What is your gender?</td>
<td>Male, Female, Transgender or other non-binary gender</td>
</tr>
<tr>
<td>olderthan18</td>
<td>Are you currently between the ages of 21 and 35?</td>
<td>Yes, No</td>
</tr>
<tr>
<td>pregnant</td>
<td>Are you currently pregnant, or trying to get pregnant?</td>
<td>Yes, No</td>
</tr>
<tr>
<td>breastfeed</td>
<td>Are you currently breastfeeding?</td>
<td>Yes, No</td>
</tr>
<tr>
<td>chrpaim</td>
<td>Do you have any sort of chronic pain or autoimmune condition (e.g., cancer, rheumatoid arthritis, lupus, HIV/AIDS)?</td>
<td>Yes (please describe), No</td>
</tr>
<tr>
<td>smtphone</td>
<td>Do you have a smartphone with a cellular data plan?</td>
<td>Yes, No</td>
</tr>
<tr>
<td>unlimtext</td>
<td>Do you have an unlimited text messaging plan?</td>
<td>Yes, No</td>
</tr>
<tr>
<td>engprim</td>
<td>Is English your primary language?</td>
<td>Yes, No</td>
</tr>
</tbody>
</table>
| engread         | If engprim is “no”:

- Do you feel you have a working knowledge of the English language? Indicate how you find reading and understanding English.

  Reading and understanding English is... | Very Difficult, Difficult, Somewhat Difficult, Somewhat easy, Easy, Very easy |

Primary neuroticism measure (Sato, 2005)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>neur1</td>
<td>Does your mood often go up and down?</td>
<td>Not at all = 1, Slightly = 2, Moderately = 3, Very much = 4, Extremely = 5</td>
</tr>
<tr>
<td>neur2</td>
<td>Do you ever feel miserable for no reason?</td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>----------------------------------------</td>
<td></td>
</tr>
<tr>
<td>neur3</td>
<td>Are you an irritable person?</td>
<td></td>
</tr>
<tr>
<td>neur4</td>
<td>Are your feelings easily hurt?</td>
<td></td>
</tr>
<tr>
<td>neur5</td>
<td>Do you often feel &quot;fed-up&quot;?</td>
<td></td>
</tr>
<tr>
<td>neur6</td>
<td>Would you call yourself a nervous person?</td>
<td></td>
</tr>
<tr>
<td>neur7</td>
<td>Are you a worrier?</td>
<td></td>
</tr>
<tr>
<td>neur8</td>
<td>Would you call yourself tense or “highly-strung”?</td>
<td></td>
</tr>
<tr>
<td>neur9</td>
<td>Do you worry too long after an embarrassing experience?</td>
<td></td>
</tr>
<tr>
<td>neur10</td>
<td>Do you suffer from nerves?</td>
<td></td>
</tr>
<tr>
<td>neur11</td>
<td>Do you often feel lonely?</td>
<td></td>
</tr>
<tr>
<td>neur12</td>
<td>Are you often troubled about feelings of guilt?</td>
<td></td>
</tr>
</tbody>
</table>

Secondary neuroticism screening (Donnellan et al., 2006)

<table>
<thead>
<tr>
<th>ipip1</th>
<th>Have frequent mood swings.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ipip2</td>
<td>Am relaxed most of the time.</td>
</tr>
<tr>
<td>ipip3</td>
<td>Get upset easily.</td>
</tr>
<tr>
<td>ipip4</td>
<td>Seldom feel blue.</td>
</tr>
</tbody>
</table>

Below are phrases describing people's behaviors. Please use the rating scale below to describe how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

<table>
<thead>
<tr>
<th></th>
<th>Very Inaccurate = 1</th>
<th>Moderately Inaccurate = 2</th>
<th>Neither Inaccurate nor Accurate = 3</th>
<th>Moderately Accurate = 4</th>
<th>Very Accurate = 5</th>
</tr>
</thead>
</table>
Appendix B: Waking Survey Measures

Sleep duration, timing, and latency

<table>
<thead>
<tr>
<th>Measure</th>
<th>Question</th>
<th>Response Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>wr_bedtimehr&lt;br&gt;wr_bedtimemin&lt;br&gt;wr_bedtimeAMPM</td>
<td>When did you go to bed last night (That is, get into bed with the intention of sleeping)?</td>
<td>Hours (0-12) Minutes (0-59) AM/PM</td>
</tr>
<tr>
<td>wr_waktimehr&lt;br&gt;wr_waktimemin&lt;br&gt;wr_waktimeAMPM</td>
<td>When did you get up this morning?</td>
<td>Hours (0-12) Minutes (0-59) AM/PM</td>
</tr>
<tr>
<td>wr_sleeplattime</td>
<td>How long did it take you to fall asleep last night?</td>
<td>0-5 minutes 6-10 minutes 11-15 minutes 16-30 minutes 31-59 minutes 1-2 hours 2-3 hours more than 3 hours</td>
</tr>
<tr>
<td>wr_sleephours</td>
<td>How many hours of actual sleep did you get last night (this may be different than the number of hours you spent in bed)?</td>
<td>1-2 2-3 3-4 4-5 5-6 6-7 7-8 8-9 9-10 10-11 11+</td>
</tr>
</tbody>
</table>

Sleep disturbance (Yu et al., 2012)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>wr_distb1</td>
<td>My sleep was restless.</td>
<td>Not at all = 1 A little bit = 2 Somewhat = 3 Quite a bit = 4 Very much = 5</td>
</tr>
<tr>
<td>wr_distb2</td>
<td>I was satisfied with my sleep.</td>
<td>Not at all = 5 A little bit = 4 Somewhat = 3 Quite a bit = 2 Very much = 1</td>
</tr>
</tbody>
</table>
| wr_distb3 | My sleep was refreshing. | Not at all = 5  
|         |                            | A little bit = 4  
|         |                            | Somewhat = 3  
|         |                            | Quite a bit = 2  
|         |                            | Very much = 1  
| wr_distb4 | I had difficulty falling asleep. | Not at all = 1  
|         |                            | A little bit = 2  
|         |                            | Somewhat = 3  
|         |                            | Quite a bit = 4  
|         |                            | Very much = 5  
| wr_distb5 | I had trouble staying asleep. | Not at all = 1  
|         |                            | A little bit = 2  
|         |                            | Somewhat = 3  
|         |                            | Quite a bit = 4  
|         |                            | Very much = 5  
| wr_distb6 | I had trouble sleeping. | Not at all = 1  
|         |                            | A little bit = 2  
|         |                            | Somewhat = 3  
|         |                            | Quite a bit = 4  
|         |                            | Very much = 5  
| wr_distb7 | I got enough sleep. | Not at all = 5  
|         |                            | A little bit = 4  
|         |                            | Somewhat = 3  
|         |                            | Quite a bit = 2  
|         |                            | Very much = 1  
| wr_distb8 | My sleep quality was… | Very poor = 5  
|         |                            | Poor = 4  
|         |                            | Fair = 3  
|         |                            | Good = 2  
|         |                            | Very good = 1  

# Appendix C: Bedtime Survey Measures

**Daily positive and negative events (DeLongis et al., 1988)**

<table>
<thead>
<tr>
<th>Hassles are irritants—things that annoy or bother you; they can make you upset or angry. <strong>UPLIFTS</strong> are events that make you feel good; they can make you joyful, glad, or satisfied. This questionnaire lists things that can be hassles and uplifts in day-to-day life. You will find that during the course of a day some of these things will have been only a hassle for you and some will have been only an uplift. Others will have been both a hassle AND an uplift.</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>UPLIFTS</th>
<th>HASSLES</th>
</tr>
</thead>
<tbody>
<tr>
<td>None or n/a = 0</td>
<td>None or n/a = 0</td>
</tr>
<tr>
<td>Somewhat = 1</td>
<td>Somewhat = 1</td>
</tr>
<tr>
<td>Quite a bit = 2</td>
<td>Quite a bit = 2</td>
</tr>
<tr>
<td>A great deal = 3</td>
<td>A great deal = 3</td>
</tr>
</tbody>
</table>

**DIRECTIONS**: Please think about how much of a hassle and how much of an uplift each item was for you today. Please indicate on the right-hand side of the page (under “HASSLES”) how much of a hassle the item was by circling the appropriate number. Then indicate on the left-hand side of the page (under “UPLIFTS”) how much of an uplift it was for you by selecting the appropriate response.

<table>
<thead>
<tr>
<th>Uplifts</th>
<th>Hassles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Your spouse, significant other, boyfriend or girlfriend</td>
<td>Your spouse, significant other, boyfriend or girlfriend</td>
</tr>
<tr>
<td>Sex or intimacy</td>
<td>Sex or intimacy</td>
</tr>
<tr>
<td>Your family</td>
<td>Your family</td>
</tr>
<tr>
<td>Pets</td>
<td>Pets</td>
</tr>
<tr>
<td>Your friends</td>
<td>Your friends</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>bd_uplift1</th>
<th>bd_stress1</th>
</tr>
</thead>
<tbody>
<tr>
<td>bd_uplift2</td>
<td>bd_stress2</td>
</tr>
<tr>
<td>bd_uplift3</td>
<td>bd_stress3</td>
</tr>
<tr>
<td>bd_uplift4</td>
<td>bd_stress4</td>
</tr>
<tr>
<td>bd_uplift5</td>
<td>bd_stress5</td>
</tr>
<tr>
<td>bd_uplift6</td>
<td>Co-workers or clients, customers, patients, etc.</td>
</tr>
<tr>
<td>---------------------</td>
<td>--------------------------------------------------</td>
</tr>
<tr>
<td>bd_uplift7</td>
<td>Your supervisor or employer</td>
</tr>
<tr>
<td>bd_uplift8</td>
<td>The nature of your work or your work load (school or other type of work) or your job security</td>
</tr>
<tr>
<td>bd_uplift9</td>
<td>Meeting deadlines or goals</td>
</tr>
<tr>
<td>bd_uplift10</td>
<td>Money or financial matters</td>
</tr>
<tr>
<td>bd_uplift11</td>
<td>Your smoking, drinking, or mood-altering drugs</td>
</tr>
<tr>
<td>bd_uplift12</td>
<td>Exercise</td>
</tr>
<tr>
<td>bd_uplift13</td>
<td>Your health, medical care, or physical abilities</td>
</tr>
<tr>
<td>bd_uplift14</td>
<td>News events, or social or political issues</td>
</tr>
<tr>
<td>bd_uplift15</td>
<td>The weather, your neighborhood, or environment (e.g., neighbors, quality of air, noise level, greenery)</td>
</tr>
<tr>
<td>bd_uplift16</td>
<td>Cooking, housework, home repairs, yardwork, or car maintenance</td>
</tr>
<tr>
<td>bd_uplift17</td>
<td>Entertainment at home (e.g., TV, music, reading, eating) or outside of the home (e.g., movies, sports, eating out, walking)</td>
</tr>
<tr>
<td>bd_uplift18</td>
<td>Amount of free time</td>
</tr>
<tr>
<td>bd_uplift19</td>
<td>Social commitments (e.g., church or community events, parties, volunteering, or gatherings with friends)</td>
</tr>
</tbody>
</table>

**Sleep impairment (Yu et al., 2012)**

<table>
<thead>
<tr>
<th>bd_imp1</th>
<th>I had a hard time getting things done because I was sleepy.</th>
<th>Not at all = 1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A little bit = 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Somewhat = 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Quite a bit = 4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Very much = 5</td>
</tr>
<tr>
<td>bd_imp2</td>
<td>I felt alert when I woke up.</td>
<td>Not at all = 5</td>
</tr>
<tr>
<td>Question</td>
<td>Response</td>
<td>Scale</td>
</tr>
<tr>
<td>----------</td>
<td>----------</td>
<td>-------</td>
</tr>
<tr>
<td>I felt tired.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
<tr>
<td>I had problems during the day because of poor sleep.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
<tr>
<td>I had a hard time concentrating because of poor sleep.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
<tr>
<td>I felt irritable because of poor sleep.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
<tr>
<td>I was sleepy during the daytime.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
<tr>
<td>I had trouble staying awake during the day.</td>
<td>Not at all = 1</td>
<td>Very much = 5</td>
</tr>
</tbody>
</table>

Positive and negative affect (McNair et al., 1981; Watson et al., 1988)

<table>
<thead>
<tr>
<th>Question</th>
<th>Response</th>
<th>Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>bd_posaff1</td>
<td>Determined</td>
<td>*High arousal PA</td>
</tr>
<tr>
<td>bd_posaff2</td>
<td>Happy/Joyful</td>
<td></td>
</tr>
<tr>
<td>bd_posaff3</td>
<td>Inspired</td>
<td></td>
</tr>
<tr>
<td>bd_posaff4</td>
<td>Attentive/Alert</td>
<td></td>
</tr>
<tr>
<td>bd_posaff5</td>
<td>Relaxed/Calm</td>
<td>*Low arousal PA</td>
</tr>
<tr>
<td>Emotional State</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>Grateful/Appreciative</td>
<td>Content/Satisfied</td>
<td></td>
</tr>
<tr>
<td>Fearful/Afraid</td>
<td>Nervous/Anxious</td>
<td></td>
</tr>
<tr>
<td>Angry/Annoyed</td>
<td>Ashamed</td>
<td></td>
</tr>
<tr>
<td>Lonely</td>
<td>Sad</td>
<td></td>
</tr>
<tr>
<td>Guilty</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*High arousal NA

*Low arousal NA
Appendix D: Sample SAS and R Code

PM = person-mean; PMC = person-mean centered; GMC = grand-mean-centered

*************************************************************
AIM ONE
*************************************************************;
*H 1.1. neuroticism predicting DNE frequency;
PROC MIXED data = sand.baseline_bed covtest noitprint method = ML;
  class ID studyday;
  model bd_stress_freq =
    neur_GMC
    age_GMC
    houseincome
    gender
    race
    ethnicity
    education
    cohort1
    cohort2
    cohort3
    cohort4
    cohort5
    cohort6
    / solution ddfm = bw notest covb;
    random intercept / subject = id type = un;
RUN;

*************************************************************
AIM TWO
*************************************************************;
*H 2.1. neuroticism moderating the link between DNEs and NA;
PROC MIXED data = sand.baseline_bed covtest noitprint method = ML;
  class ID studyday;
  model bd_NA_tot =
    bd_stress_freq_PMC
    neur_GMC
    bd_stress_freq_PMC*neur_GMC
    age_GMC
    houseincome
    gender
    race
    ethnicity
    education
    cohort1
    cohort2
    cohort3
    cohort4
    cohort5
    cohort6
    / solution ddfm = bw notest covb;
    random intercept bd_stress_freq_PMC/ subject = ID type = un;
RUN;
R code for graphing simple slopes of significant interactions:

```
xx <- c(-1.9289,1.9289)  # change to alter plot dims
yy <- c(9.3713,30.1629)  # change to alter plot dims
leg <- c(-1.9289,11.9702)  # change to alter legend location
x <- c(-1.9289,1.9289)  # x-coords for lines
y1 <- c(12.8365,16.3819)
y2 <- c(22.8631,30.1629)
xx <- c(-1.9289,1.9289)  # change to alter plot dims
yy <- c(9.3713,30.1629)  # change to alter plot dims
leg <- c(-1.9289,11.9702)  # change to alter legend location
x <- c(-1.9289,1.9289)  # x-coords for lines
y1 <- c(12.8365,16.3819)
y2 <- c(22.8631,30.1629)
plot(xx,yy,type='n',font=2,font.lab=2,xlab='Daily negative experience frequency',ylab='Negative Affect',main='Neuroticism x Daily Negative Experience Frequency on Negative Affect')
lines(x,y1,lwd=3,lty=1,col=1)
lines(x,y2,lwd=3,lty=5,col=2)
points(x,y1,col=1,pch=16)
points(x,y2,col=1,pch=16)
legend(leg[1],leg[2],legend=c('-1 SD Neuroticism','+1 SD Neuroticism'),lwd=c(3,3),lty=c(1,5),col=c(1,2))
```

AIM THREE

*H 3.1. neuroticism predicting average NA;
PROC REG data = sand.personmeans;
model bd_NA_tot_PM =
  neur_GMC
  age_GMC
  houseincome
  gender
  race
  ethnicity
  education
  cohort1
cohort2
cohort3
cohort4
cohort5
cohort6;
RUN;

*H 3.3. neuroticism predicting sleep impairment;
PROC MIXED data = sand.Baseline_bed_actigraphy covtest noitprint method = ML;
  class ID studyday;
  model bd_imp =
    neur_GMC
    age_GMC
    houseincome
    gender
    race
    ethnicity
    education
cohort1
cohort2
cohort3
cohort4
cohort5
cohort6
/ solution ddfm = bw notest covb;
   random intercept / subject = id type = un;
RUN;

*H 3.4. neuroticism and average NA predicting sleep impairment:
PROC MIXED data = sand.Baseline_bed_actigraphy covtest noitprint method = ML;
   class ID studyday;
   model bd_imp =
neur_GMC
bd_NA_tot_PM
age_GMC
houseincome
gender
race
ethnicity
education
cohort1
cohort2
cohort3
cohort4
cohort5
cohort6
/ solution ddfm = bw notest covb;
   random intercept / subject = id type = un;
RUN;

R code for generating 95% CIs for examining mediated effects:
require(MASS)
a=0.5213
b=0.01501
rep=20000
conf=95
pest=c(a,b)
acov <- matrix(c(
  0.0182412718, 0,
  0, 0.000013
),2,2)
mcmc <- mvrnorm(rep,pest,acov,empirical=FALSE)
ab <- mcmc[,1]*mcmc[,2]
low=(1-conf/100)/2
upp=((1-conf/100)/2)+(conf/100)
LL=quantile(ab,low)
UL=quantile(ab,upp)
LL4=format(LL,digits=4)
UL4=format(UL,digits=4)
hist(ab,breaks='FD',col='skyblue',xlab=paste(conf,'% Confidence Interval','LL',LL4,'  UL',UL4),
main='Distribution of Indirect Effect')
Appendix E: Sample Scored Actigraphy Record using a Graphical User Interface
EDUCATION

Ph.D. 2017  Biobehavioral Health, The Pennsylvania State University
M.S. 2015  Biobehavioral Health, The Pennsylvania State University
B.A. 2012  Psychology (with honors, magna cum laude), Beloit College

SELECTED HONORS, FELLOWSHIPS, and AWARDS

2017  Penn State Alumni Association Dissertation Award
2017  American Psychosomatic Society Young Scholar Award
2013-2016  National Science Foundation Graduate Research Fellowship
2016  American Psychological Association Dissertation Research Award
2016  Graduate Student Research Endowment, Penn State
2016  Kligman Graduate Fellowship Endowment, Penn State
2016  Edward & Helen Skade Hintz Graduate Enhancement Award, Penn State
2015  Graduate Student Research Endowment, Penn State
2015  Edward & Helen Skade Hintz Graduate Enhancement Award, Penn State
2015  Gerald E. McClearn Graduate Student Award, Penn State
2015  Graduate Scientific Achievement Award, Penn State
2012  Fund for Excellence in Graduate Recruitment, Penn State

SELECTED PUBLICATIONS


SELECTED PRESENTATIONS

