June 2019

Linking Sleep and Aggression: The Role of Response Inhibition and Emotional Processing

Melanie L. Bozzay
University of South Florida, melanie_bozzay@brown.edu

Linking Sleep and Aggression:

The Role of Response Inhibition and Emotional Processing

by

Melanie L. Bozzay

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
Department of Psychology
College of Arts and Sciences
University of South Florida

Major Professor: Edelyn Verona, Ph.D.
Mark Goldman, Ph.D.
Geoffrey Potts, Ph.D.
Robert Schlauch, Ph.D.
Brent Small., Ph.D.

Date of Approval:
June 26, 2019

Keywords: ERP, violence, cognition, experimental

Dedication

This thesis is dedicated to the mentors, friends, and family who have supported me in my scientific endeavors. I would like to thank Dr. Edelyn Verona (my faculty advisor), for her tireless support throughout my graduate training and for her thoughtful contributions to my professional and personal development. I was fortunate to have been supported in many ways by my good friends and labmates (Bethany Edwards, Amy Hoffman, Sean McKinley, Julia McDonald), who have motivated me to continue with and complete this work. This work is also dedicated to my parents and siblings, and grandparents Jack and Amber King, who have been so supportive throughout this process. Lastly, I am beyond grateful to Jordan Hall – my best friend and strongest supporter. Without him, none of this work would have been possible.

Acknowledgements

This project was supported in part by grant R21MH109853 from the National Institute of Mental Health, awarded to Dr. Edelyn Verona. It was also supported by grants awarded to Melanie Bozzay, MA from Psi Chi, Sigma Xi, and the American Psychological Association.

Table of Contents

List of Tables iv

List of Figures v

Abstract vii

Overview and Scope of the Problem 1
- The Relevance of Sleep Duration 2
- Sleep Duration and Aggressive Behavior 4
- Sleep Duration and Response Inhibition 6
- Sleep Duration and Negative Emotional Processing 7
- Emotional Processing, Response Inhibition, and Aggression 8
- Operationalizations of Response Inhibiton and Emotional Processing 10
- Relevance of the Proposed Study 13
- Aims and Hypotheses 14

Method 17
- Participants 17
- Procedures 18
  - Recruitment 18
  - Sleep Tracking between Session 1 and Session 2 19
  - Overview of Session 2 19
- Sleep Tracking and Measurement 21
  - Sleep Duration 21
  - Sleep Duration Measurement Strategy 23
  - Supplemental Daily Assessments 24
- Laboratory Assessments of Emotional Processing and Response Inhibition 26
- Laboratory Assessment of Aggression 27
  - Changes in Affect 30
  - Debriefing and Effectiveness of Deception 31
- Clinical and Trait Measures 32
  - Demographics 32
  - Last Month Sleep Quality 32
  - Aggression Proneness and Lifetime Violent Acts 33
  - Substance Use Problems 34
  - Internalizing Symptoms 35
- Physiological Data Acquisition 36
  - Offline Data Processing 36

Data Analysis 42
Supplemental Analyses 40
Power Analysis 41

Results 42
Sample Descriptives 42
Aggression Paradigm Validity 44
Provocation 44
Construct Validity of the Aggression Paradigm 46
Aim 1: Linking Sleep Duration with Aggression 47
Sleep Relations with Laboratory Aggression 47
Sleep Relation with Aggression Proneness and Lifetime Aggressive Acts 48
Sleep Relations with Daily Reports of Hostility and Aggression 49
Aim 2: Linking Sleep Duration with Response Inhibition and Emotional Processing 50
Analyses of Electrode Site 50
Overall Task Effects 52
Sleep Effects on P3 54
Sleep Effects on N2 60
Behavioral Results 61
Commission Errors 61
Reaction Time 62
Potential Confounds of Aims 1 and 2 64
Aim 3: Explaining the Sleep-Aggression Relationship 65
Preliminary Analyses 65
Analyses of Indirect Effects 66
Supplemental Analyses 72

Discussion 74
Aim 1: Linking Sleep Duration with Aggression 74
Aim 2: Linking Sleep Duration with Response Inhibition and Emotional Processing 77
Aim 3: Explaining the Sleep-Aggression Relationship 80
Limitations 82
Strengths and Conclusions 83

References 85

Appendix A: General Recruitment 107

Appendix B: Targeted Recruitment 108

Appendix C: Example General Flyer 109

Appendix D: Example Specific Flyer 110

Appendix E: Screening Procedures 111

Appendix F: IRB Approval 114

Appendix G: Sleep Diary 116

Appendix H: Emotional Go/No-Go Task 119

Appendix I: Phase 2 of the Aggression Paradigm 120

Appendix J: Post-Study Questionnaire 121

Appendix K: Post-Study Interview 124

Appendix L: Participant Demographics Form 126

Appendix M: Montages Used in Data Analyses 137

List of Tables

Table 1:	Sample Characteristics	17
Table 2:	Associations between sleep duration, aggressive tendencies, lifetime history of aggressive behavior, and three-day reports of substance use and hostile urges	43
Table 3:	RMANOVA Effects on Average Shock Intensity	48
Table 4:	RMANOVA Effects by Site and within Site	53
Table 5:	RMANOVA Condition Effects as a Function of Sleep Duration	60
Table 6:	Associations between sleep, aggression, and indices of response inhibition and emotional processing	67
Table 7:	Indirect effects linking sleep duration with measures of aggressive behavior	69

List of Figures

Figure 1: Study procedures 20

Figure 2: Sample size across analyses 38

Figure 3: Average PANAS Negative Affect scores across Session 2 45

Figure 4: Average PANAS Hostility and Irritability scores across Session 2 46

Figure 5: Average shock intensity across blocks by sleep duration groups 49

Figure 6: Grand average ERP waveforms. Figure A: Frontocentral. Figure B: Parietal 51

Figure 7: Amplitude of P3 responses as a function of word category and site. 52

Figure 8: Amplitude of frontocentral P3 as a function of trial type (go vs. no-go) and continuous scores on sleep duration 55

Figure 9: Amplitude of Frontocentral P3 as a function of word category and sleep duration. 56

Figure 10: Amplitude of frontocentral P3 as a function of word category and sleep duration simple slopes 56

Figure 11: Amplitude of frontocentral P3 responses as a function of word category and trial type 57

Figure 12: Amplitude of frontocentral P3 as a function of word category, trial type and sleep duration. 58

Figure 13: Amplitude of frontocentral P3 as a function of word category, trial type, and sleep duration simple slopes 59

Figure 13: Commission errors as a function of word category and sleep duration 62

Figure 15: Reaction time as a function of word category and sleep duration. 63

Figure 16: Reaction time as a function of word category and sleep duration simple slopes. 63

Figure 17: Models linking sleep duration, response inhibition (Go/No-Go N2 and P3), emotional processing (Negative-Neutral and Positive-Neutral P3), and indices of aggression. 70

Figure 18: Models linking sleep duration, response inhibition during emotional conditions (FC P3), and aggression 71

Figure 19: Models linking sleep duration and aggression via commission errors 71

Figure 20: Models linking sleep duration and aggression via reaction time 72

Abstract

Although shorter sleep duration is theorized to increase the risk of engaging in aggressive behavior, experimental studies examining this relationship yield conflicting findings. Since sleep serves in part to regulate the functioning of prefrontal brain regions, insufficient sleep may deleteriously impact the individual's ability to inhibit rash action and alter emotional processing, which could in turn increase aggressive tendencies. However, no studies have examined the extent to which naturally occurring insufficient sleep is linked to aggression or potential mechanisms of this relationship, limiting understanding of and the generalizability of extant findings. Thus, the present study examined whether cognitive (deficits in response inhibition) and emotional processes (increased negative emotional processing) help explain relationships between sleep duration and aggression. Approximately 143 participants between the ages of 18 and 40 were recruited from a larger, grant-funded aggression study. Participants wore Fitbit Flex sleep-tracking devices and kept a sleep diary to monitor sleep duration over a three-day period prior to the laboratory session. At the laboratory session, electrophysiological indices of emotional processing and response inhibition (P3 and N2) were measured via an Emotional Go/No-Go task, and aggression under provocation was measured using a laboratory aggression paradigm. Mixed-model repeated measure ANOVAs tested the relationships between sleep duration, emotional processing, response inhibition, and aggression, controlling for potential confounds (e.g., substance abuse, gender). Path analyses examined whether emotional processing and response inhibition mediated the sleep-aggression relationship. As expected, less sleep duration was associated with greater intensity of aggression observed in the laboratory.

Interestingly, despite showing increased inhibition processing towards emotional stimuli, lower sleepers performed similarly across emotional conditions, indicating that the emotional processing biases apparent at lower levels of sleep did not translate to better performance. Moreover, although inhibitory and emotional processing related to sleep and aggression, albeit in somewhat different patterns, these mechanisms did not explain the sleep-aggression link. These results provide the first evidence that shorter sleep duration predicts laboratory aggressive behavior, and preliminarily suggests that shorter sleepers work harder in emotional contexts to inhibit behavior comparably to neutral contexts. Implications of these findings for understanding aggression will be discussed.

Overview and Scope of the Problem

Despite the known health concerns associated with short sleep (e.g. increases in morbidity and mortality; Van Cauter, Spiegel, Tasali, & Leproult, 2008; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002), reports of regularly sleeping less than recommended (<7) hours a night have increased over the past few decades (Van Cauter et al., 2008; Kronholm et al., 2008). Although the impact of sleep on health outcomes is well-known (Bixler, 2009), fewer studies have examined the effects that sleep duration may have on behavioral health in particular, including engagement in risky or maladaptive behaviors. Correlational studies suggest that sleep duration is implicated in a variety of maladaptive behaviors that are costly to both the individual and society (e.g. self-injury, Junker, Bjorngaard, Gunnell, & Bjerkeset, 2013; substance abuse, Wong, Brower, Fitzgerald, & Zucker, 2004; aggression, see Krizan & Herlache, 2016 for a review; suicide, see Pigeon, Pinquart, & Connor, 2012 for a review). Notably, these behaviors often co-aggregate in individuals, perhaps due to shared propensities toward impulsivity or disinhibition (Anestis, Soberay, Gutierrez, Hernandez, & Joiner, 2014; Iacono et al., 1999; Littlefield & Sher, 2010; Mullins-Sweatt et al., 2013) and increased responsivity to negative emotional stimuli **(**Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Cooney, Litt, Morse, Bauer, & Gaupp, 1997; Glenn, Blumenthal, Klonsky, & Hajcak, 2011**)**. However, there are few experimental or quasi-experimental studies linking sleep duration with potential mechanisms associated with disinhibited behaviors (see Krizan & Herlache, 2016 for a review of laboratory aggression studies), limiting knowledge needed to inform more targeted prevention efforts. The purpose of the proposed study is to examine whether cognitive (deficits in response inhibition)

and emotional processes (increased emotional processing) link sleep duration with aggression, one such disinhibitory behavior that is more readily examined in a laboratory setting (Krizan & Herlache, 2016).

The Relevance of Sleep Duration

Perhaps as a consequence of technological advancements (e.g., smartphones) and increased work demands of industry, reports of regularly sleeping less than recommended have increased over the past few decades (Van Cauter et al., 2008; Kronholm et al., 2008), particularly in modernized countries. Indeed, although there is some variability in sleep needs between individuals, at least 7 hours are recommended per night (Hafner, Stepanek, Taylor, Troxel, & van Stoke, 2016). That approximately 30% of American adults report sleeping less than 7 hours per night (Hafner et al., 2016) is notable, since experimental research indicates that decrements in cognition and performance emerge following less than seven hours of sleep (Belenky et al., 2003; Van Dongen, Rogers, & Dinges, 2003) and become increasingly pronounced in the presence of multiple nights of disturbed sleep (Dinges & Kribbs, 1991; Doran, Van Dongen, & Dinges, 2001). Moreover, the rate at which these deficits increase occurs as a function of the magnitude of sleep restriction (Belenky et al., 2003; Van Dongen, Maislin, Mullington, & Dinges, 2003; Cote et al., 2009). Indeed, individuals who regularly sleep less than 7 hours exhibit deficits in cognition and performance that parallel levels seen among individuals under conditions of total sleep deprivation (Van Dongen et al., 2003). Even when shorter sleep is not chronic, the neurobehavioral deficits incurred by even one poor night of sleep may take several days to rebound, since a longer sleep period during one night (e.g., 10 or more hours), and/or multiple nights of recovery sleep, are need to restore these functions following short sleep

(Banks, Van Dongen, Maislin, & Dinges, 2010). Thus, even a few nights of shorter sleep may have negative implications for an individual's daytime functioning.

There are a variety of sleep disorders that have deleterious impacts on daytime functioning (American Psychiatric Association, 2013), including disorders of initiating and maintaining sleep (e.g., insomnias, sleep apnea syndrome, restless leg syndrome) and parasomnias (e.g., sleepwalking, sleep terrors). Shorter sleep duration is a symptom of these disorders (American Psychiatric Association, 2013), but is also a naturally occurring behavior in the general population. That is, shorter sleep does not occur exclusively in the context of sleep disorders, occurring at a much a higher prevalence than sleep disorders (30% compared to 0.2-4% in these disorders; see Barclay & Gregory, 2013 for a review). Examining the role of reduced sleep duration, rather than more specific sleep diagnoses, in risk for aggression would allow for the analysis of these relationships in varying populations (not just those in medical settings). Thus, in the present study, we focus on naturalistic assessment of sleep duration in examining the link between sleep and aggressive behavior.

Sleep duration can be measured via several methodologies. In correlational studies, sleep duration can be measured as it occurs naturalistically via self-report (e.g. sleep diaries), mobile commercial (e.g., Fitbit) and medical (e.g., Actigraph) devices, and intensive observation and recording (e.g., polysomnography) of brain wave data, methods that demonstrate acceptable validity and reliability in measuring sleep duration (e.g., Evenson et al., 2015; McCall & McCall, 2012). To examine whether extreme sleep loss produces deleterious waking consequences, experimental studies manipulate sleep duration by limiting the hours of sleep an individual experiences through total (e.g., restricting a full night of sleep or several nights of sleep) or partial (e.g., restricting several hours of sleep) sleep deprivation. Although these naturalistic and

experimental methods are associated with varying durations of sleep, they produce similarly deleterious neurobehavioral effects, including reduced psychomotor vigilance, reduced energy, and impaired executive functioning (Reynolds & Banks, 2010; Van Dongen et al., 2003). Thus, these results suggest varying ranges of sleep duration impact the same mechanisms, and provide some rationale for expecting results from experimentally-induced shortened sleep to generalize to real-word consequences of sleep loss.

Sleep Duration and Aggressive Behavior

Unfortunately, only a handful of studies have examined the sleep-aggression relationship, and those that measured sleep duration naturalistically (versus induced short sleep experimentally) yield conflicting findings. Correlational studies mostly employ sleep diaries or duration subscales of validated sleep measures to assess sleep duration, and find small to moderate associations between sleep duration and self-reported aggression in youth samples (school children, Scharf et al., 2013; Yokomaku et al., 2008; adolescents in school, Lemola et al., 2012; Meijer et al., 2000; troubled adolescents, Haynes et al., 2006; Ireland & Culpin, 2006), young adults (college students, Randler et al., 2013; male soldiers; Semiz et al., 2008) and incarcerated middle-aged men (Vogler et al., 2014). The drawback of these studies is that they are cross-sectional, precluding assumptions about temporal ordering of effects. The longitudinal studies in this area have employed the same measures, finding small sleep-aggression relationships over several years in young children (Sheridan et al., 2013) as well as for next day aggression in psychiatric intensive care (Langsrud et al., 2018). Although these cross-sectional and longitudinal studies suggest that there is a relationship between shorter sleep duration and aggressive behavior, the robustness and generalizability of these findings are currently unknown. Indeed, besides the fact that only a handful of these studies have been conducted, the studies in

adults in particular have been conducted in samples that are predominantly male and high risk (e.g., incarcerated men, psychiatric inpatients, or soldiers). As such, the extent to which the sleep-aggression relationship holds in adults in the general population is unclear.

Notably, the three published experimental sleep studies (i.e., induced sleep deprivation) in the area of aggression differ in their results from the correlational studies. In one experiment, young adult military service members experiencing 55 hours of continuous wakefulness demonstrated both greater tendencies to blame others for problems and reduced willingness to accept blame on a projective test (Kahn-Greene et al., 2006). In another study, a subset of rested controls as well as participants who had been deprived of 24 hours of sleep completed a cognitive depletion procedure; participants who had undergone the cognitive depletion were more aggressive, *regardless of sleep deprivation condition* (Vohs, Glass, Maddox, & Markman, 2011). Another study using a laboratory aggression paradigm (i.e., point subtraction task) found unexpected results: provoked men deprived of 33 hours of sleep failed to increase point-stealing (i.e., aggressive) behavior to the same extent as provoked rested controls (Cote, McCormick, Geniole, Renn, & MacAulay, 2013).

In sum, correlational studies provide preliminary support for the role of shorter sleep duration in aggressive behavior, although this relationship has not been studied in varying adult samples. The results of these correlational studies differ from those of several existing experimental studies showing no changes in aggression or *less* aggression following total sleep deprivation (Cote et al., 2013; Vohs et al., 2011). Given these limitations, more research is needed to understand the nature of the sleep-aggression relationship. In the present study, we examine whether shorter sleep duration will predict higher levels of laboratory-assessed

aggression, in part as a function of response inhibition deficits and increased negative emotional processing resulting from sleep loss.

Sleep Duration and Response Inhibition

Shorter sleep duration may increase the propensity to engage in aggression by impairing cognitive response inhibition processes, which are required to restrain inappropriate or undesired actions that could interfere with attaining goals (Lezak et al., 2004). Response inhibition encompasses two primary processes: attention to incoming stimuli, and prevention of an automatic response, both processes that rely on limited cognitive resources to function (Lezak et al., 2004). Importantly, response inhibition is regulated by the prefrontal cortex (PFC; Botvinick, Braver, Barch, Carter, & Cohen 2001), a region of the brain found to be particularly vulnerable to sleep loss (Harrison & Horne, 2000; Horne, 1993). Indeed, the PFC is theorized to rejuvenate from waking activities during sleep through Non-Rapid-Eye-Movement (NREM) and Rapid-Eye-Movement (REM) episodes (Vyazovskiy & Delogu, 2014). The slow-wave oscillations that take place during NREM are thought to enable brain network recovery of a range of processes spanning molecular, cellular, and network levels (Vyazovskiy & Harris, 2013), with the REM stage following NREM thought integral in identifying brain networks still requiring recovery during the next NREM cycle (see Vyazovskiy & Delogu, 2014 for a review). Since these stages repeat approximately every 70 to 120 minutes throughout the night, and are iterative in nature, reduced sleep duration may not allow sufficient time for different components of the brain, including the PFC, to recover from a lengthened period of wakefulness. Indeed, research indicates that the PFC is impaired following a night of sleep deprivation, evidenced by reduced blood flow to prefrontal areas that correspond to deteriorations in performance on executive control tasks including inhibition (Blagrove et al., 1995; Harrison et al., 2000; Horne, 1993). As

such, shorter sleep duration may impair self-control by reducing the metabolic resources available for attentional control and the engagement of response prevention processes in the PFC. Thus, we expect that individuals with shorter sleep duration will be more likely to engage in aggression, in part due to having less effort available to expend on inhibition processes.

Sleep Duration and Negative Emotional Processing

In addition to impacting response inhibition processes, shorter sleep duration may also contribute to aggressive propensities by increasing processing of negative stimuli. Sleep appears to play an integral role in maintaining the homeostasis of emotional brain function (Levin & Nielsen, 2009; Walker & van der Helm, 2009), as demonstrated by changes in emotional reactivity and mood following sleep deprivation. Reduced inhibitory connections between the prefrontal cortex and amygdala have been observed following a night of sleep deprivation, and linked with increased amygdala reactivity in response to negative emotional stimuli (Yoo et al., 2007). Indeed, larger pupillary responses have also been observed when viewing negative emotional images following sleep deprivation (Franzen et al., 2009).

Cognitive changes linked with shorter sleep duration appear to also contribute to changes in emotional processing. Indeed, when fatigued, a greater proportion of limited attention and energy resources is allocated towards negative or threatening stimuli relative to neutral stimuli (Barclay & Ellis, 2013). Furthermore, the consolidation of negative emotional information in memory can be disrupted by shorter sleep, and these disruptions have been linked with persistent amygdala reactivity during later recollection (Sterpenich et al., 2007). Perhaps as a consequence of these changes in responsivity to negative emotional stimuli, shorter sleep duration has been linked with increased feelings of negative affect that are distally (anxiety, depression, and

irritability, Dinges et al., 1997) and proximally (anger and hostility; Haack & Mullington, 2005; Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007) related to feelings of aggression.

Alternatively, it is possible that individuals with shorter sleep duration allocate more effort to processing *emotional* information more broadly, rather than negative information specifically. Indeed, similar to the results for negative emotional stimuli reported by Yoo et al. (2007), functional magnetic resonance imaging research has correlated sleep deprivation (1 night) with increased brain activation to positively valent visual stimuli (e.g., images from the International Affective Picture System), in conjunction with decreased functional connectivity with cognitive control regions (Gujar, Yoo, Hu, & Walker, 2011; Volkow et al., 2009). Attending to valent information in the presence of limited cognitive resources may enable focusing on information that could be useful for survival, and thus inherently such a process could be adaptive for individuals with shorter sleep. However, to our knowledge, no studies examining emotional processing in real-time at the neural level (e.g., via event-related potentials) have included both negatively and positively valenced stimuli. Thus, whether there are emotional processing biases in the presence of shorter sleep duration as it naturally occurs, and whether such biases may be specific to negative information, or emotional information more broadly, are important empirical questions. However, we expect that increased negative emotional processing in particular will relate to shorter sleep duration and aggression, since responsivity to negative emotional stimuli is heavily implicated in aggression (see Davidson et al., 2000 for a review).

Emotional Processing, Response Inhibition, and Aggression

We expect that emotional processing interacts with response inhibition to increase the risk of aggression. Specifically, increased processing of negative emotional stimuli can prime instinctive, mood-congruent responses to situations. Indeed, negative affect activates mood-

congruent information in working memory (Dolcos et al., 2003) and increases expectations that punishing and aversive events will occur (Handley et al., 2004). These factors may activate multiple, competing response options in working memory (Botvinick et al., 2001; Brown & Braver, 2005). While cognitive control processes can be engaged to resolve this conflict and determine behavior, energy resources are limited following shorter sleep, which promotes reliance on habitual responses (e.g., aggression), and decreased goal-directed responding (e.g., alternative responses or behavior inhibition; Dias-Ferreira et al., 2009).

In turn, negative emotional processing (e.g., regarding a potentially threatening situation) can impact cognition by diverting attention and processing resources from other functions (Carretie et al., 2001; McKenna & Sharma, 1995; Wyble, Sharma, & Bowman, 2008), impairing performance on tasks (e.g. situational reappraisal) that rely on higher level information (as seen in results from Algom, Chajut, & Lev, 2004; De Houwer & Tibboel, 2010; McKenna & Sharma, 2004; Pessoa, 2009; Verbruggen & De Houwer, 2007). This diversion of resources could decrease the likelihood that the instinctive response (e.g., aggression) will be inhibited. Since engaging in alternative strategies may first require inhibition of the primed impulsive response, this resource depletion could also decrease the likelihood that alternative responses to the situation will be attempted. Indeed, sleep-deprived individuals have been found less likely to reappraise negative information and, perhaps consequently, found more likely to blame others (Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006; Mauss et al., 2013), tendencies that have also been implicated in mood-congruent responding to affective stimuli (Yoo et al., 2007). As a result, the individual may engage in more aggressive responding to situations appraised as threatening or distressing. Thus, we expect reduced response inhibition as a function of negative emotional processing, under conditions of shorter sleep.

Operationalizations of Response Inhibition and Emotional Processing

Taken together, we expect that response inhibition and emotional processing may separately and interactively explain the relationship between shorter sleep and aggressive responding. In order to disentangle the nature of these relationships, we will use an ERP task to examine the effort devoted to different aspects of response inhibition and emotional processing (cognitive indices), as well as the accuracy and latency of responses (behavioral indices). Response inhibition requires diversion of attention to incoming stimuli (e.g., to aid in distinguishing between stimuli warranting different responses), and subsequent prevention of an automatic response (Lezak et al., 2004). In electrophysiological studies using typical Go/No-Go tasks (e.g., Falkenstein et al., 1999), which require responding to certain stimuli (Go stimuli) but not others (No-Go stimuli), the difference in amplitude of the N2/P3 components measured during Go versus No-Go conditions indexes deployment of cognitive resources to the attention (No-Go N2) and motor inhibition (No-Go P3) components of response inhibition. The N2 is a negative-going component of the EEG waveform occurring 200-400 ms after stimulus presentation that appears maximally at frontocentral sites (Bekker, Kenemans, & Verbaten, 2004; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2004). While the No-Go N2 is thought to index top-down mechanisms needed to inhibit an incorrect tendency to respond (Falkenstein, 2006), its functional specificity is debated. The No-Go N2 has been associated with detection of a conflict between initiated and required responses, action monitoring, and effortful attention (Donkers & van Boxtel, 2004; Nieuwenhuis et al., 2003; van Veen & Carter, 2002; Yeung et al., 2004), suggesting it mirrors a wide range of cognitive control processes. However, the literature appears to largely agree that it emerges due to employing cognitive resources involved in inhibitory control. The N2 is followed by the P3, a positive deflection that is

maximal at frontocentral sites in the Go/No-Go task (Falkenstein et al., 1999; Smith, Johnstone, & Barry, 2008), peaking between 300-600 ms after the presentation of the stimulus (Kopp et al., 1996; Pfefferbaum et al., 1985). It has been linked with inhibition of the motor system in the premotor cortex (Falkensetein et al., 1999; Smith et al., 2008). Behavioral indices, such as commission errors (e.g., incorrectly responding to No-Go stimuli), can provide additional observable markers of failures of response inhibition.

However, few studies have used cognitive and behavioral indices to examine the association between sleep duration and response inhibition. One total sleep-deprivation Go/No-Go study found that participants with 43 hours of continuous wakefulness compared to a rested control group displayed smaller No-go N2 and P3 amplitudes and more commission errors (Qi, Shao, Miao, Fan, Bi, & Yang, 2010), suggestive of impairments in inhibition demands or response conflict associated with sleep deprivation. Supporting these findings, a study comparing the effects of short sleep (e.g., 6 hours of sleep or less per night for 4 nights) versus long sleep (e.g. 9 hours of sleep per night for 4 nights) in a Go/No-Go Task found that short sleep participants made more commission errors (Demos et al., 2016). These two studies preliminarily support an association between sleep deprivation and impaired response inhibition processes. However, research is needed to replicate these results and determine whether they extend to naturalistic sleep duration.

Emotional processing, or attention to emotion, may also be measured through electrophysiological methods. The P3 is theorized to comprise a cognitive index of the allocation of resources toward environmental stimuli that are motivationally salient (Hajcak & MacNamara, 2010). Since emotional stimuli provide motivationally significant information (e.g., Lang & Bradley, 2010), they may naturally capture more processing resources and elicit enhanced P3

(Hajcak et al., 2010). Indeed, an increased parietal P3 has been observed across a variety of paradigms for emotional relative to neutral pictures (oddball paradigm, Delplanque, Silvert, Hot, & Sequeira, 2005; picture viewing paradigm, Palomba, Angrilli, & Mini, 1997), and words (affective lexical decision task, Carretie, Hinojosa, Albert, & Sotillo, 2008; structural and affective processing task, Naumann, Bartussek, Diedrich, & Laufer, 1992). In the proposed study, the difference in amplitude of the P3 in response to negative or positive versus neutral stimuli will be examined as an index of negative emotional processing (Negative-Neutral Words; Positive-Neutral Words).

Finally, interactive contributions of response inhibition and emotional processing to the sleep-aggression relationship will be identified using an experimental paradigm in which both of these processes are challenged. The Emotional-Linguistic Go/No-Go task (based on Goldstein et al., 2007 and modified by Verona, Sprague, & Sadeh, 2012) presents go and no-go trials embedded in blocks of negative and neutral stimuli, which is particularly suited to examine the degree to which emotional processing may modulate response inhibition. In this task, cognitive (e.g., P3) and behavioral indices (e.g., commission errors and reaction time) can provide measurements of the extent to which facilitated processing of emotional stimuli may decrease response inhibition process (e.g., reduced emotion P3 under no-go trials) and increase impulsive responding (e.g., greater errors and shorter reaction time to negative versus neutral stimuli). To date, no study has examined the modulation of response inhibition by emotional processing in the context of sleep duration, and only two studies have examined behavioral indices, with conflicting results (Anderson & Platten, 2011; Rossa et al., 2014). As such, the proposed study will fill important gaps in the literature.

Relevance of the Proposed Study

This study will be the first to examine the extent to which *naturally occurring* sleep duration may predict *subsequent* engagement in aggressive behavior. This use of naturalistic sleep duration, rather than experimentally-induced sleep restriction, to temporally predict aggression is a methodological strength that may aid in resolving the conflicting findings between correlational and experimental studies in this area (see Krizan & Herlache, 2016 for a review). Although naturalistic shorter sleep and experimental sleep deprivation produce some similar daytime consequences (e.g., impaired executive functioning; Reynolds & Banks, 2010; van Dongen et al, 2003), it is possible that symptoms such as extreme levels of fatigue, amotivation, and disengagement predominantly resulting from total/partial sleep deprivation (Alhola & Polo-Kantola, 2007; Cote, Milner, Osip, Baker, & Cuthbert, 2008) actually produce the *decreased* aggression observed in experimental studies. If the proposed study finds that naturalistic levels of shorter sleep are positively related with subsequent aggression, this may provide some explanation for diverging findings, suggesting that varying levels of sleep duration differentially drive aggression (e.g., partial/total sleep deprivation linked with less aggression, less extreme levels of sleep loss linked with more aggression). The absence of a relationship would suggest that other variables (e.g., emotional agitation) may explain the reported sleep-aggression link.

This study will also contribute to the literature by improving on criticisms of prior laboratory aggression tasks. Typical laboratory aggression studies in relation to sleep have utilized competition-based reaction time aggression paradigms (Cote et al., 2013; Vohs et al., 2011). These paradigms require sustained attention and vigilance, and thus may be particularly susceptible to the fatigue and amotivation that is endemic to total or partial sleep deprivation

(Cote et al., 2008), resulting in greater likelihood of *reduced* aggression in sleep-deprivation experiments that may not parallel sleep-aggression effects as they naturally occur. Laboratory aggression paradigms are criticized more generally as lacking in ecological validity, in part because they do not appear to relate to aggressive tendencies or violent behavior, and do not provide non-aggressive alternative response options (Ferguson & Rueda, 2009). We will address some of these criticisms by using a laboratory task drawing on provocation to elicit aggressive responses toward the provocateur (Verona, Sadeh, & Curtin, 2009; Verona, Patrick, & Lang, 2002), allowing the participant to select varying severities of aggressive responses or a non-aggressive response. This task may be better suited to examine tendencies towards aggression under conditions of sleep-deprivation due to its utilization of an instance of provocation to induce aggression, instead of provoking aggression during a time of sustained vigilance and in the guise of competition. Although this task remains subject to criticisms of aggression paradigms more broadly regarding other limitations to ecological validity, including distance between the participant and confederate and permissiveness of an authority figure (the experimenter) (see Ritter & Eslea, 2005, for a review; see Tedeschi & Quigley, 1996 for a review), aggression observed in the laboratory on similar tasks has been found to relate to self-reported aggression and hostility (Cherek, Moeller, Schnapp, & Dougherty, 1997; Verona et al., 2002; Verona & Kilmer, 2007), as well as violent criminal behavior (Cherek et al., 1997). To address these possible limitations, we will also evaluate naturalistic sleep relationships with aggressive tendencies and aggressive behavior outside of the laboratory (e.g., self-report measures) for concordance.

Finally, despite theoretical papers positing that response inhibition and emotional processes may underlie the sleep-aggression relationship (Krizan & Herlache, 2016), no studies

have directly tested these hypotheses. The use of the Emotional Go/No-Go task is a methodological strength of this study. It enables teasing out the unique contributions of emotional processing (e.g., across varying inhibitory control demands: go versus no-go trials), and response inhibition (e.g., across negative, positive, and neutral stimulus emotional categories), as well as the nature of their likely interaction to aggressive responding in the presence of shorter sleep. It also enables examination of the extent to which these variables may be sleep-related mechanisms that are prospectively associated with laboratory aggression. Understanding the extent to which these processes may contribute to impulsive responding, and, in particular aggressive behavior, is an important next step in understanding actions that could be taken to reduce aggression.

Aims and Hypotheses

There are three primary aims of the proposed study. The first aim is to examine the association between sleep duration across three nights and subsequent observed aggression. We expect that 1) shorter sleep will be associated with greater engagement in observed and self-reported aggressive tendencies and behavior. The second aim focuses on possible mechanisms of this relationship, specifically whether sleep duration impacts response inhibition, particularly under negative emotional conditions. We expect 2) reduced response inhibition (i.e., frontocentral N2 or P3 in no-go – go trials) in individuals with shorter sleep (Sleep Duration x Go/No-Go trial), indicative of reduced engagement of response inhibition processes under poor sleep. We also expect that 3) emotional processing will be heightened by shorter sleep duration, such that shorter sleepers will exhibit increased parietal P3 to emotional stimuli compared to neutral stimuli (Sleep Duration x Emotional Category). Finally, we expect that 4) there will be an interaction between sleep duration, inhibitory demands, and emotional category, such that shorter

sleepers will display reduced frontocentral no-go P3 during emotional versus neutral word categories (Sleep Duration x Go/No-Go Trial x Emotional Category). For Hypotheses 3 and 4, due to the dearth of literature examining the N2 in conjunction with emotional processing, we will include the N2 in exploratory analyses. Although our ERP indices are the main dependent measures in this aim, we will secondarily examine behavioral responses on the task. We expect that shorter sleep will be linked with 5) more commission errors (responding when they should not) and 6) shorter reaction times to emotional stimuli compared to neutral stimuli, reflecting facilitated processing of emotional stimuli.

The third aim is to explore if response inhibition and emotional processing explain variance in the link between sleep duration and aggression. If this relationship is primarily due to response inhibition, we expect that 7) the No-Go N2 or P3 will explain the sleep-aggression relationship. If this relationship is primarily due to increased emotional processing, we expect that 8) the amplitude of the P3 to negative versus neutral or positive blocks will explain this relationship. However, if this relationship is due to the confluence of response inhibition and emotional processing, we expect that 9) the No-Go P3 during negative, but not neutral or positive stimuli, will explain the sleep-aggression relationship. This study will aid in furthering understanding of emotional and response inhibition processes that may underlie the relationship between sleep duration and aggressive behavior.

Method

Participants

A total of 141 participants were recruited for this study as part of a larger, grant-funded study on aggression. Participants were required to be between the ages of 18 and 40, as a way of capturing the age range in which aggression is most pronounced in adulthood (Archer, 2004), and minimizing the likelihood of age-related cognitive decline in the sample (Salthouse, 2009). Participants with qualitative differences in brain function that could contribute to disinhibition, such as specific medical (e.g., Parkinson's disease, epilepsy, traumatic brain injury) and mental health (e.g., history of bipolar disorder, schizophrenia, or pervasive developmental disorder) conditions, or with auditory or visual impairments (e.g., colorblind), were excluded from the parent study and the present study. Demographic information for the full sample is presented in Table 1. Approximately 48% of the sample were "poor" sleepers (<7 hours), above the 34% reported in epidemiological research within the general population (Hafner et al., 2016).

Table 1. Sample Characteristics

	Full Sample (*n*=141)
Age *(M (SD))*	29.32(6.34)
Missing *(n(%))*	2(1.4)
Gender *(n(%))*	
Male	65(46.1)
Female	67(47.5)
Transgender (M to F)	2(1.4)
Transgender (F to M)	1(0.7)
Other	1(0.7)
Missing	5(3.5)
Race *(n(%))*	
Caucasian	79(55.2)
African American	42(29.4)
Asian	9(6.3)

Table 1. (Continued)

American Indian or Alaskan Native	4(2.8)
Other	8(5.6)
Missing	1(.7)
Ethnicity (*n*(*%Hispanic*))	23(16.1)
Missing	8(5.8)
Employment Status *(n(%))*	
Employed	93(65.0)
Unemployed	21(14.7)
Homemaker	7(4.9)
Other (e.g., Retired)	21(14.7)
Missing	1(.7)
Income *(n(%))*	
<$15,000	27(18.9)
$15-30,000	40(28.0)
$30-45,000	27(18.9)
$45-60,000	23(16.1)
$60-75,000	8(5.6)
>$75,000	15(10.5)
Missing	3(2.2)
Recruitment Source *(n(%))*	
Friend/Relative	14(9.8)
Electronic Ads/Flyers	127(89.5)
Other	1(0.7)
Missing	1(0.7)

Procedures

Recruitment. Participants were obtained from a larger study, wherein community-based participants were recruited from Hillsborough County through flyers, the local newspaper, and electronic advertisements on Craigslist and Facebook employment sites (see Appendices I.A.-I.D.). Individuals who indicated interest in participating in the larger study completed a brief screening over the phone assessing the presence of medical or mental health issues or auditory or visual impairments that could serve as exclusionary criteria (See Appendix E.). Exclusionary criteria were the same for the parent study and present study.

The larger study included two sessions (see Figure 1), with Session 2 of primary interest to the current analyses. In Session 1, participants completed measures and interviews assessing individual differences in sleep quality over the month prior to participation (measured via the

Pittsburgh Sleep Quality Index; Buysse et al., 1989), aggressive tendencies, and substance use and addiction, among other variables of interest to the parent study. In this session, participants also underwent a shock threat procedure (Moberg & Curtin, 2009) while completing a version of the Attention Network Task (e.g., Fan, McCandliss, Sommer, Raz, & Posner, 2002) to examine the extent to which the interplay between negative valence (e.g., threat) and cognitive control mechanisms (e.g., attentional control) may relate to aggression proneness (which was operationalized in the laboratory aggression paradigm in Session 2). The mechanisms examined in this task are unrelated to the focus of the current study.

Recruitment for the current sleep study occurred at the end of Session 1, when all participants were invited to participate. To minimize demand characteristics, participants were told that the purpose of the sleep study was to examine how sleep patterns relate to emotions and behaviors. Those who were interested reviewed the informed consent form and signed consent. Then, they completed a brief training regarding how to correctly complete a daily sleep diary for the next 3 days and the correct use of an electronic sleep tracking instrument, the Fitbit Flex device. The participants were not provided account information for the Fitbit Flex, and thus did not have access to sleep tracking information that could confound their reports in the sleep diary.

Sleep Tracking between Session 1 and Session 2. For three days before their participation in Session 2 of the parent study, participants wore the Fitbit Flex and completed a sleep diary through an online survey. Participants were prompted via an automatic text or an email message sent from a study Google Voice account to place the Fitbit Flex in sleep mode each night and complete the diary each morning. Participants who did not have reliable internet access were given paper sleep diaries (n=6).

Overview of Session 2. In Session 2, participants returned the Fitbit Flex trackers and completed tasks relevant to this study (see descriptions in next section). Participants were asked to shower and to avoid drinking alcohol or using drugs or caffeine in the 12 hours prior to the study session. This was to better facilitate electrical scalp activity recording during study tasks and reduce potential confounds.

At the beginning of the lab session, each participant and study confederate (matched on gender and ethnic minority status) drew slips of paper from a cup to learn their purported roles in the later laboratory aggression paradigm. Next, they completed an Emotional Go/No-Go task, followed by a two-part laboratory aggression paradigm. Then, they were debriefed about the true purpose of the study. Debriefing involved explaining the need for deception (in the aggression paradigm), and diffusing any negative emotions the participant was feeling due to involvement in the study. Participants were compensated $10 a day for each sleep diary completed ($15 bonus for completing all days) and $35 for the laboratory session, for a possible total of $80. All study procedures were approved by the university IRB (see Appendix F.).

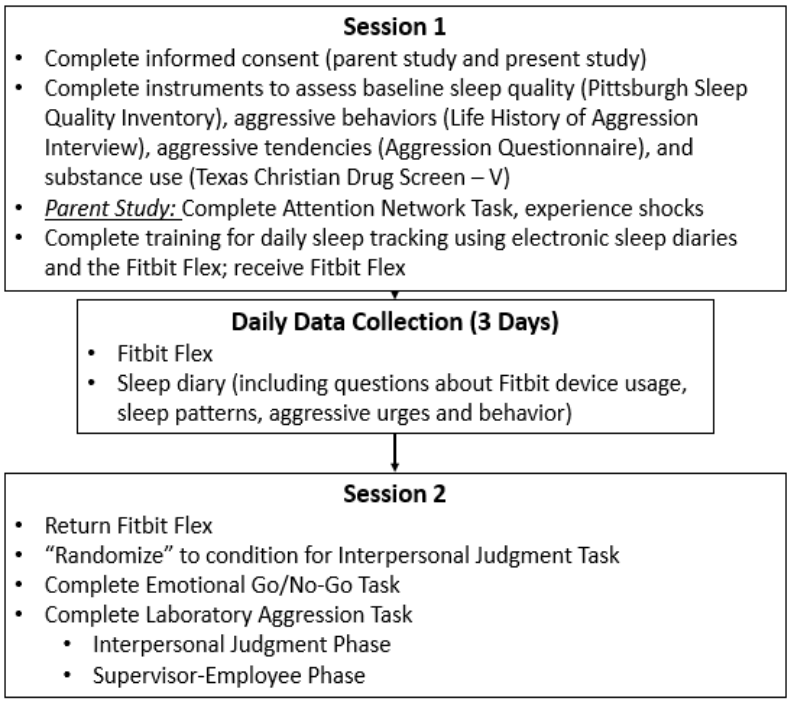


Figure 1. Study procedures

Sleep Tracking and Measurement

Sleep Duration. Objective sleep duration was measured using the Fitbit Flex (http://help.fitbit.com) during a three-day period before Session 2. The three-day time period was selected consistent with research suggesting that the average of three consecutive nights of sleep duration are sufficient to produce moderately stable within-subject assessments of sleep duration (Gaines, Vgontzas, Fernandez-Mendoza, Basta, Pejovic, Fan He, & Bixler, 2015). The Fitbit Flex is worn on the nondominant wrist that can be manually placed in and out of sleep mode to track sleep variables (e.g., time asleep, number and duration of times awakened). There are several reasons why Fitbit devices are an acceptable, more affordable alternative to gold-standard sleep tracking methodologies including wearable actigraphy devices, which enable monitoring of sleep restriction as it occurs in the real world (Montgomery-Downs, Insana, & Bond, 2012). First, research finds that Fitbit devices demonstrate high inter-device sleep

monitoring reliability (see Evenson et al., 2015 for a review). Second, all wearable sleep measurement devices (e.g., including Fitbit and actigraphy) are more likely to misidentify wakefulness as sleep compared to laboratory procedures that identify sleep by tracking brain wave data (e.g., polysomnography), and thus are more likely to *overestimate* sleep duration. However, they provide a noninvasive and more ecologically valid measure of sleep duration compared to laboratory methods such as polysomnography (Montgomery-Downs et al., 2012). The Fitbit Flex has been found to identify differences in sleep quantity between individuals with primary insomnia and sex-matched good sleepers, providing some support for the validity of these devices. Nevertheless, consistent with recommendations in the literature for Fitbit devices and actigraphy (Montgomery-Downs et al., 2012), we used a subjective sleep measure (i.e., sleep diary) to supplement Fitbit Flex data, to derive an average of sleep time over a 3 day period. The procedure for doing so is described and outlined more fully below.

Per recommendations in the sleep literature (e.g., Montgomery-Downs et al., 2012), a daily sleep diary was used to supplement the Fitbit Flex[1] to derive an estimate of daily total sleep time (see Appendix G). Sleep diaries are the gold standard in *subjective* sleep measurement, and are frequently used in clinical settings and trials to identify and monitor sleep disorders and establish diagnoses (Agargun, Tekeoglu, Gunes, Adak, Kara, & Ercan, 1999; Yatani, Studts, Cordova, Carlson, & Okeson, 2002; Fictenberg, Putnam, Mann, Zafonte, & Millard, 2001). Sleep diaries demonstrate high agreement (Kawada, 2008) with actigraphy for wake (77.5%) and sleep (86.1%) in university students. Although sleep diaries among poor sleepers in particular can be susceptible to recall bias (Edinger & Fins, 1995; Putilov, 2015), and tend to *underestimate*

[1] Additional analyses using only sleep diary information yielded similar results to those reported in this manuscript. We elected to report analyses involving Fitbit data corrected via diary data to be consistent with recommendations in the literature to use objective data to assess sleep duration when possible, as well as to utilize a more precise assessment of sleep duration.

total sleep time compared to polysomnography (*Mean Difference* = 54.5, *SD* = 14.1; McCall & McCall, 2012), they yield a reliable and valid index of sleep time (see Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006 for a review). Since the accuracy of sleep reporting can be limited by the participant's insight, emotional wellbeing, and ability to accurately report arousal during the nocturnal period (Sadeh et al., 1995), the literature recommends using objective devices, which more closely approximate results from polysomnography (McCall & McCall, 2012), to capture total sleep time (Buysse et al., 2006; Montgomery-Downs et al., 2012). However, sleep diaries are recommended to cross-validate information obtained from objective sleep devices such as the Fitbit (Boyne, Sherry, Gallagher, Olsen, & Brooks, 2013; Buysse et al., 2006; Vallieres & Morin, 2003; Montgomery-Downs et al., 2012; Werner, Molinari, Guyer, & Jenni, 2008). The sleep diary in this study was based on the National Sleep Foundation Sleep Diary. It inquired about the same sleep variables captured by the Fitbit Flex (e.g., number of minutes asleep, number and duration of times awakened), with additional questions on sleep quality (e.g., number of minutes in bed, time to fall asleep), mental or physical factors that may have disturbed sleep (e.g., drug and alcohol use), extra questions about mood and urges toward aggression, and adherence to the use of the Fitbit tracker.

Sleep Duration Measurement Strategy. Consistent with randomized controlled clinical trials (e.g., Ashworth et al., 2015; van der Zweerde, 2016), we used the sleep diary to supplement objective sleep device information by ascertaining the time to bed, time out of bed, number of times woken up during the night, and times that may have been misinterpreted as sleep (e.g., sitting still for long periods of time, such as in a car or at the movies; Buysse et al., 2006). While sleep tracking data via the sleep diary were available for most subjects for all three nights (*n*=134, 94%), fewer subjects had tracked sleep duration for all three nights via the Fitbit (*n*=108,

76%).[2] For nights in which no Fitbit data was available (e.g., device failure or failure to manually cue sleep/wake modes), we substituted subjective sleep duration data from the sleep diary. Diary data were substituted in 18.5-21.2% of cases each day; across all three days, diary data were used to substitute one day of data for 24 subjects, two days for 6 subjects, and 3 days for 18 subjects. As such, an average of 0.62 days (*SD* = 1.03) were imputed with diary data per subject. Moreover, consistent with procedures used in clinical trials (Ashworth et al., 2015; van der Zweerde, 2016), sleep diary entries that differed from Fitbit data by 30 minutes or more regarding time to sleep or time awoken were used to correct Fitbit sleep duration data. On average, Fitbit data for 30% of entries per night were corrected using this method, and data for 7% of the sample was corrected across all three nights. These corrections were made largely in cases in which the user did not correctly tap the Fitbit into awake mode (e.g., overestimation).[3] After these changes were made, the time of night in which participants fell asleep reported in Fitbit and diary entries differed on average by 13 minutes (*SD*: 11 minutes) per night (range 0-30 minutes), whereas the time in which participants awoke differed by an average of 8.7 minutes (*SD*: 8.5 minutes; range 0-30 minutes). Correlations between diary and Fitbit measures of total sleep time across each night were high (*r*s of .89-.93). Sleep duration in minutes (measured predominantly using the Fitbit, and using diary information when Fitbit data were not available) was the primary measure of sleep used in analyses.

Supplementary Daily Assessments. During the three-day sleep tracking period, participants completed supplementary questions within the sleep diary asking about severity of

[2] The majority of missing data was due to device failure or subject failure to manually cue sleep/wake modes; most subjects had Fitbit data available for at least one night (*n*=123, 86%). Our rates of usable Fitbit data fall in the higher range of studies that use manually activated devices to track sleep (reporting a range of 14-87% of usable sleep data; Lillehei et al., 2015).

[3] As existing studies that correct Fitbit data using the sleep diary do not report the percentage of data corrected using this method, it is unclear if the proportion of data corrected in this study is comparable to rates in the extant literature.

urges to engage in verbal-relational aggression (1 item: “In the last day, have you had an urge or wanted to get back at someone or hurt someone emotionally or socially?”) and physical aggression (1 Item: “In the last day, have you felt an urge or wanted to hurt someone else physically?”). They also completed questions regarding actual engagement in verbal-relational (“Insult or call someone names;” 6 items) and physical aggression (e.g., “Pull or twist someone’s arm or hair;” 9 items). Finally, they completed 10 mood ratings each day from the Negative Affect subscale from the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988; Cronbach’s Alphas ranged from .86-.87 per day).

Preliminary analysis of the collected data uncovered fairly low endorsement of urges to engage in physical aggression (with a range of 5-8 subjects endorsing any urges across days) and actual engagement in physically aggressive behaviors (with a range of 5-8 subjects endorsing at least one type of physical aggression across days). More but still modest endorsement was found for urges to engage in verbal-relational aggression (with a range of 8-19 subjects endorsing any urges across days) and actual engagement in verbal-relational aggression (with a range of 1-12 subjects endorsing at least one of six different types of verbal-relational aggression across days). Thus, reported analyses of three-day hostile or aggressive behaviors rely solely on daily mood ratings of hostility/irritability and *urges* to engage and *actual engagement* in verbal-relational aggression.

Participants also reported their substance use over the prior day to enable examination of relationships between proximal substance use and sleep duration (specifically, to control for the influence of substance use on sleep duration in analyses). Participants indicated their daily quantity of alcohol use (e.g., measured in number of standard drinks; “0,” “1 or 2,” “3 or 4,” “5 or 6,” or “7 to 9”) and whether or not they had used any of several categories of drugs (e.g.,

marijuana, opiates) each day. With regard to alcohol use, 36% of the sample consumed alcohol at least once (with between 23% and 28% endorsing alcohol use each day)[4], and 18% used marijuana[5] at least once (with between 17% and 19% reporting marijuana use each day) over the three-day period. To examine whether alcohol and drug use accounted for relationships that emerged in our analyses, an average alcohol use variable (using the 5-point scale of number of standard drinks above) and a binary marijuana use variable (any use versus no use) across the three-day period were used as covariates in supplementary analyses of study aims.

Laboratory Assessments of Emotional Processing and Response Inhibition

To measure response inhibition processes in conjunction with emotional processing, participants completed the Emotional-Linguistic Go/No-Go task (based on Goldstein et al., 2010), modified for ERP studies (Verona et al., 2012; See Appendix H.) in Session 2. The current study further modified the task to incorporate positively-valenced words, along with the already-validated negative and neutral words. The word list was comprised of 32 emotionally neutral (e.g., umbrella, lamp), 32 negative (e.g., violent, hate), and 32 positive (e.g., mighty, terrific) words selected from the Affective Norms for English Words (Bradley & Lang, 1999) and matched on word length and frequency of use in the English language. The negative words included in this study were selected to be particularly salient for participants with histories of behavioral dysregulation and aggression (see Sprague & Verona, 2010, for validation), since more personally salient, emotionally activating words compared to more general negative words elicit prolonged behavioral effects and reduced go/no-go differentiation on cognitive indices

[4] The majority of subjects reported drinking 1 - 4 standard drinks each day, with a very small number endorsing drinking 5 or more standard drinks each day (a range of 0.8-2.4% of subjects reported this level of use each day).
[5] Subjects who endorsed drug use over the three-day period largely endorsed marijuana use; less than 5 subjects endorsed any other illicit drug use (e.g., ecstasy, opioids). Due to low endorsement, and since marijuana versus other drugs have different mechanisms of action (and are often examined separately in the substance use literature), we included only marijuana use in study analyses.

(perhaps due to more heavily depleting inhibitory processing resources; Sprague & Verona, 2010; Verona & Bresin, 2015).

The Emotional Go/No-Go task requires utilizing inhibitory control to respond to *features* of the word (normal vs. italicized font) rather than the word *content* (negative vs. neutral vs. positive). Participants were instructed to quickly but accurately press a button to words appearing in a normal font (Go trial) and to inhibit this response to words in italicized font (No-Go trial). The task included 20 practice trials with neutral words not used elsewhere in the task to ensure that participants understand the task instructions, and contained 6 blocks for each of the three emotion word categories, for a total of 18 blocks. Words were randomized within each emotional category, and the sequence of emotional category blocks was counterbalanced across participants. Specific words selected for No-Go trials differed across blocks, with rest period provided between blocks. To establish a prepotent response to the task, each word category block was comprised of fewer No-Go trials (9 per block, for a total of 54 No-Go trials per condition across blocks) than Go trials (23 per block, with a total of 414 trials across blocks). Each word trial was presented for 1400 ms, then followed by a 750-1000 intertrial interval. This task took approximately 20 minutes to complete.

Laboratory Assessment of Aggression

The aggression paradigm in Session 2 was based on a task previously developed by Buss (1961) and modified in our laboratory (Verona, Sadeh, & Curtin, 2009). At the very beginning of Session 2, prior to the Emotional Go/No-Go task, the participant and a lab confederate matched on gender and minority status were introduced to each other. They drew slips of paper from a cup to determine their study roles across two phases: an interpersonal judgment phase and employee-supervisor phase. The role assignment process was rigged so that the real participant

was always assigned to be the "judgee" in the interpersonal judgment phase, where they complete an essay about their personal qualities (which will be negatively evaluated by the confederate), and then to be the "supervisor" in the subsequent employee-supervisor (providing "feedback" to the confederate). During the initial introduction and role assignment, the confederate followed a specific script and set of behavioral instructions to portray him or herself as annoying and somewhat rude to the study participant, to increase the believability of their behavior in the later interpersonal judgment phase (where they provided negative judgments). The participant and confederate did not have direct contact at any other point during the experiment.

The interpersonal-judgment phase began following the completion of the Emotional Go/No-Go Task and was meant to induce provocation to increase potential of aggressive behavior during the employee-supervisor task. In this phase, the participant was given 5-7 minutes to write an essay about his or her personal qualities. The confederate reviewed the essay as "poor" on an essay feedback form that was standardized across participants, providing negative ratings on dimensions related to the essay and on scales such as participant attractiveness, friendliness, and likeability. The confederate also wrote a comment describing the participant's essay as "defensive" and "uninteresting." The confederate purportedly completed this evaluation of the participant essay within approximately two minutes, despite being asked to be "thoughtful and considerate" in their responses. Research staff also commented within earshot of the participant that this was a "fast" turnaround of this portion of the task. A member of the research staff then appeared to accidentally leave behind this feedback form for the participant to view to induce anger and hostility towards the confederate. 96.4% of participants read the

feedback form; participants who did not read the form, and thus were presumably not provoked, were excluded from analyses of this task (n = 6).

Next, in the employee-supervisor phase (see Appendix I.), aggressive behavior toward the confederate was measured. The participant was asked to provide "supervisor" feedback on the correctness of responses by the confederate "employee." Participants were told that the employee would perform a recall task in a separate room. Participants were shown the employee's responses on a computer monitor and told to provide feedback, as quickly as possible, to the correctness of responses using a multi-button feedback box. The box was comprised of a range of possible responses. One button was designated as "correct," to be pressed when the confederate employee responded correctly. When the employee provided an incorrect response, the supervisor decided whether to press a button to provide response feedback but no shock (0), or any of 7 buttons representing increasing shock intensities (1-7). Notably, participants had been familiarized with the intensity of the shocks during the shock procedure during Session 1 (the Attention Network Task), which increased the credibility of the task and the likelihood that participants would believe they were actually shocking the confederate. However, no actual shocks were administered to the confederate employee during the experiment. To avoid making the participant feel that he or she was unduly hurting the employee, no auditory or visual feedback was conveyed from the employee when the shocks were administered.

The employee-supervisor task was comprised of four task blocks, with 10 trials per block. Approximately 40% of the trials across blocks involved an incorrect response from the employee (confederate), and required a shock button or a no-shock response from the supervisor (participant). Consistent with prior research (Verona et al., 2009), aggressive behavior was

operationalized as the mean level of shock participants administered to the confederates across the whole task (level and increase in shock across blocks). Average shock intensity was computed by summing the shock levels administered, and then dividing by the total number of trials in which the participant administered a shock. This approach enabled inclusion of an index of average shock intensity in analyses that adjusted for participant differences in the number of shocks administered (e.g., some participants incorrectly shocked on 'Correct' trials,[6] some participants marked 'Incorrect' trials as 'Correct,' and some participants shocked more frequently than others).[7] When participants chose the no-shock feedback option for all confederate incorrect responses across the task (n = 30; 24.2%), their average shock intensity was recorded as "0." Across blocks, participants shocked at an average level of 2.33 (*SD*=2.06, Range: 0-7). Performance on this task relates to self-reported tendencies toward hostility and aggression (Verona et al., 2002).

Changes in Affect. To measure changes in affect across the experiment, participants completed the PANAS (1) at the beginning of the laboratory session, (2) following the Emotional Go/No-Go Task, (3) following the provocation in the interpersonal judgement phase,

[6] A large proportion of participants incorrectly shocked participants once (61.6%) or twice (11.2%) over the course of the whole experiment. A very small number of subjects (*n*=6) incorrectly shocked three or more times (at incorrect shock levels: 0-5) across the course of the experiment. As incorrect shocking appeared to be a commonly occurring participant behavior, and appeared to have largely been administered due to participant error (e.g., when participants misinterpreted confederate correct responses as incorrect, but responded appropriately on the vast majority of remaining trials), we only included shocks administered on 'Correct' trials in our calculation of average shock intensity.

[7] We also considered the possibility that patterns of aggression may vary across subjects. Therefore, we ran supplemental analyses with shock frequency instead of average shock intensity (e.g., to capture instances where someone is aggressive by choosing more frequent shocks that may vary in severity levels), computed as the number of shocks (versus choice of the no-shock option) administered per block. We also used shock severity in supplemental analyses (e.g., to capture instances where others may shock less frequently, but at higher levels), computed as the total summed shock severity within each block. In both analyses, results were similar in size and direction of effects to results examining average shock intensity across block. Thus, to be consistent with operationalizations of aggression using this paradigm in other studies (e.g., Verona et al., 2009), we report results involving average shock intensity across blocks in the present study.

and (4) following completion of the employee-supervisor (aggression) phase. Participants rated how they were feeling "right now" (in the present moment) across 20 mood adjectives. The PANAS is comprised of a 10-item Positive Affect subscale (e.g., "excited," "strong") and a 10-item Negative Affect subscale (e.g., "hostile," "scared"), and items were rated using a 5-point Likert scale (ranging from 1=*very slightly or none at all* to 5= *extremely*). When the PANAS is administered using short-term instructions, it has been found to be sensitive to fluctuations in mood over short time periods (Watson et al., 1988). The Negative Affect scale correlates highly with measures of distress and depressive and anxiety symptoms (Watson et al., 1988). PANAS scores for participants who read the essay feedback in the Interpersonal Judgment Task and did not deduce the purpose of the paradigm (i.e., received the mood manipulation and believed that it was real) were included in analyses.

Debriefing and Effectiveness of Deception. To assess participants' experiences and perceptions of the aggression task, particularly the effectiveness of the deception procedure, participants completed a brief self-report questionnaire and a short interview with study staff. The questionnaire (See Appendix J.) was administered immediately following the lab aggression task. The participant answered questions regarding his/her experience with different aspects of the paradigm, including perceptions of the confederate that may contribute to aggressive behavior (e.g., "If you interacted with any other participants as part of this experiment, please give your reactions to those participants") rated on a 7-point Likert Scale assessing perceptions spanning several dimensions (e.g., ranging from "Immature" to "Mature"). Participants also answered questions on a 10-point Likert Scale assessing impressions of the employee's performance (e.g., "How well did you think you could have done on the digit span task that the other participant (employee) worked on?") and explanations for their use of shock feedback (e.g.,

"To what extent did you choose higher shock levels because you were upset at the employee or about the employee's performance?").

The subsequent interview (See Appendix K.) was comprised of 7 open-ended questions regarding the participant's experience of the interpersonal judgment task (e.g., "Would you have rather been in the rater role?") and the employee-supervisor task (e.g., "Did you ever become annoyed or distressed at the employee's performance? When did this happen? What did you do when you felt annoyed at him/her?"), particularly with regard to factors driving aggressive behavior (or lack thereof). Effectiveness of the deception manipulation was assessed by asking open ended questions from participants regarding what they thought the purpose of the study was (e.g., "What did you think we were trying to investigate in this study?"), about their experience working with the confederate, and whether anything seemed "off or unusual" to them during the task. Data for 13 participants (9%) were excluded from aggression analyses due to lack of deception or other anomalies (e.g., deduced purpose of paradigm or identified confederate as study staff: *n*=7, 5%; did not read negative essay feedback: *n*=6, 4%).

Clinical and Trait Measures

Several clinical and trait measures were administered to gain additional information about relevant constructs. All measures were administered in Session 1 of the parent study. Specifically, we collected information to ascertain participants' typical sleep quality during the month prior to the study and substance use disorder symptoms that could confound experimental findings. We also collected information about participants' aggression proneness and lifetime engagement in aggressive behaviors to obtain endorsements of real life aggression, beyond participants' performance on the laboratory aggression paradigm.

Demographics. Participants completed a brief demographics form (see Appendix L.) regarding age, gender, race/ethnicity, income, and substance use habits (e.g., need for smoke breaks).

Last Month Sleep Quality. The Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1989) was administered to assess sleep quality and duration over the month prior to Session 1. It is a 19-item self-report measure with items rated on scales ranging from 0 to 3 (with 3 indicating worse functioning). The PSQI yields a total score summed across seven subscales (sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction). It provides total score cut-offs to characterize the degree to which sleep quality and disturbances are experienced as impairing (possible range of scores 0 to 21; ≤ 5 = normal, 6-10 = moderately impaired, ≥ 11 = severely impaired). The PSQI has been found to have good convergent and divergent validity with assessments of insomnia and sleep apnea (Buysse et al., 1989), adequately differentiates between good and bad sleepers (Grandner, Kripke, Yoon, & Youngs, 2006) and demonstrates good reliability in community populations (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Buysse et al., 1989; Lund et al., 2010). Total score on the PSQI was used to describe sleep quality in the sample for the month prior to study participation.

Aggression Proneness and Lifetime Violent Acts. Measures of aggression proneness and violence history were administered to examine broader associations between sleep restriction with self-reported aggressive tendencies and behavior. First, the Aggression Questionnaire was used to assess self-reported *aggression proneness* (AQ; Buss & Warren, 2000). It is one of the most widely used self-report aggression measures, consisting of 34 items across 5 scales measuring Physical Aggression (9 items; e.g., “Given enough provocation, I may hit another

person;" Cronbach's Alpha=.87), Verbal Aggression (5 items; e.g., "When people annoy me, I may tell them what I think of them;" Cronbach's Alpha=.74), Anger (7 items; e.g., "I sometimes feel like a powder keg ready to explode;" Cronbach's Alpha=.84), indirect aggression (5 items; e.g., "When people are bossy, I take my time doing what they want, just to show them;" Cronbach's Alpha=.62), and Hostility (8 items; "At times I feel I have gotten a raw deal out of life;" Cronbach's Alpha=.79). Participants rated each statement using a 5-point Likert scale (1= "extremely uncharacteristic of me" to 5= "extremely characteristic of me"). The AQ total score and subscales demonstrate good reliability when used with college students and young adults in the community (Buss & Warren, 2000). Scores on the AQ correlate with real-world aggressive behaviors (Bettencourt, Talley, Benjamin, & Valentine, 2006).

Second, self-reported lifetime number of *aggressive acts* were measured by the Life History of Aggression interview (LHA; Brown et al., 1982). Participants reported the frequency that they engaged in 11 types of aggressive and antisocial behaviors since the age of 13, and interviewers rated the reports using a 5-point scale (ranging from 0=*no events* to 5= *so many events they can't be counted*). The LHA is comprised of 3 subscales that are summed to create a total score of lifetime aggressive behaviors (Coccaro, Berman, & Kavoussi, 1997). The Aggression subscale includes five items measuring aggressive behavior, including temper tantrums, physical fights, verbal fights, assaults on persons and assaults on property (Cronbach's Alpha=.72). The Self-Directed Aggression subscale includes two items measuring non-suicidal self-injurious behavior and suicide attempts (inter-item r=.21). The Antisocial Behavior subscale is comprised of four items describing disciplinary action at school, problems with supervisors at work, and antisocial behavior with or without police involvement (Cronbach's Alpha=.63). The LHA demonstrates good inter-rater agreement, internal consistency, and test-retest reliability in

clinical and community samples (Coccaro et al., 1997). Scores on the Aggression subscale relate to aggressive behavior in the laboratory, and to biological variables (e.g., serotonin; Coccaro, Berman, Kavoussi, & Hauger, 1996) that are theoretically linked with aggression (Berman, McCloskey, Fanning, Schumacher, & Coccaro, 2009).

Substance Use Problems. Symptoms of alcohol (11 items; e.g., tolerance) and drug use disorder [8] (11 items; e.g., withdrawal) over the prior year were assessed using the MINI International Neuropsychiatric Interview for the DSM-5 (MINI 7.0; Sheehan, 1998). Presence of symptoms (threshold vs non-threshold) in the last year were rated for drug and alcohol use disorder separately by clinical psychology doctoral students who had been thoroughly trained by a Ph.D.-level licensed clinical psychologist. The MINI demonstrates good validity and reliability in assessing substance use disorders (Sheehan, 1998). In this study, alcohol and drug use symptom counts were used as covariates in supplemental analyses to control for chronic substance abuse tendencies.

Internalizing Symptoms. The presence of symptoms of overall depression was measured in two way. First, the DSM-5 diagnostic criteria for major depressive disorder were assessed via the MINI 7.0, in terms of the number of depressive symptoms experienced in past 2 weeks (9 items; e.g., threshold vs not of persistent depressed mood). Second, symptoms of depressive anhedonia in the past two weeks were measured using the Mood and Anxiety Symptom Questionnaire Anhedonic Depression scale (22 items; e.g., "I feel like nothing is very enjoyable"), rated on a 5-point Likert scale ranging from 1 ("not at all") to 5 ("extremely") (Cronbach's Alpha = .89). The MINI and MASQ demonstrate good validity and reliability in assessing recent depressive symptoms (Sheehan, 1998; Watson et al., 1995). Symptom counts for

[8] During interviews, the most problematic drug endorsed by subjects (e.g., the most interfering and/or distressing) was used as the index drug on which drug dependence symptom ratings were based.

the MINI and the total score on the MASQ Anhedonic Depression Scale were used as covariates in supplemental analyses to control for depressive symptoms.

Cognitive symptoms of *anxiety* were measured using the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is comprised of 16 items measured on a 5-point Likert scale ranging from 1 ("not at all typical of me") to 5 ("very typical of me") (e.g., "Once I start worrying, I cannot stop;" Cronbach's Alpha=.94). Physiological symptoms of anxiety were measured using the Anxious Arousal subscale of the MASQ (Watson et al., 1995). This MASQ subscale is comprised of 17 items rated on a 5-point Likert scale ranging from 1 ("not at all") to 5 ("extremely") (e.g., "I get afraid that I'm going to die;" Cronbach's Alpha=.86). The PSWQ and MASQ show strong psychometric properties in the assessment of anxiety symptoms (Meyer et al.,1990; Watson et al., 1995). Total scores for the PSWQ and the MASQ Anxious Arousal subscale were used as covariates in supplemental analyses to control for anxiety symptoms.

Physiological Data Acquisition

Data Collection. During the Emotional Go/No-Go Task, ERPs were recorded using Electrical Geodesics system hydrocel 64-channel sensor nets and amplifiers (EGI, Eugene, OR). Nets were placed on participants' heads using known anatomical landmarks (e.g., mastoids, nasion, inion). Consistent with other research examining emotion and inhibitory control (e.g. Verona et al., 2012), electrodes were selected from the frontocentral (3 electrodes) and parietal (4 electrodes) midline scalp sites (See Appendix M.). Analog signals were digitized online at 250 Hz and bandpass-filtered (.15-200 Hz), and amplified using Net Amps amplifiers. Electrodes underneath the eyes imbedded in the nets were used to record eye movements. Impedances were kept below 50 kΩ. Stimuli were presented on a flat-panel display using E-Prime software (PST

Inc., Pittsburgh, PA), and behavioral responses were collected with a 4-key keypad that interfaces to E-Prime.

Offline Data Processing. Offline data processing was completed in Netstation software. Data were re-referenced to average head and epoched 200 ms before and 800 ms after stimulus onset, and a 0.10 to 30 Hz filter was applied with a baseline correction. Trials with artifact deflections greater than 140 mV in absolute value, or with eye movements greater than 55 mV in absolute value, were discarded, with a moving average of 80 ms. Channel replacement was performed for channels where more than 20% of trials were discarded. Following data processing, an average of 80% of trials were retained for Go conditions (Range 9.3-98.8%, 94-427 trials) and 77% for No-Go conditions (Range: 10.42-100%, 15-144 trials). Next, participants with less than 50% usable trials for any condition were excluded from analyses, to ensure that the minimum number of trials needed for a statistically stable (e.g., internally consistent) N2 (e.g., 20 trials) and P3 (e.g., 14 trials) were available within each condition (Rietdijk, Franken, & Thuri, 2014). Following these procedures, a total of 111 subjects were retained in ERP analyses[9]. Average ERP waveforms were calculated across trials within each condition. A 30 Hz Butterworth filter was applied with a baseline correction. The P3 component was defined as the adaptive mean peak amplitude (+/-50ms) within 400 to 600 ms post-stimuli at frontal or parietal sites. The N2 was defined as the adaptive mean peak amplitude (+/- 10ms) within 200 to 350 ms post-stimuli at parietal sites.

We also examined behavioral indices of response inhibition in additional analyses of primary aims. Commission errors were defined as the number of erroneous "go" responses made

[9] Subjects excluded from ERP analyses did not differ from included subjects on demographic variables or primary study variables (e.g., marijuana or alcohol use, sleep duration, aggressive tendencies and behavior, shock intensity).

during No-Go trials, and reaction time was calculated by averaging across reaction times across Go trials. All calculations were computed within each emotion word category.

Data Analysis

Prior to testing study hypotheses, data were screened in SPSS for violations of assumptions of normality by inspecting variable skewness and kurtosis, results of the Kolmogorov-Smirnov Test (Massey, 1951), and outliers. Since regression-based methods assume the dependent variable is normally distributed (Cohen, Cohen, West, & Aiken, 2002), a log transformation was applied for non-normal dependent variables (including the Self-Directed Aggression subscale of the LHA, verbal-relational aggressive urges and behaviors on the sleep diary and substance use symptom variables) to better meet the underlying assumptions of these models (Keene, 1995). Sample sizes varied across analyses due to validity of data across tasks (see Figure 2).

To test Aim 1 (e.g., relationship between sleep duration and aggressive behavior), a repeated measures ANOVA (RMANOVA) was conducted to examine the effect of sleep duration (average minutes of sleep duration across 3 days) on average shock intensity across blocks of the aggression paradigm (**Hypothesis 1**). Secondary correlational analyses were also conducted to examine the association between sleep duration and self-reported aggressive tendencies and lifetime aggressive acts, as measured by questionnaires/interviews in Session 1, and verbal-relational aggressive urges and behaviors reported during the three-day sleep tracking period.

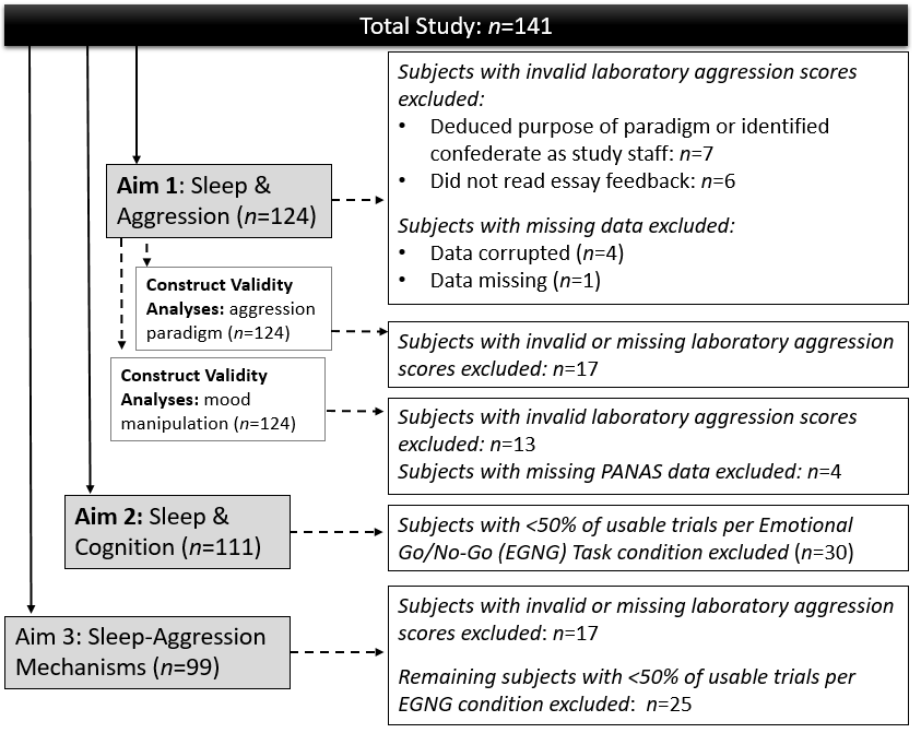


Figure 2. Sample size across analyses

Next, prior to testing Aim 2 (e.g., relationship between sleep duration, response inhibition, and emotional processing), a series of RMANOVA analyses were conducted to examine the effects of electrode site (frontal, frontocentral, parietal) on N2 and P3 amplitude to verify the specificity and consistency of frontal and parietal effects suggested in the literature (Carretie et al., 2008; Bekker et al., 2004). To test Aim 2, mixed-model RMANOVA analyses

were used to examine the separate and interactive effects of sleep duration, Emotional Go/No-Go Task trial type (Go versus No-Go; **Hypothesis 2**) and emotion word category (Negative versus Neutral versus Positive; **Hypothesis 3**) on the amplitude of the N2 and P3 at frontocentral and parietal regions, to be consistent with prior research (Bekker et al., 2004). We were particularly interested in a Sleep Duration x Emotion Word Category (3) x Trial Type (2) interaction in regard to the frontocentral N2 and P3 amplitudes (**Hypothesis 4**). Significant interaction effects were decomposed and interpreted within different levels of sleep duration using simple slopes (with relationships of interest examined at levels of average sleep duration, and one standard deviation above and below average duration; Aiken & West, 1991), as well as with follow-up analyses using correlations. For behavioral measures, we used RMANOVAs to examine the separate and interactive effects of sleep duration and emotion word category on number of commission errors (**Hypotheses 5 and 6**) and on reaction time on "go" trials (**Hypothesis 7**).

Finally, to test whether emotional processing and response inhibition explained variance in the relationship between sleep and aggression (Aim 3), we used path analytic modeling in Mplus[10]. In separate analyses, we examined whether the No-Go P3 or N2 (response inhibition: no-go versus go; **Hypothesis 8**), emotion P3 (emotional processing: negative versus neutral; **Hypothesis 9**) or No-Go P3 in the negative word condition specifically (response inhibition modulated by emotion category; **Hypothesis 10**) explained the sleep duration-aggression relationship. In order to test the response inhibition components in Hypotheses 8 and 10, the No-Go P3 and N2 were calculated by computing the difference in N2 or P3 amplitude between Go

[10] We did not use multiple imputation nor a maximum likelihood estimator to allow use of all subjects in our Aim 3 analyses, as the subjects missing data on the Aggression Paradigm and the Emotional Go/No-Go tasks were not missing at random (e.g., subjects were missing systematically due to not receiving the aggression manipulation/falling asleep or were removed for having less than 50 percent of trials), which is an assumption of these methods (Sterne et al., 2009).

and No-Go conditions. The recommended approach is to resample the collected data 10,000 times to provide a percentile-based and bias-corrected confidence interval for the indirect effect (Preacher & Hayes, 2004; Shrout & Bolger, 2002; Zhao, Lynch, & Chen, 2010). A significant indirect effect was considered as present when zero was not contained in its confidence interval (MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008).

Supplemental Analyses. Several supplemental analyses were conducted to evaluate potential confounds of results. First, as literature suggests that males are generally more aggressive than females (Staniloiu & Markowitsch, 2012), and substance use is commonly implicated in both aggressive behavior (Boles & Miotto, 2003) and poor sleep (Brower, 2003), it is possible that any sleep-aggression relationship observed may be better accounted for by gender or substance use. However, since the primary focus of this study was to examine the mechanisms that link sleep and aggression, and gender differences and relationships with substance use were not primary aims of this study, we did not power this study to examine these potential differences. Nonetheless, we included substance abuse disorder symptoms, three-day reports of alcohol and cannabis use and gender as covariates in separate supplemental analyses of the primary aims to preliminarily examine whether these variables may account for variance contributed to aggression by poor sleep. We also included sleep measurement type (binary variable, coded as any subjective diary data used versus only objective Fitbit data used) and depressive and anxiety symptoms as covariates in analyses to ensure that differences in sleep measurement methods and internalizing symptoms did not account for our findings.

Power Analysis. A power analysis using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) indicated that 100 participants would be necessary for .8 power to detect a medium effect (*f*=.25; Cohen, 1988) for a 3-way interaction in a mixed-model repeated measures analysis of

variance. According to guidelines for mediation analyses, assuming .8 power and an alpha of .05, 78 participants were required to detect a medium effect for bootstrap tests of mediating effects (Fritz & MacKinnon, 2007).

Results

Sample Descriptives

Descriptive information for measures of aggression and sleep and intercorrelations across study measures are displayed in Table 2. Across the three nights prior to Session 2 in the present study, participants slept 6 hours and 59 minutes on average (*SD*=79 minutes, Range: 191-510 minutes). This corresponded well (r =.41, p<.001) to Session 1 reports on the PSQI of last month average number of hours[11] slept per night (*M*=6 hours and 34 minutes, *SD*=96 minutes, Range: 2-10 hours or 120-600 minutes), suggesting that our measure of recent sleep was somewhat representative of participants' more prolonged sleep patterns.

Per our 3-day measure of sleep duration, approximately 46.8% of participants slept less than 7 hours on average. This average measure of sleep duration was consistent with the proportion of participants who reported sleeping less than 7 hours on average across the prior month (49.6%).

On average, participants endorsed low-to-moderate problems with sleep quality on the PSQI (M=6.7, SD=3.41, Range=1-17), which was comparable to levels of endorsement in community samples (e.g., M=6.3, SD=3.4; Buysse et al., 2010). Approximately 48% of the sample endorsed moderate (36%) to severe (12%) problems with sleep quality on the PSQI.

[11] The PSQI only requests information about sleep duration in terms of total hours, and is thus not as fine-grained as our measure of sleep duration. Moreover, it is solely measured via self-report. However, despite these differences, it corresponds well with our measure of sleep duration in this study.

Table 2. Associations between sleep duration, aggressive tendencies, lifetime history of aggressive behavior, and three-day reports of substance use and hostile urges.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Sleep Duration (Minutes)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Aggressive Tendencies (AQ)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
2. Anger	-0.20*	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
3. Hostility	-0.22**	0.56***	--														
4. Verbal Aggression	-0.23**	0.63***	0.53***	--	--	--	--	--	--	--	--	--	--	--	--	--	--
5. Physical Aggression	-0.20*	0.47***	0.45***	0.55***	--	--	--	--	--	--	--	--	--	--	--	--	--
6. Indirect Aggression	-0.17*	0.54***	0.49***	0.58***	0.58***	--	--	--	--	--	--	--	--	--	--	--	--
7. Total Aggression	-0.26**	0.82***	0.82***	0.78***	0.80***	0.77***	--	--	--	--	--	--	--	--	--	--	--
Lifetime History of Aggressive Behavior (LHA)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
8. Aggression	-0.12	0.28**	0.33***	0.31***	0.49***	0.38***	0.45***	--	--	--	--	--	--	--	--	--	--
9. Consequences	-0.11	0.17*	0.27**	0.24**	0.48***	0.21*	0.35***	0.70***	--								
10. Self-Directed Violence	-0.07	0.32***	0.26**	0.07	0.19*	0.15	0.27**	0.33***	0.26**	--	--	--	--	--	--	--	--
Substance Use Symptoms(MINI)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
11. Alcohol Use Disorder Symptoms	-0.14	0.02	0.09	0.16	0.10	0.13	0.10	0.14	0.18*	-0.08	--	--	--	--	--	--	--
12. Drug Use Disorder Symptoms	-0.24**	0.25**	0.25**	0.34**	0.28**	0.36***	0.35***	0.28**	0.40***	0.16	0.20*	--	--	--	--	--	--
3-Day Substance Use (Sleep Diary)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

Table 2. (Continued)

13. Alcohol Use	-0.02	0.01	-0.10	0.01	0.02	0.0	-0.03	0.01	0.06	0.06	0.35***	0.24**	--	--	--	--	--
14. Marijuana Use	-0.23*	.22*	0.10	0.27**	-.18	0.22*	0.23*	0.28**	0.17	0.14	0.53***	0.20*					
3-Day Mood & Urges (Sleep Diary)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
15. Hostile Mood	-0.12	0.24**	0.27**	0.32**	0.32***	0.30**	0.34***	0.21*	0.23*	0.29**	0.08	0.14	0.13	-0.01	--	--	--
16. Irritable Mood	-0.06	0.35***	0.30**	0.36***	0.30**	0.34***	0.40***	0.26**	0.20*	0.16	0.11	0.16	0.07	0.01	0.66***	--	--
17. Verbal-Relational Aggression Urges	0.01	0.23**	0.25**	0.31**	0.10	0.22*	0.26**	0.10	0.00	0.11	0.12	0.03	-0.07	0.12	0.23*	0.26**	--
18. Verbal-Relational Acts	-.11	.42***	.34***	.44***	.46***	.43***	.53***	.32***	.23**	.12	.12	.26**	.18*	.01	.35***	.40***	.44***
M	419.32	13.54	17.67	13.39	14.94	13.32	72.39	9.24	4.31	0.15	0.22	0.20	0.19	0.20	1.24	1.73	0.32
SD	79.65	5.70	6.04	4.11	6.27	4.11	20.51	4.92	3.15	0.26	0.27	0.31	0.25	0.31	0.40	0.68	0.05
Range	191.33-610	7-32	8-35	5-25	8-40	8-30	34-152	0-23	0-12	0-.95	0-1.04	0-1.00	0-0.90	0-1.00	1-3.00	1-4.00	0.3-0.56

Note. AQ = Aggression Questionnaire. LHA = Life History of Aggression Interview. MINI = MINI International Neuropsychiatric Interview. *p<.05, **p<.01, ***p<.001. n=143

Aggression Paradigm Validity

Provocation. A one-way RMANOVA was conducted to examine the effect of time (4 time points) on average PANAS ratings of affect across experimental Session 2 (n=124). We expected that change would not simply be linear, since our mood induction, EGNG paradigm, aggression task, and aftermath of the aggression task would be expected to differentially influence mood across the course of the session. Thus, across analyses, rather than utilizing the omnibus F test with post-hoc tests (which would examine whether affect differed at specific time points, but would not examine the *trajectory* of change across the session), we relied on a priori polynomial orthogonal contrasts to elucidate the pattern of change in affect across the course of the session. A linear contrast would represent increased or decreased affect across the session, whereas a quadratic contrast would indicate that affect

increased to a certain point before decreasing (or vice versa). A cubic contrast would indicate that affect decreased to a certain point, then increased before decreasing (or vice versa).

As expected given our session design, there was a significant cubic ($F(1,123) = 13.05$, $p<.001$, $\eta_p^2=.10$) effect of time on negative affect, such that negative affect decreased slightly following the start of the session, increased sharply following the mood induction, and declined at the end of the aggression paradigm (see Figure 3). Most relevant to our aims, ratings of hostility (linear time: $F(1,123) = 6.06$, $p<.05$, $\eta_p^2=.05$) increased across the session, and ratings of irritability peaked in the middle of the session, declining at the end of the aggression paradigm (quadratic time: $F(1,123) = 5.00$, $p<.05$, $\eta_p^2=.04$). This pattern for hostility, $t(123)=4.51$, $p<.001$, $d=0.40$, and irritability ratings, $t(123)=3.22$, $p<.01$, $d=0.29$) was particularly apparent in changes from before to after the provocation induction (i.e., interpersonal judgment essay evaluation; See Figure 4). In contrast, ratings of nervousness decreased over the course of the session (linear; $F(1,123) = 6.37$, $p<.05$, $\eta_p^2=.05$), whereas ratings for other aspects of negative affect (e.g., scared, ashamed) did not significantly change over time. Collectively these results indicate that the mood manipulation induced negative affect, with some specificity for angry affect.[12]

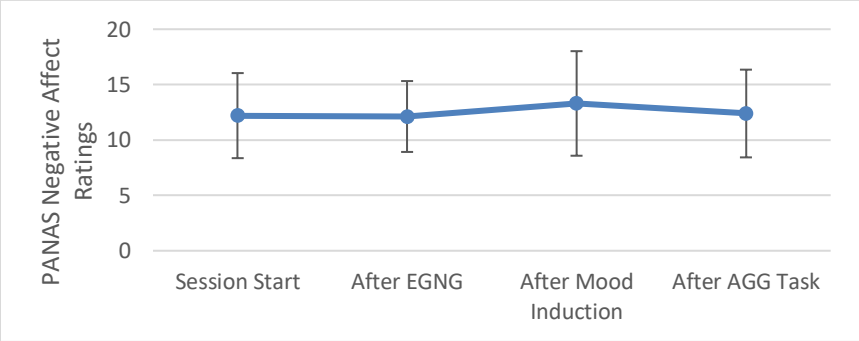


Figure 3. Average PANAS Negative Affect scores across Session 2

[12] Supplemental RMANOVA analyses were conducted to examine whether sleep duration was associated with differences in negative affect across the session. There was a significant between-subjects effect of sleep duration on negative affect ratings ($F(1,124) = 6.00$, $p<.05$, $\eta p2=.05$), such that less sleep was associated with greater ratings of negative affect overall.

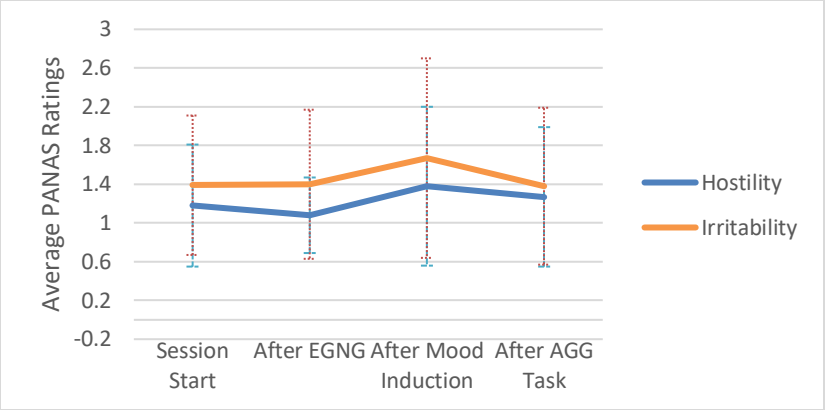


Figure 4. Average PANAS Hostility and Irritability scores across Session 2

Construct Validity of the Aggression Paradigm. We examined the construct validity of our measure of laboratory aggression by correlating trait measures of aggressivity and history of violence with average shock intensity (n=124). As expected, average shock intensity was significantly related to AQ total score (r=0.25, p<.01) and the anger (r=0.19, p<.05), verbal (r=0.20, p<.05), physical (r=0.30, p<.01), and indirect (r=0.21, p<.05) aggression subscales, with these effects similar in size to those reported in other studies using this paradigm (about moderate in size; Verona et al., 2009). However, it was not related to the hostility subscale of the AQ, or lifetime frequency of aggressive acts, as measured by the LHA. Moreover, in the post-study questionnaire, average shock intensity was highly associated with participants' reports of selecting higher shock levels due to being upset at the confederate (r=0.67, p<.001), as well as reports of shocking to encourage the confederate to improve his/her performance (r=0.70, p<.001). Average shock intensity was also associated with more negative perceptions of the confederate's attractiveness (r =-0.35, p<.001), competence (r=-0.24, p<.05), friendliness (r=-0.17, p=.06), maturity (r=-0,19, p<.05), and likeability (r=-0.19, p<.05). For the most part, shock intensity correlated to a smaller degree with perceptions of laboratory personnel (e.g., helpfulness, efficiency, trustworthiness; rs .001-.10; ps>05), the laboratory session (e.g., useful,

boring; *r*s -.12-.10; *ps*>.05; difficult: *r*=.19, *p*<.05) and impressions of the confederate's performance on the employee-supervisor task (*r*=.02, *p*>.05). Thus, choice of shocks seemed to relate more to hostility toward the confederate than negative affect more generally. Taken together, these results confirm that shock intensity selections during the aggression procedure were related to aggressive tendencies in real life and influenced by hostile intentions and/or being upset at the confederate/employee during the experiment. These data support the construct validity of the aggression paradigm in this study.

Aim 1: Linking Sleep Duration with Aggression

Sleep Relations with Laboratory Aggression. A Sleep Duration x Task Block (1-4) RMANOVA was conducted on average shock intensity scores (*n*=124). In so doing, we examined Block effects in terms of a priori polynomial contrasts to elucidate the pattern of change in aggression across the course of the task. A linear Block effect would represent increased or decreased aggression across the session, whereas a quadratic Block effect would indicate that aggression increased to a certain point before decreasing (or vice versa). Research finds a typical increase in lab aggression across time (Goldstein et al., 1975), and research using this aggression paradigm has found significant linear and quadratic Block effects (e.g., Verona & Kilmer, 2007).

The main effect of sleep duration on overall aggression trended towards significance, such that sleep duration related negatively with average shock intensity (*r*=-.15, *p*=.08). Additionally, the quadratic effect of Block, and the Sleep Duration x quadratic Block interaction, were significant (see Table 3).

Inspection of zero-order relationships with sleep duration and aggression indicated that overall sleep duration was inversely associated with aggression, with this relationship peaking in

the middle of the paradigm and declining towards the end of the task (Block 1 r= -.10, p>.05, Block 2 r= -.2, p<.01, Block 3 r= -.23, p<.01, Block 4 r= -.11, p>.05).

Table 3. RMANOVA Effects on Average Shock Intensity.

Predictor	***F***	**df**	***p***	η_p^2
Block (Linear)	0.80	(1,121)	.37	.01
Block (Quadratic)	6.45	(1,121)	<.05	.05
Block (Cubic)	0.32	(1,121)	.57	.00
Sleep Duration x Linear Block	0.23	(1,121)	.63	.00
Sleep Duration x Quadratic Block	4.89	(1,121)	<.05	.04
Sleep Duration x Cubic Block	0.10	(1,121)	.75	.00
Sleep Duration (Between Subjects)	3.61	(1,121)	.06	.03

We then examined the interaction at varying levels of sleep duration using the Aiken & West (1991) simple slopes method (calculating +/- 80 minutes from the sample's average sleep time of 418 minutes to index high and low sleep scores). Overall, *shorter* sleep durations were associated with greater changes in average shock intensity across blocks (See Figure 5). Among short sleepers (-1SD, or 5.65 hours: Linear Block Trend: F(1,121)=3.88, p=.051, η_p^2=.03; Quadratic Block: F(1,121)=8.75, p<.01, η_p^2=.07) and average sleepers (7 hours: Linear Block: F(1,121)=5.27, p<.05, η_p^2=.04, Quadratic Block Trend: F(1,121)=3.77, p=.06, η_p^2=.03), average shock intensity increased across the course of the experiment, peaking during middle blocks, and declining towards the end of the experiment. In contrast, there was not a significant Block effect at *high* levels (+1SD, or 8.27 hours) of sleep duration (F(2.62, 316.51)=.81, p=.49, η_p^2=.01), indicating that individuals with high levels of sleep demonstrated similar shock intensities across blocks.

Sleep Relations with Aggression Proneness and Lifetime Aggressive Acts. Secondary analyses for this aim involved calculating correlations between sleep duration and self-reported aggressive tendencies and lifetime aggressive acts, summarized in Table 2. Sleep duration was

not associated with a lifetime history of aggressive acts (as per the LHA), but was negatively associated with all forms of aggressive tendencies (as per the AQ) examined, with effects small to nearly moderate in size. Given that aggressive tendencies are indexing current functioning, whereas lifetime history of aggression is assessing past behaviors regardless of recency, the relationship of the three-day sleep duration with the former versus the latter makes sense.

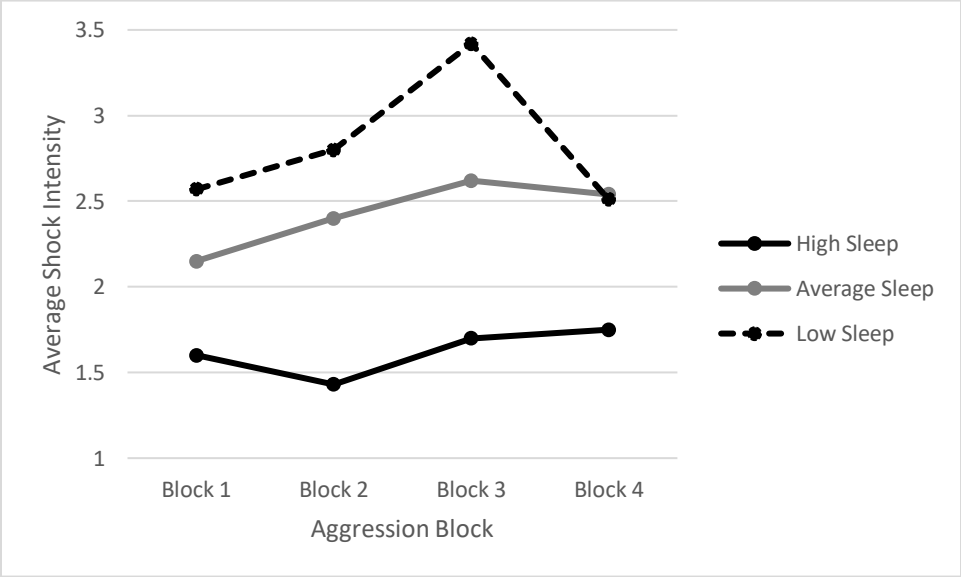


Figure 5. Average shock intensity across blocks by sleep duration groups

Sleep Relations with Daily Reports of Hostility and Aggression. Due to low endorsement of physically aggressive behavior and urges, analyses of relationships between sleep duration and daily aggression focused on urges to engage in verbal-relational aggression and engagement in these behaviors (e.g., insult, yell, exclude them). We examined the correlation between summed aggressive urges across all three days and average sleep duration to increase the power available in our analyses (n=141). Notably, no significant association

emerged between sleep and verbal-relational urges (r=0.03, p >.05) and engagement (r =-.10, p>.05), which likely reflects the relatively low endorsement of these in our sample.[13]

In other analyses, correlations between daily ratings of sleep duration and of PANAS hostility and irritability (rs of -.10 to -.01), as well as three-day average ratings of sleep duration and these moods were very small (rs of -.10 and -.12, respectively). Notably, there was relatively low endorsement of these moods, such that nearly all respondents endorsed very slight/none to a little hostility or irritability across days (Hostility *Ms*: 1.21-1.28, *SDs*: .52-.73; Irritability *M*s: 1.68-1.81, *SD*s: .79-1.02). Despite this low endorsement, these mood ratings still evidenced nearly moderate relationships with aggression (AQ) measures, providing some support for the validity of these items. As such, our results suggest that there was not a relationship between sleep duration and daily fluctuations in mood in our sample.[14]

Aim 2: Linking Sleep Duration with Response Inhibition and Emotional Processing

Grand-average waveforms for each Trial Type x Emotion Word Category at frontocentral and parietal sites are illustrated in Figure 6 (n=111).

Analyses of Electrode Site. For *P3*, electrode region (ordered in site analyses as midline frontal, parietal, and frontocentral sites) and its interactions with Trial Type (Go, No-Go) and Emotion Word Category (ordered as Negative, Neutral, and Positive) were included in preliminary analyses of each model. As before, we relied on a priori polynomial contrasts to

[13] Notably, these items evidenced moderate correlations with other measures of aggression (e.g., AQ subscales), suggesting that there was enough variability to have robust unimethod correlations. However, given the small relationships between sleep and aggression identified in our study, it is still possible that there was not enough variability to detect sleep-daily aggression effects.

[14] We also computed additional correlational analyses between daily sleep diary measures of total sleep time and measures of mood from the PANAS. Daily diary sleep duration and mood were largely not correlated, although sleep duration and irritability were correlated on Day 3 (r=-.17, p<.05). Daily diary sleep duration was also not linked with aggressive urges/behavior. Interestingly, however, daily diary (but not objective Fitbit) sleep duration was moreso associated with mood ratings of distress or anxiety on some days, but not associated or associated in the opposite direction with these affect scores on other days (e.g., rs of -.15 to .24), not showing consistent relationships between sleep and reported daily fluctuations in negative affect.

interpret patterns of site and emotion word category effects, based on our hypotheses about where key contrasts will be found. For site effects, a quadratic contrast would indicate that P3 amplitude increased or decreased from back to front of head (parietal versus frontal/frontocentral sites), whereas a significant linear contrast would represent a difference in P3 amplitude for the frontal versus frontocentral region. For emotion word category effects, a linear comparison would indicate that P3 amplitude differed for negative versus positive stimuli (i.e., valence effect), whereas a quadratic contrast would indicate that amplitude for neutral stimuli differed in comparison to emotional stimuli (i.e., emotional arousal effect).

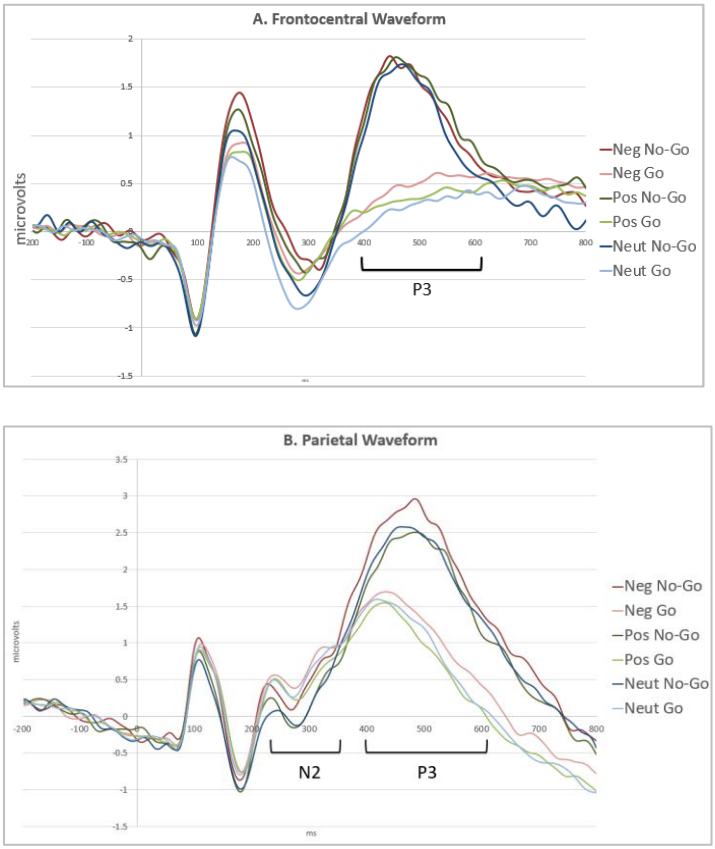


Figure 6. Grand average ERP waveforms. Figure A: Frontocentral. Figure B: Parietal

There were significant *linear* (frontocentral vs. frontal) and *quadratic* (parietal vs. frontal/frontocentral) effects of site, such that P3 amplitude increased from front to back of head,

and was larger at frontocentral versus frontal sites (see Table 4). As expected, and consistent with the extant literature, there was a significant *linear* Site x Trial Type interaction, such that Go/No-Go differentiation was maximal frontocentrally versus frontally. Also consistent with the extant literature, there was a significant *quadratic* Site x *linear* (negative vs. positive) Emotion Word Category interaction, such that P3 difference to *negative vs. positive* stimuli was maximal parietally relative to frontal/frontocentral sites (with contrast effects ranging in η_p^2=.10 parietally versus η_p^2=.06 frontocentrally and .01 frontally; See Figure 7). The P3 amplitude difference between emotional (negative/positive) vs. neutral words did not vary across electrode sites as a function of trial type (no interaction between site, trial type, and quadratic emotion word effects).

For the <u>*N2*</u>, visual inspection of the waveforms indicated a clear N2 parietally, but not in the waveforms of other regions. Thus, we conducted analyses involving the N2 only parietally.

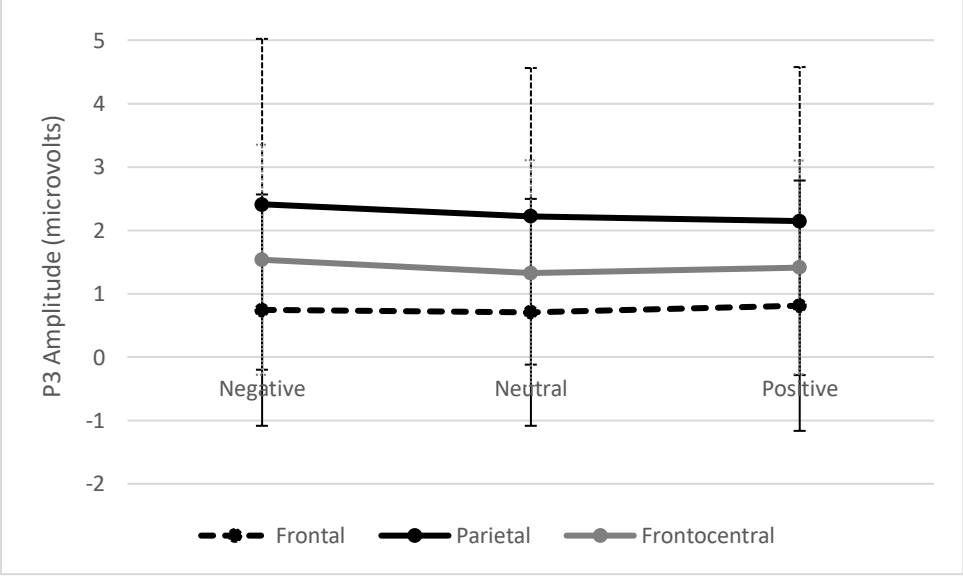


Figure 7. Amplitude of P3 responses as a function of word category and site

Overall Task Effects. Prior to conducting primary analyses (e.g., including sleep duration in the model), we examined whether typical effects of the task were found (i.e., go/no-

go, emotion word) to ascertain the validity of our task (See Table 4). We conducted an Emotion Word Category (3) x Trial Type (2) RMANOVA on P3 amplitude at frontocentral and parietal sites separately. *Frontocentrally*, there was an effect of Trial Type (greater P3 for No-Go Trials), and a *quadratic* effect of Emotion Word Category (greater P3 for emotional vs. neutral words). These results are as expected, based on previous ERP research with this task (see Verona et al., 2012). The Emotion Word Category x Trial Type interaction was not significant, indicating that the No-Go effect did not differ as a function of Emotional Word Category prior to inclusion of sleep duration in the model.

Results were largely similar *parietally* (significant effect of Trial Type), although there was a *linear* (rather than quadratic) effect of Emotion Word Category). There was a larger P3 for negative versus positive stimuli, suggesting that patterns of emotional processing diverged slightly by site.

Table 4. RMANOVA Effects by Site and within Site (P3).

Predictor	***F***	**df**	***p***	η_p^2
Task Effects by Site				
Site (Linear)	17.70	(1,110)	<.001	.14
Site (Quadratic)	18.38	(1,110)	<.001	.14
Emotion Word (Linear)	6.09	(1,110)	<.05	.05
Emotion Word (Quadratic)	7.34	(1,110)	<.01	.06
Trial Type	129.25	(1,110)	<.001	.54
Linear Site x Linear Emotion Word	3.19	(1,110)	.08	.03
Linear Site x Quadratic Emotion Word	0.84	(1,110)	.36	.01
Quadratic Site x Linear Emotion Word	4.39	(1,110)	<.05	.04
Quadratic Site x Quadratic Emotion Word	0.22	(1,110)	.64	.00
Linear Site x Trial Type	32.85	(1,110)	<.001	.23
Quadratic Site x Trial Type	2.67	(1,110)	.11	.02
Linear Site x Linear Emotion Word x Trial Type	0.34	(1,110)	.56	.00
Linear Site x Quadratic Emotion Word x Trial Type	0.41	(1,110)	.80	.00
Quadratic Site x Linear Emotion Word x Trial Type	0.47	(1,110)	.50	.00

Table 4. (Continued)

Quadratic Site x Quadratic Emotion Word x Trial Type	0.54	(1,110)	.46	.01
Task Effects within Site				
Frontocentral Site				
Emotion Word (Linear)	2.20	(1,110)	.14	.02
Emotion Word (Quadratic)	6.49	(1,110)	<.05	.06
Trial Type	97.89	(1,110)	<.001	.47
Linear Emotion Word x Trial Type	0.11	(1,110)	.74	.00
Quadratic Emotion Word x Trial Type	0.04	(1,110)	.85	.00
Parietal Site				
Emotion Word (Linear)	12.30	(1,110)	<.01	.10
Emotion Word (Quadratic)	0.60	(1,110)	.44	.01
Trial Type	46.78	(1,110)	<.001	.30
Linear Emotion Word x Trial Type	0.92	(1,110)	.34	.01
Quadratic Emotion Word x Trial Type	1.24	(1,110)	.27	.01

Sleep Effects on P3. *Frontocentral site.* Sleep duration was added into the model as a between subjects continuous factor (Sleep Duration x Emotion Word Category (3) x Trial Type (2)) (see Table 5). Interactions with Sleep Duration were decomposed through several methods, when applicable: (a) via follow up correlations, (b) by using the Aiken & West (1991) simple slopes method to examine effects at varying levels of sleep duration, and/or (c) by examining interactions of Sleep Duration x Trial Type within emotion word categories, and Sleep Duration x Emotion Word Category within trial types (to aid in decomposing the 3-way interactions only).

Results of the RMANOVA indicated that there was not a significant between-subjects effect of sleep duration on P3 amplitude, but there was a significant quadratic effect of Emotion Word Category (positive/negative vs. neutral) on frontocentral P3 amplitude, showing higher amplitude P3 to emotional relative to neutral words. The hypothesized Sleep Duration x Trial Type interaction trended to significance. There were also significant Sleep Duration x quadratic Emotion Word Category, quadratic Emotion Word Category x Trial Type, and Sleep Duration x

quadratic Emotion Word Category x Trial Type interactions (the latter superseding the two way Sleep Duration x Trial Type interaction).

Examination of the Sleep Duration x Trial Type interaction indicated that sleep duration was correlated with P3 go/no-go differentiation to a small degree (r= .12, p=.21), such that differentiation increased at higher levels of sleep (See Figure 8). Examined within simple slopes, Go/No-Go differentiation was smaller at low levels of sleep ($F(1,108)=28.60$, $p<.001$, $\eta_p^2=.21$), with large differentiation observed at average ($F(1,108)=94.19$, $p<.001$, $\eta_p^2=.47$) and high levels of sleep ($F(1,108)=67.14$, $p<.001$, $\eta_p^2=.38$).

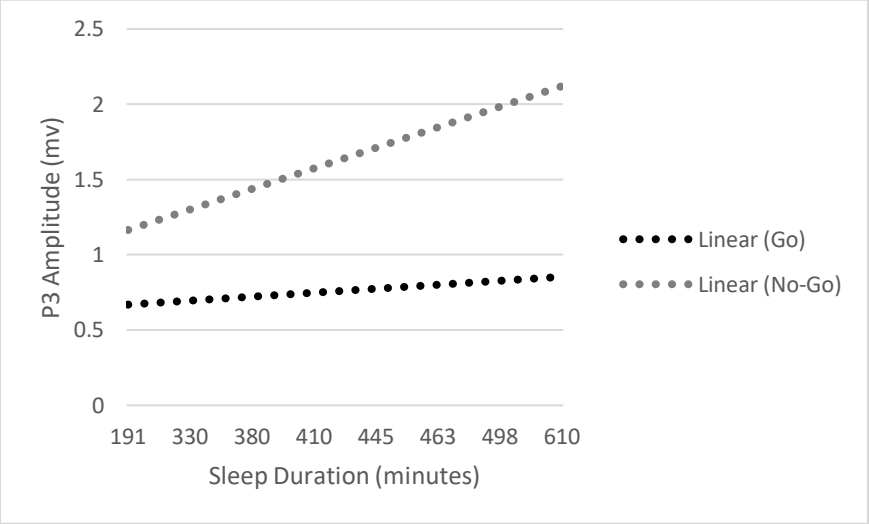


Figure 8. Amplitude of frontocentral P3 as a function of trial type (go vs no-go) and continuous scores on sleep duration

Next, we decomposed the hypothesized Sleep Duration x quadratic Emotion Word Category interaction. Overall, shorter sleep duration was linked with greater P3 differentiation to emotional versus neutral word blocks (emotion-neutral P3; r=-.29, $p<.01$; See Figure 9). Simple slopes analyses yielded a significant quadratic effect of Emotion Word Category at average ($F(1,108)=8.19$, $p<.01$, $\eta_p^2=.07$) and low (-1SD; $F(1,108)=18.24$, $p<.001$, $\eta_p^2=.14$) sleep duration but not at high sleep duration (+1SD; $F(2,216)=.58$, $p=.56$, $\eta_p^2=.01$). These results indicated an

enhanced P3 in emotional relative to neutral blocks at lower levels of sleep, but overall enhanced P3 across conditions for those higher in sleep (See Figure 8).

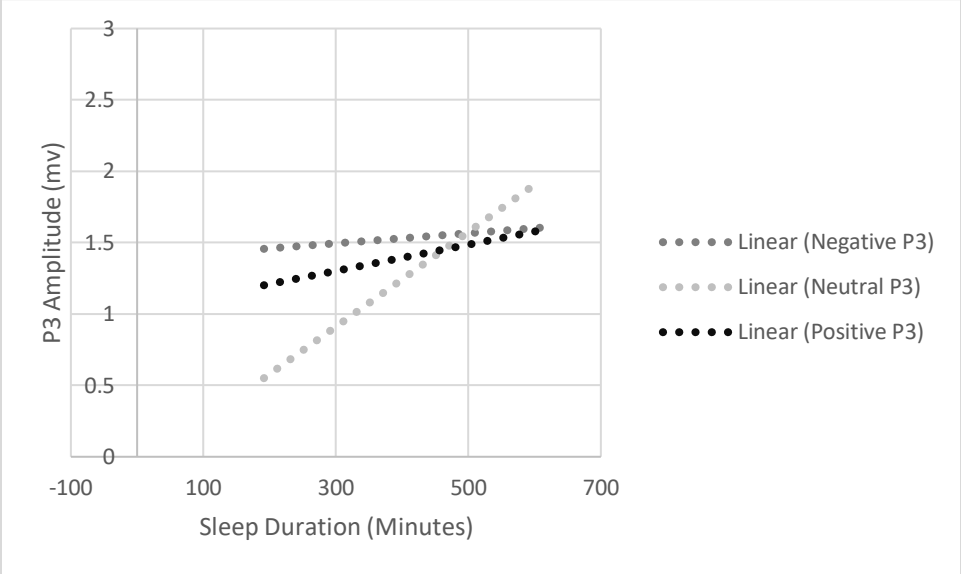


Figure 9. Amplitude of Frontocentral P3 as a function of word category and sleep duration

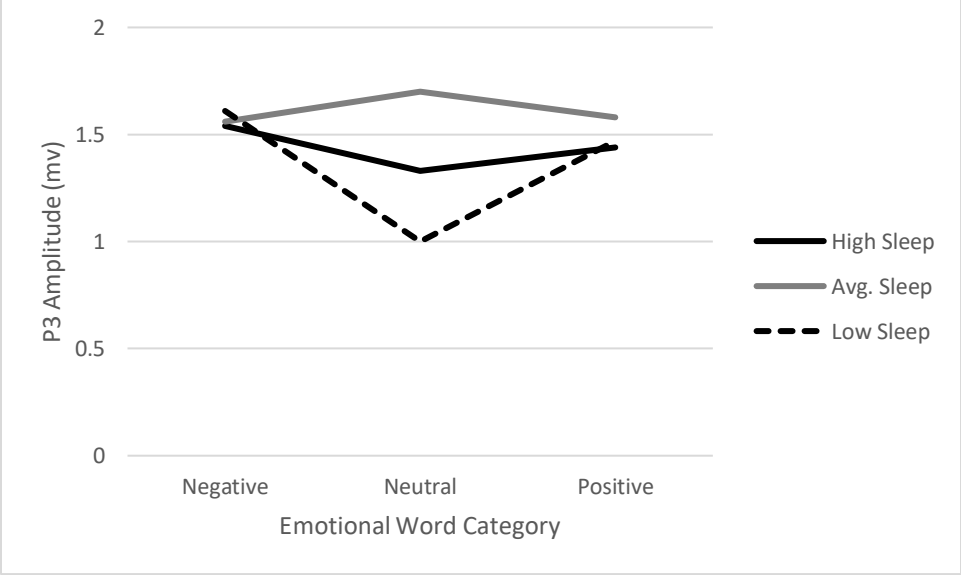


Figure 10. Amplitude of Frontocentral P3 as a function of word category and sleep duration simple slopes

We next decomposed the quadratic Emotion Word Category x Trial Type interaction by examining the main effect of Emotion Word separately within Go and No-Go Trial Types (See Figure 11). There was a significant linear ($F(1,108)=4.88$, $p<.05$, $\eta_p^2=.04$) effect of Emotion Word for Go trials, such that there was an increased P3 for negative words relative to neutral and positive words. There were also significant or nearly significant quadratic effects of Emotion Words for Go ($F(1,108)=3.39$, $p=.07$, $\eta_p^2=.03$) and No-Go ($F(1,108)=10.76$, $p<.01$, $\eta_p^2=.09$) trials, such that there was increased P3 for emotional versus neutral words across trial types.

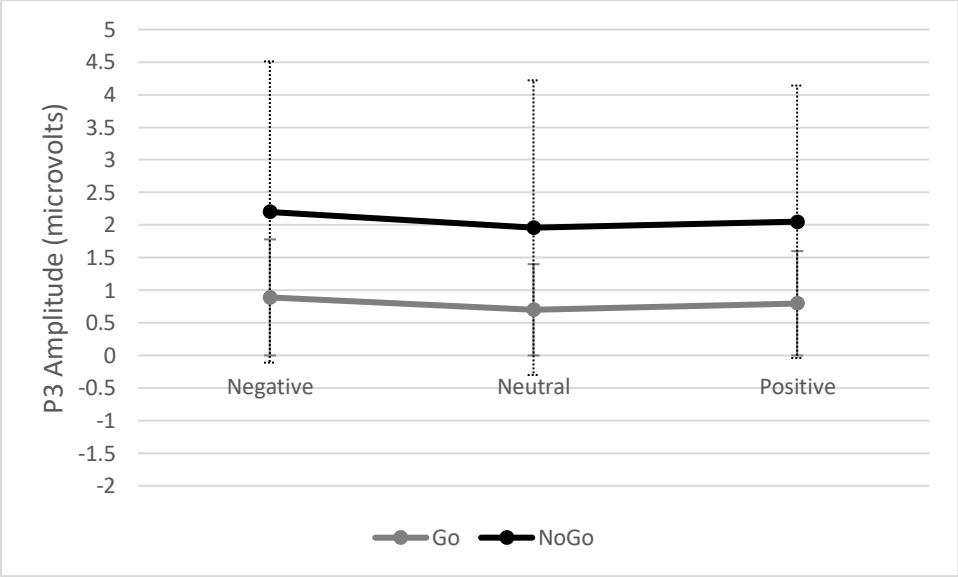


Figure 11. Amplitude of frontocentral P3 responses as a function of word category and trial type

Next, we decomposed the Sleep Duration x quadratic Emotion Word Category x Trial Type interaction. At the zero-order level, within no-go trials, shorter sleep duration was related to a small extent with smaller negative ($r=.08$, $p=.40$) and positive ($r=.04$, $p=.72$) no-go P3, and to a moderate extent with smaller neutral no-go P3 ($r=.20$, $p<.05$). In contrast, within go trials, shorter sleep was related to larger negative P3 ($r=.-.06$, $p=.55$), but less neutral ($r=.06$, $p=.55$) and positive ($r=.06$, $p=.53$) go P3, all to a small extent (See Figure 12).

We inspected this interaction by examining the Sleep Duration x Trial Type interaction separately within word categories. Within neutral ($F(1,108)=5.90$, $p<.05$, $\eta_p^2=.05$) and negative ($F(1,108)=3.13$, $p=.08$, $\eta_p^2=.03$) words, the Sleep Duration x Trial Type interaction was significant or nearly significant, such that Go versus No-Go differentiation increased with more sleep during these conditions (Neutral: $r=.18$, $p=.06$; Negative: $r=.16$, $p=.09$). It was not significant within positive ($F(1,108)=2.03$, $p=.16$, $\eta_p^2=.02$) words.

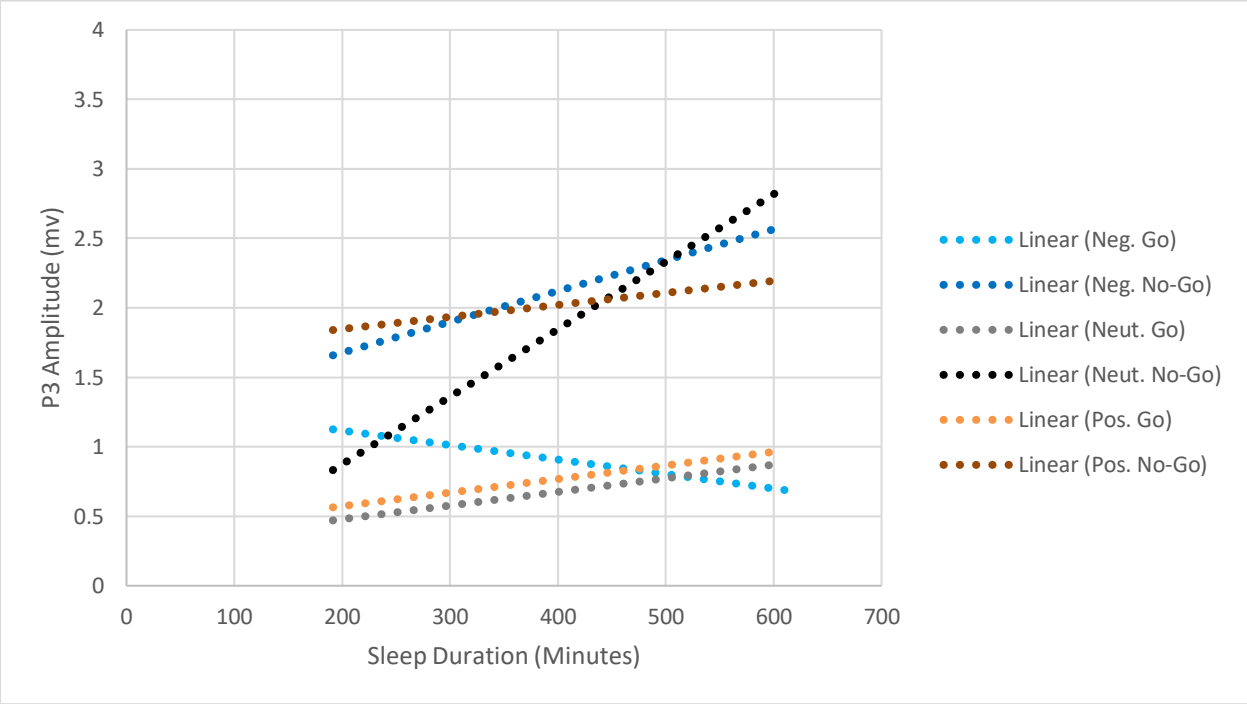


Figure 12. Amplitude of frontocentral P3 as a function of word category, trial type and sleep duration.

Next we decomposed by examining Sleep Duration x Emotion Word Category effects within levels of Trial Type. Within Go trials, there was a significant sleep duration x linear Emotion Word Category effect ($F(1,108)=4.11$, $p<.05$, $\eta_p^2=.04$) such that there was a larger P3 for negative relative to positive words at lower levels of sleep. In contrast, within No-Go trials, there was a significant sleep duration x quadratic Emotion Word Category effect ($F(1,108)=9.32$,

p<.01, η_p^2=.08) such that there a larger P3 for emotional relative to neutral words at lower levels of sleep.

Next, decomposing using simple slopes of sleep, we found that at low levels of sleep (-1SD), the quadratic Emotion Word Category x Trial Type interaction trended to significance (F(1,108)=2.79, p=.10, η_p^2=.02), showing that P3 go/no-go differentiation was *increased* for emotional versus neutral words at low levels of sleep duration. This interaction was not significant at average or high levels (+1SD) of sleep. In particular, low sleepers showed *reduced* Go/No-Go differentiation when confronted with neutral (F(1,108)=13.20, p<.001, η_p^2=.11) but not emotional (Positive: F(1,108)=28.34, p<.001, η_p^2=.21; Negative: F(1,108)=20.83, p<.001, η_p^2=.16) words. These results diverged from our expectations. Instead of go/no-go differentiation decreasing within emotional blocks among low sleepers, the emotional context may have facilitated go/no-go differentiation (made it more salient) among those obtaining less sleep (See Figure 13).

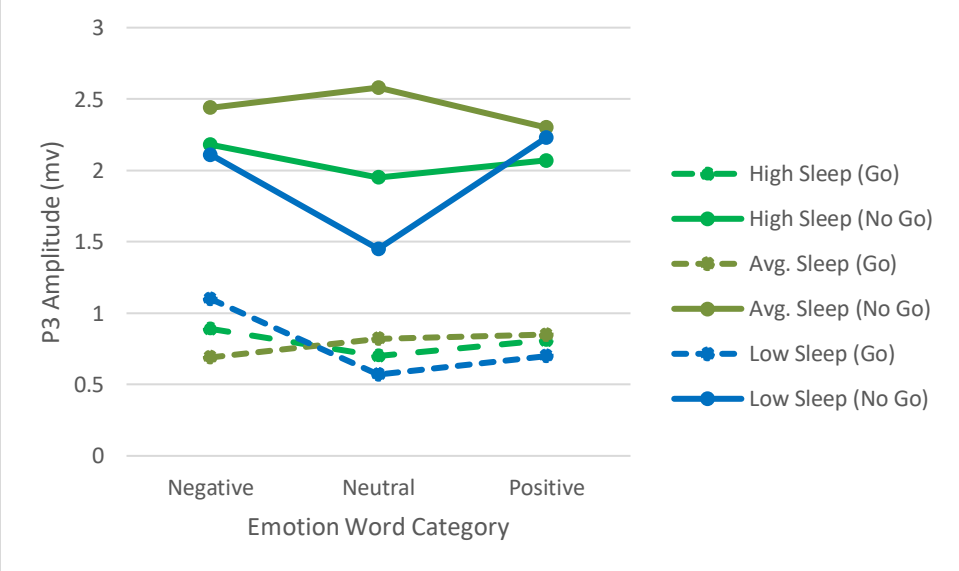


Figure 13. Amplitude of frontocentral P3 as a function of word category, trial type, and sleep duration simple slopes

Parietal effects. Contrary to our expectations across hypotheses, a Sleep Duration x Emotion Word (3) RMANOVA conducted on P3 amplitude at the parietal site did not yield any significant results (see Table 5). No other effects were significant either, except that sleep duration was marginally related to parietal P3 amplitude, with lower sleep linked with smaller P3 amplitude. Together, these findings suggest that sleep duration is not associated with P3 emotion or inhibitory processing at parietal sites.

Sleep Effects on N2. Contrary to our expectations, a Sleep Duration x Emotion Word (3) x Trial Type (2) RMANOVA conducted on parietal N2 amplitude did not yield any significant results (See Table 5). No other effects were significant either. Together, these findings suggest that sleep duration is not associated with the attentional component of inhibition processing, and moreover, that this aspect of inhibition does not vary as a function of emotional categorization of stimuli.

Table 5. RMANOVA Condition Effects as a Function of Sleep Duration

Predictor	***F***	**df**	***p***	η_p^2
Frontocentral Analyses (P3)				
Emotion Word (Linear)	0.25	(1,108)	.62	.00
Emotion Word (Quadratic)	14.37	(1,108)	<.001	.12
Trial Type	0.02	(1,108)	.90	.00
Sleep Duration (Between Subjects)	0.76	(1,108)	.39	.01
Linear Emotion Word x Sleep Duration	0.06	(1,108)	.81	.00
Quadratic Emotion Word x Sleep Duration	11.43	(1,108)	<.01	.10
Trial Type x Sleep Duration	2.66	(1,108)	.11	.02
Linear Emotion Word x Trial Type	2.85	(1,108)	.09	.03
Quadratic Emotion Word x Trial Type	4.32	(1,108)	<.05	.04
Linear Emotion Word x Trial Type x Sleep Duration	3.20	(1,108)	.08	.03
Quadratic Emotion Word x Trial Type x Sleep Duration	4.32	(1,108)	<.05	.04
Parietal Analyses (P3)				
Emotion Word (Linear)	0.36	(1,108)	.55	.00
Emotion Word (Quadratic)	1.10	(1,108)	.30	.01
Trial Type	0.09	(1,108)	.77	.00
Sleep Duration (Between Subjects)	3.38	(1,108)	.05	.03

Table 5. (Continued)

Linear Emotion Word x Sleep Duration	1.53	(1,108)	.22	.01
Quadratic Emotion Word x Sleep Duration	1.47	(1,108)	.23	.01
Trial Type x Sleep Duration	0.84	(1,108)	.36	01
Linear Emotion Word x Trial Type	0.32	(1,108)	.57	.00
Quadratic Emotion Word x Trial Type	0.00	(1,108)	.97	.00
Linear Emotion Word x Trial Type x Sleep Duration	0.16	(1,108)	.69	.00
Quadratic Emotion Word x Trial Type x Sleep Duration	0.04	(1,108)	.85	.00
Parietal Analyses (N2)				
Emotion Word (Linear)	.00	(1,108)	.99	.00
Emotion Word (Quadratic)	.11	(1,108)	.75	.00
Trial Type	1.74	(1,108)	.19	.02
Sleep Duration (Between Subjects)	0.16	(1,108)	.69	.00
Linear Emotion Word x Sleep Duration	0.19	(1,108)	.67	.00
Quadratic Emotion Word x Sleep Duration	0.01	(1,108)	.92	.01
Trial Type x Sleep Duration	0.33	(1,108)	.57	.00
Linear Emotion Word x Trial Type	0.07	(1,108)	.79	.00
Quadratic Emotion Word x Trial Type	1.32	(1,108)	.25	.00
Linear Emotion Word x Trial Type x Sleep Duration	0.07	(1,108)	.79	.00
Quadratic Emotion Word x Trial Type x Sleep Duration	0.81	(1,108)	.37	.01

Behavioral Results. We also conducted additional analyses to examine whether sleep duration and word category impacted performance on the task in terms of commission errors (i.e., erroneously responding to No-Go trials) and reaction time during Go trials (n=108) [15].

Commission Errors. Results of RMANOVA analyses indicated that there was a trending main effect of sleep duration on overall commission errors, such that sleep duration related negatively with number of errors (r=-.18, p=.06; See Figure 14). The Emotion Word Category and Sleep Duration x Emotion Word Category interactions were not significant.

[15] Additional behavioral data for 2 subjects were excluded from the broader valid EGNG data due to corrupt behavioral files.

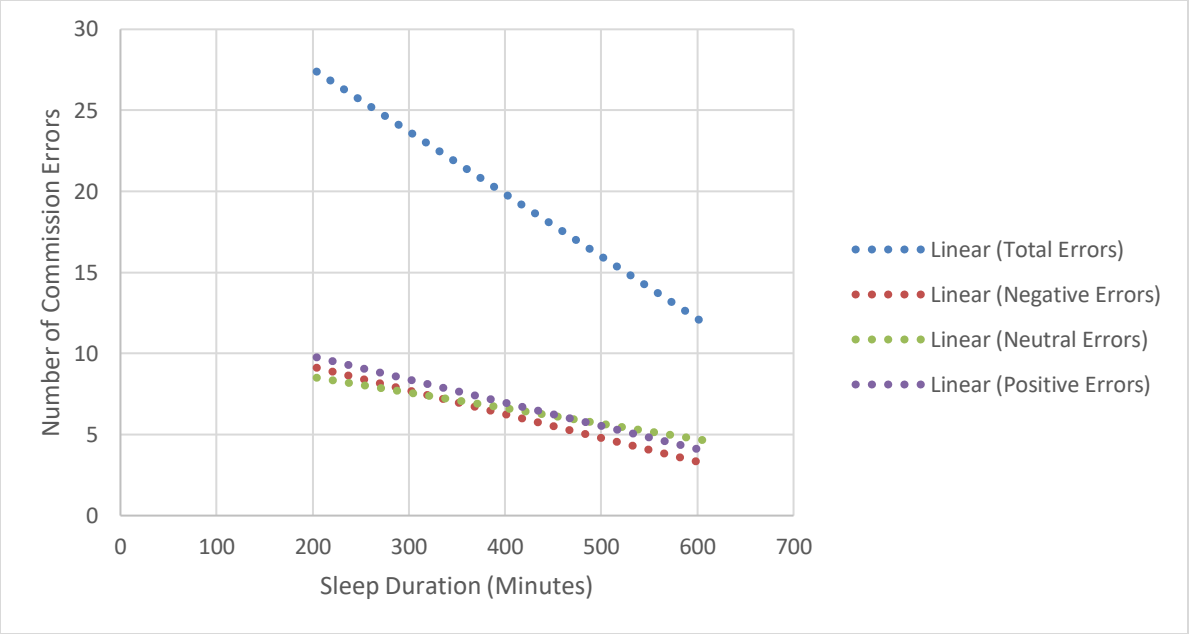


Figure 14. Commission errors as a function of word category and sleep duration.

Reaction Time. Longer sleep duration was associated with longer reaction time to negative (r=.06, p<.70), and not associated with neutral (r=.02, p=.87) or positive (r=-.01, p=.93) trials at the zero-order level (See Figure 15). However, RMANOVA analyses indicated that there was a trending Sleep Duration x linear Emotion Word Category interaction on reaction time. Using simple slopes analyses, there was a trending quadratic Emotion Word Category effect (F(1,105)=3.16, p=.07, η_p^2=.03) at low levels of sleep (-1SD), such that there was greater reaction time for emotional relative to neutral words. However, at average and high levels of sleep, there were significant linear (Average: F(1,105)=7.88, p<.01, η_p^2=.07; High: F(1,105)=11.08, p<.01, η_p^2=.10) and significant or nearly significant quadratic (Average: F(1,105)=6.70, p<.05, η_p^2=.06, High: F(1,105)=3.30, p=.07, η_p^2=.03) effects of Emotion Word Category, such that there was greater reaction time for negative versus positive words, and for emotional relative to neutral words. Notably, higher sleepers showed especially slow reaction time to negative words, whereas low sleepers showed slower reaction time to both emotion

words relative to neutral. Across emotion word categories, average sleepers had the fastest reaction times (see Figure 16).

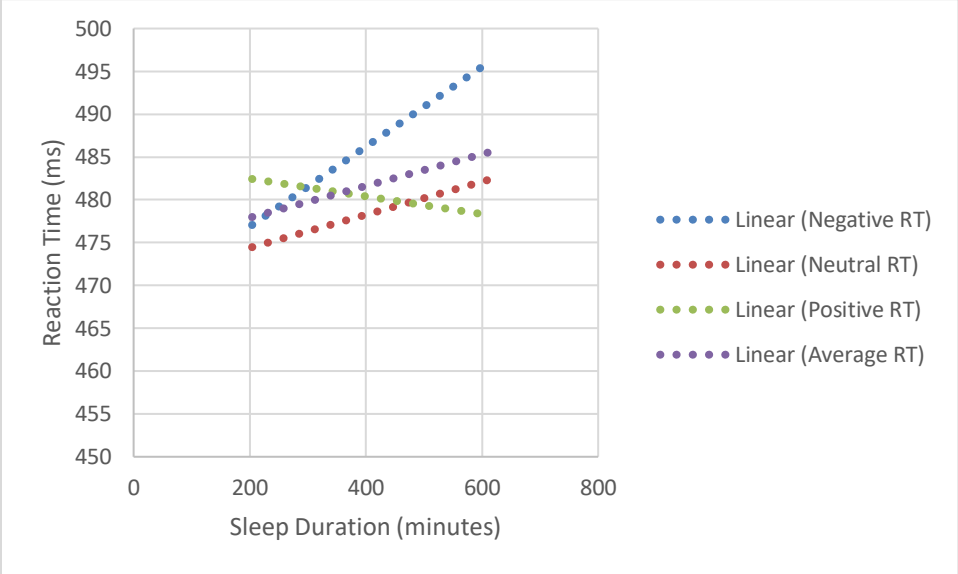


Figure 15. Reaction time as a function of word category and sleep duration.

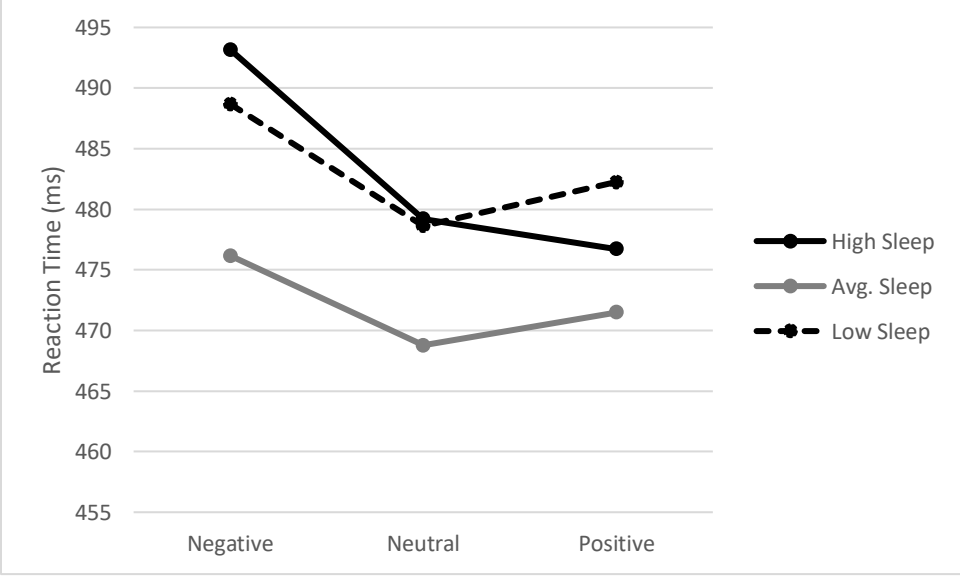


Figure 16. Reaction time as a function of word category and sleep duration simple slopes.

Potential Confounds of Aims 1 and 2

In order to examine third variables that may account for significant findings across aims, we included gender, past year alcohol use disorder symptoms, drug of choice use disorder symptoms, three-day average alcohol use, three-day any marijuana use (binary, coded as presence or absence of use), sleep measurement type (binary variable, coded as any subjective diary data used versus only objective Fitbit data used), and current depressive (measured via the MINI interview module and the MASQ) and anxiety symptoms (i.e., worry symptoms via the Penn State Worry Questionnaire and physiological anxiety via the MASQ) as covariates in separate analyses of Aims 1 and 2. Inclusion of these covariates largely did not alter the size or direction of our findings, with several exceptions. First, after 3-day marijuana use was included as a covariate in laboratory aggression analyses (Aim 1), the quadratic Block and Sleep Duration x quadratic Block interaction were no longer significant, but were similar in size (η_p^2=.03 with either covariate in the model for both effects versus original η_p^2 of .04 and .05. respectively) and interpretation to the original analyses. Second, after including physiological anxiety symptoms in the model, there was neither a quadratic block effect of aggression, nor an interaction of sleep and block (all η_p^{2s} <.01). However, there was still a trending between-subjects effect of sleep on overall aggression (and no significant relationship between anxiety symptoms and overall aggression). These results suggest that the quadratic trajectory of aggression observed in our initial analyses is related to physiological (but not cognitive) symptoms of anxiety, and that shorter sleep is overall still related to greater aggression. Third, after drug use disorder symptoms and 3-day marijuana use were included in frontocentral P3 analyses (Aim 2), the Sleep Duration x Emotion Word Category x Trial Type interaction was no longer significant, although it was similar in effect size (η_p^2=.02 and .03 respectively versus .03 in original analyses). Notably, drug use disorder symptoms and 3-day marijuana use did not significantly correlate with laboratory

aggression (drug use symptoms: *r*s = .03-.14, across blocks, all *p*s >.05; 3-day marijuana use *r*s = -.06 to .06, all *p*s >.05) or frontocentral P3 amplitude across task conditions (drug use symptoms: *r*s -.01 to .09, all *p*s >.05; 3-day marijuana use: *r*s -.03 to .13; all *p*s >.05). Thus, it is possible that including these covariates in our models reduced the effects enough that they were no longer significant. Since these effects were no longer significant, but remained similar in effect size and interpretation, these results collectively suggest that our experimental findings were not accounted for by these potential confounds.

Aim 3: Explaining the Sleep-Aggression Relationship

Preliminary Analyses. Prior to examining whether indices of response inhibition (No-go minus Go P3), emotional processing (Emotion vs Neutral P3), and their interplay explained variance in the relationship between sleep and aggression, we examined zero-order relationships between these variables. In so doing, we also calculated an index of changes in shock intensity during the aggression task to capture the quadratic block effect of shock intensity that was a key finding in Aim 1, along with the overall average shock intensity index.

As displayed in Table 6, between the two indices of aggression, the index of quadratic change across block showed stronger relationships with psychophysiological indices of response inhibition and emotional processing. First, *greater* quadratic change in shock intensity related to a small-to-moderate extent with *decreased* Go/No-Go differentiation as measured by the frontocentral P3 (r = -.19, *p*=.06; index of motor response inhibition), especially for negative words (r = -.26, *p*<.001; index of response inhibition during negative emotional condition). Quadratic change in shock intensity and sleep duration showed small-to-moderate relationships with Go/No-Go differentiation in the negative (Sleep: *r*=.14, *p*=.15; Quadratic Shock: *r*=-.26, *p*<.01) and neutral (Sleep: *r*=.18, *p*=.06; Quadratic Shock: *r*=-.12, *p*>.05) word conditions.

Average shock intensity was largely unrelated to these P3 indices, although it was similarly related to negative Go/No-Go differentiation (r=-.16, p=.12). Notably, sleep, average shock intensity, and quadratic change in shock intensity did not relate to the parietal N2 go/no-go differentiation (index of the attentional component of response inhibition; rs -.04 and .05, ps>.05). Second, *shorter* sleep duration was related to increased processing of positive (r=-.22, p<.05) and negative stimuli (r-.25, p<.05) (compared to neutral stimuli) as measured by the frontocentral, but not parietal, P3; however, average shock intensity and quadratic change in shock were largely not related to the negative emotional processing index (e.g., negative – neutral), although quadratic change was related to frontocentral positive emotional processing index (r=.18, p=.08) . Third, *shorter* sleep duration and *greater* quadratic change in shock intensity were related to greater commission errors (behavioral index of response inhibition), evidencing small-to-moderate relationships (Sleep r=-.18, p=.08; Quadratic Change r=.16, p>.05), particularly under negative (Sleep r=-.20, p<.05; Quadratic Change r=.24, p<.05) and positive (Sleep r=-.19, p=.06; Quadratic Change r=.18, p=.08) conditions. In contrast, average shock intensity was not related to commission errors. Finally, sleep duration, average shock intensity, and quadratic change in shock intensity were not correlated with reaction time, regardless of emotional condition.

Analyses of Indirect Effects (full n=99)**.** Results of path analyses examining whether response inhibition and emotional processing explained variance in the sleep-aggression relationship were unexpected (see Table 7). Across analyses, neither indices of *response inhibition* or *emotional processing* nor their interplay explained variance in the sleep-aggression relationship, whether aggression was indexed as average shock intensity or quadratic change across blocks (see Figures 15 and 16). Moreover, *behavioral* indices of impulsive responding

Table 6. Associations between sleep, aggression, and indices of response inhibition and emotional processing.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Sleep Duration	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
2. Average Shock Intensity	-.17	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
3. Shock Intensity Change Across Blocks (Quadratic block effect)	-.20*	.02	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Response Inhibition	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
4. GNG (FC P3)	.12	-.09	-.19	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
5. GNG (PAR N2)	.05	.01	-.04	-.27**	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Emotional Processing (P3)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
6. Neg. vs. Neutral (FC)	-.22*	-.03	.01	.12	-.11	--	--	--	--	--	--	--	--	--	--	--	--	--	--
7. Pos. vs. Neutral (FC)	-.25*	.09	.18	-.14	.37***	.40**	--	--	--	--	--	--	--	--	--	--	--	--	--
8. Neg. vs. Neutral (PAR)	.16	-.03	.11	.18	-.11	-.08	-.04	--	--	--	--	--	--	--	--	--	--	--	--
9. Pos. vs. Neutral (PAR)	.06	.09	.08	.16	.03	-.01	-.10	.61***	--	--	--	--	--	--	--	--	--	--	--
Response Inhibition in Emotional Conditions (FC P3)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
10. Neg. GNG	.14	-.16	-.26**	.85***	-.20*	-32***	-.17	.05	.12	--	--	--	--	--	--	--	--	--	--
11. Pos. GNG	-.04	.03	-.09	.79***	-.32***	.11	.23*	.14	.07	.48***	--	--	--	--	--	--	--	--	--
12. Neutral GNG	.18	-.07	-.12	.88***	-.21*	-.09	-.39***	.23*	.19	.68***	.53***	--	--						
Behavioral Indices	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
13. Commission Errors	-.18	.06	.16	-.31**	.03	.01	.19	.10	-.02	-.28**	-.22*	-.26**	--	--	--	--	--	--	--
14. Commission Errors (Neg.)	-.20*	.06	.24*	-.26**	.02	.02	.21*	.12	-.02	-.22*	-.18	-.25*	.95***	--	--	--	--	--	--
15. Commission Errors (Neutral)	-.13	.07	.09	-.29**	.03	.01	.19	.11	-.01	-.29**	-.17	-.26*	.95***	.85***	--	--	--	--	--

Table 6. (Continued)

16. Commission Errors (Pos.)	-.19	.06	.18	-.34**	-.07	-.01	.14	.07	-.03	-.31**	-.28**	-.24*	.96***	.88***	.87***	--	--	--	--
17. Reaction Time	.01	.06	.08	-.30**	-.07	-.03	.19	-.14	.01	-.18	-.21*	-.36***	-.01	-.01	.02	-.04	--	--	--
18. RT (Neg.)	.02	.02	.05	-.29**	-.07	-.05	.17	-.14	.00	-.17	-.19	-.35***	0.05	-.04	-.01	-.08	.99***	--	--
19. RT (Neutral)	.01	.10	.11	-.31**	.24*	-.03	.20*	-.13	.01	-.19	-.20*	-.36***	.01	.02	.03	-.01	.99***	.97***	--
20. RT (Pos.)	-.01	.05	.07	-.30**	-.01	-.02	.19	-.15	.01	-.17	-.22*	-.36***	.01	.01	.04	-.02	.99***	.96***	.97***

Note. Correlations between EGNG and aggression task variables (*n*=99). Correlations between EGNG and other variables (*n*=111). Correlations between sleep variables and other variables (*n*=143). GNG = No-Go – Go trials. FC = Frontocentral, PAR = Parietal. RT = Reaction Time. Pos = Positive. Neg = Negative.

and reaction time overall and within emotional conditions did not explain the sleep-aggression relationship (See Figures 17 and 18).

Taken together, these results indicate that psychophysiological and behavioral indices of response inhibition and emotional processing did not explain variance in the sleep-aggression relationship. Across models, relationships between sleep and aggression, and most variables examined in indirect effects (e.g., psychophysiological indices of response inhibition and emotional processing) and aggression, remained similar in size to their correlations at the zero-order level. This suggests that sleep duration and these variables may contribute *independent variance* in explaining aggressive behavior (and, thus, may be linked with aggression via different mechanisms).

There was one notable exception to this pattern. Specifically, in the model with sleep duration as the IV and commission errors as the indirect effect, relationships between commission errors overall and within emotional categories and shock intensity *decreased*

Table 7. Indirect effects linking sleep duration with measures of aggressive behavior.

Indirect Effect	*B*(SE)	β	95% CI	*p*
Average Shock Intensity				
Response Inhibition				
Attentional (Par GNG N2)	0.00(0.00)	-.01	[-.04,.02]	.69
Motor (Fc GNG P3)	0.00(.00)	-.01	[-.06,.02]	.75
Emotional Processing				
Negative vs. Neutral (FC P3)	0.00(.00)	.01	[-.04,.08]	.65
Positive vs. Neutral (FC P3)	0.00(.00)	-.01	[-.09,.01]	.18
Negative vs. Neutral (PAR P3)	0.00(.00)	.02	[-.04,.09]	.49
Positive vs. Neutral (PAR P3)	0.00(00)	-.01	[-.07,.06]	.77
Response Inhibition in Emotional Conditions				
Negative GNG (FC P3)	0.00(.00)	-.02	[-.08,.01]	.42
Positive GNG (FC P3)	0.00(.00)	.00	[-.03,.02]	.88
Neutral GNG (FC P3)	0.00(.00)	-.03	[-.11,.02]	.44
Behavioral Indices				
Commission Errors	0.00(.00)	-.01	[-.06,.04]	.83
Reaction Time	0.00(.00)	.00	[-.02,.03]	.96
Behavioral Indices in Emotional Conditions				
Negative Commission Errors	0.00(.00)	.00	[-.06,.04]	.89
Neutral Commission Errors	0.00(.00)	-.01	[-.05,.02]	.70
Positive Commission Errors	0.00(.00)	-.01	[-.07,.03]	.87
Negative Reaction Time	0.00(.00)	.00	[-.02,.02]	.93
Neutral Reaction Time	0.00(.00)			.96
Positive Reaction Time	0.00(.00)	.00	[-.02,.02]	.98
Quad. Shock Change Across Blocks				
Response Inhibition				
Attentional (Par GNG N2)	0.00(.00)	.00	[-.01,.02]	.88
Motor (Fc GNG P3)	0.00(.00)	-0.01	[-.07,.04]	.60
Emotional Processing				
Negative vs. Neutral (FC P3)	0.00(.00)	.01	[-.04,.08]	.65
Positive vs. Neutral (FC P3)	0.00(.00)	-.03	[-.09, .01]	.18
Negative vs. Neutral (Par P3)	0.00(.00)	0.02	[.00, .07]	.81
Positive vs. Neutral (Par P3)	0.00(.00)	0.00	[-.02, .04]	.29
Response Inhibition in Emotional Conditions				
Negative GNG (FC P3)	0.00(.00)	-0.03	[-.10, .02]	.32
Positive GNG (FC P3)	0.00(.00)	0.01	[-.02, .05]	.62
Neutral GNG (FC P3)	0.00(.00)	-0.01	[-.05, .03]	.50
Behavioral Indices				
Commission Errors	0.00(.00)	-0.02	[-.10, .04]	.57
Reaction Time	0.00(.00)	0.00	[-.02, .03]	.94
Behavioral Indices in Emotional Conditions				
Negative Commission Errors	0.00(.00)	-0.04	[-.13, .03]	.40
Neutral Commission Errors	0.00(.00)	0.00	[-.06, .06]	.97
Positive Commission Errors	0.00(.00)	-0.02	[-.09, .05]	.66

Table 7. (Continued)

Negative Reaction Time	0.00(.00)	0.00	[-.02, .03]	.90
Neutral Reaction Time	0.00(.00)	0.00	[-.02, .03]	.92
Positive Reaction Time	0.00(.00)	0.00	[-.02, .03]	.98

Note. GNG = No-Go – Go trials. FC = Frontocentral, PAR = Parietal.

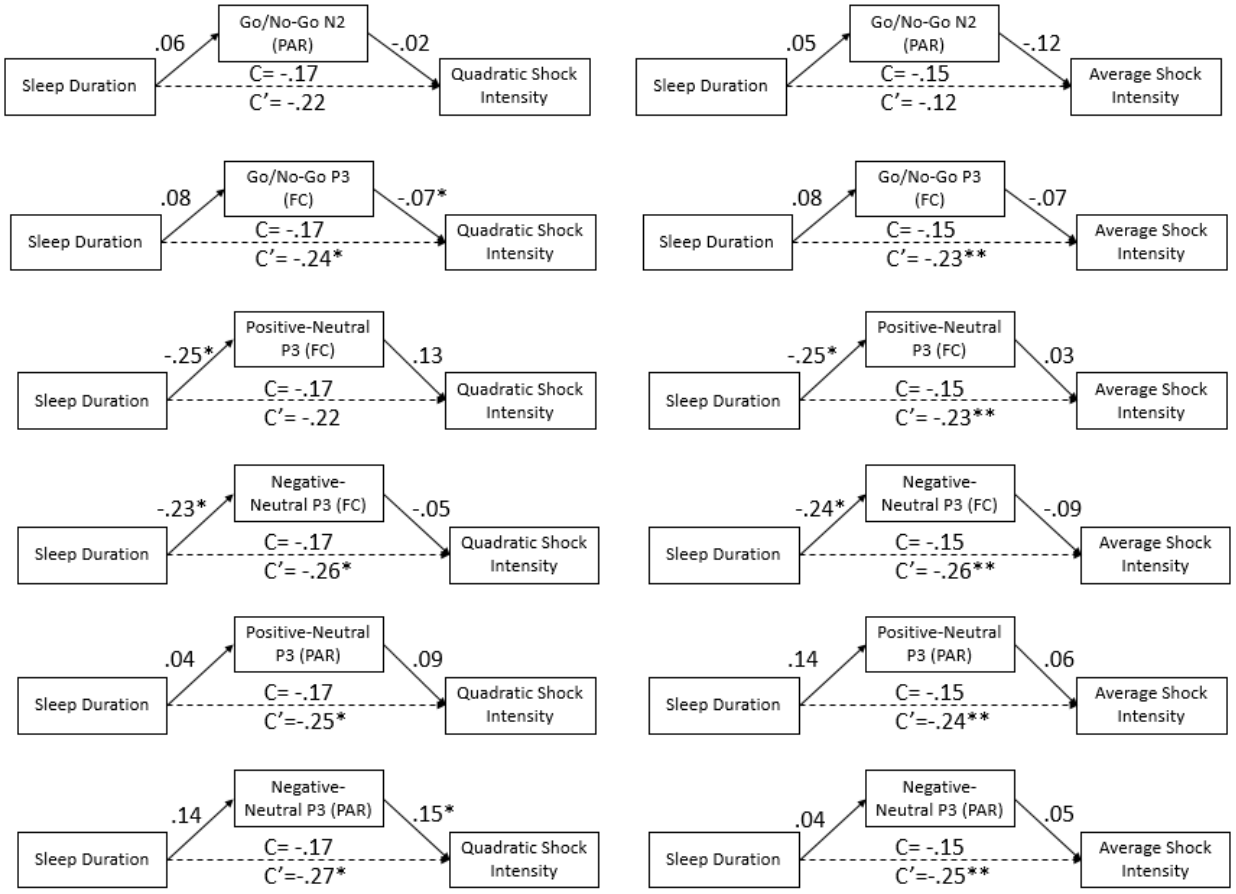


Figure 17. Models linking sleep duration, response inhibition (Go/No-Go N2 and P3), emotional processing (Negative-Neutral and Positive-Neutral P3), and indices of aggression
Note. PAR=Parietal; FC= Frontocentral.

in size (from nearly moderate to small, or small to very small effects). Moreover, sleep duration appeared to *suppress* relationship between commission errors during negative emotion word blocks and aggression in particular, such that the effect size decreased from nearly moderate to small, and changed to a negative association from a positive one. As such, sleep duration and an

index of impulsive decision-making appear to explain similar variance (and potentially capture the same variance) in laboratory aggressive behavior, with sleep duration emerging as the stronger predictor.

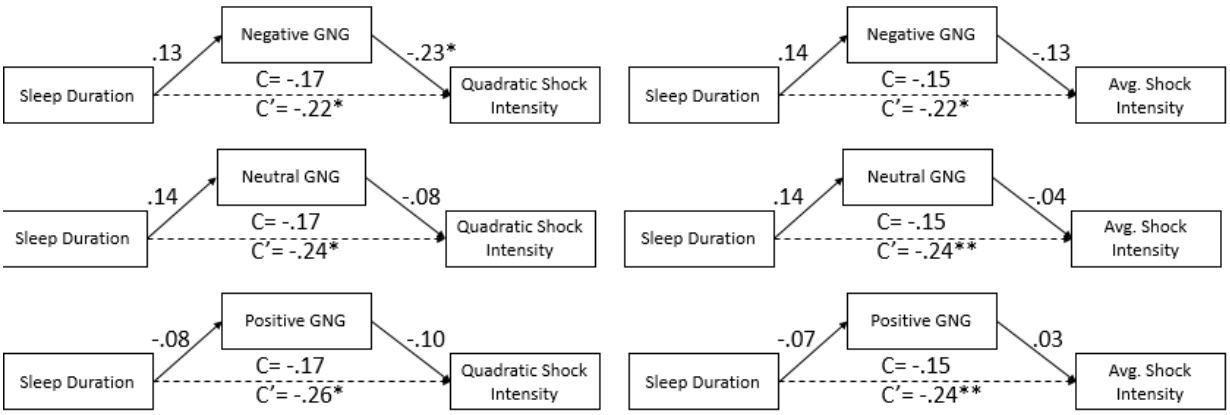


Figure 18. Models linking sleep duration, response inhibition during emotional conditions (FC P3), and aggression

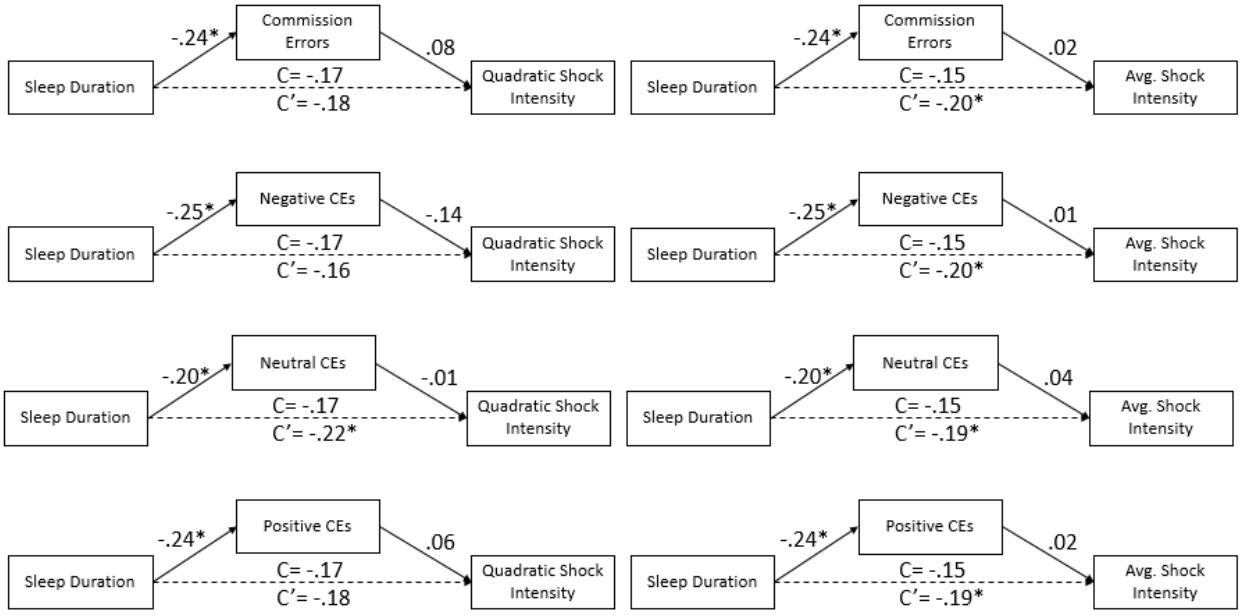


Figure 19. Models linking sleep duration and aggression via commission errors

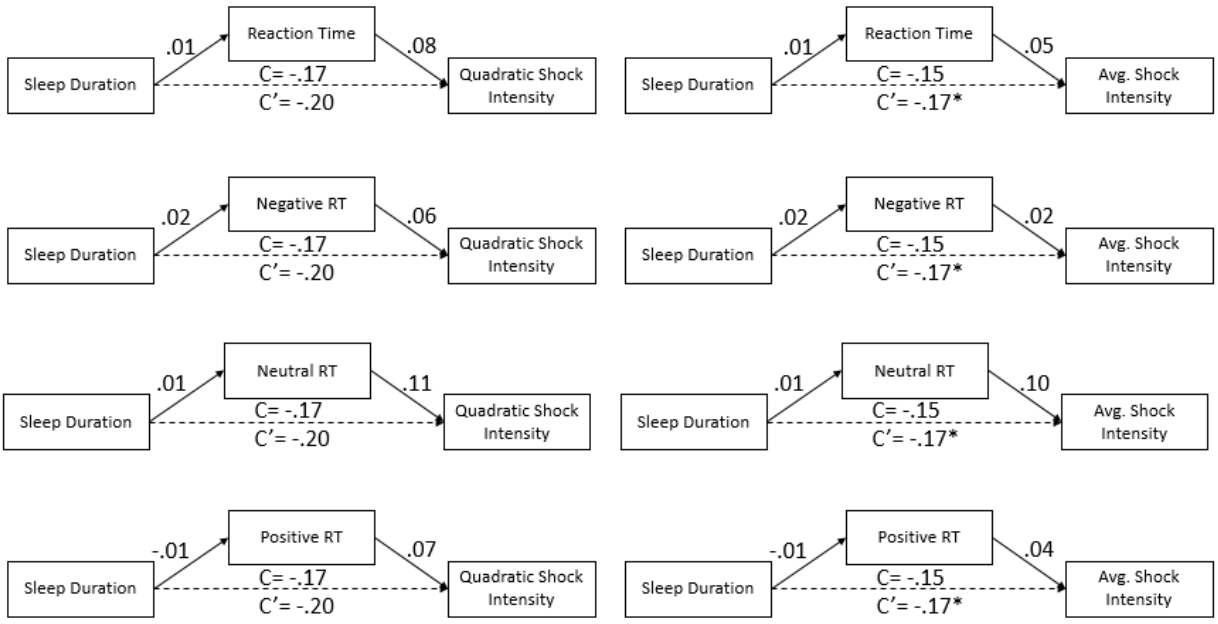


Figure 20. Models linking sleep duration and aggression via reaction time

Supplemental Analyses. First, we computed supplemental analyses to examine whether changes in hostility, irritability, or overall negative affect (measured by the PANAS and computed via a difference score) from before to after provocation explained variance in the effects of sleep duration on average shock intensity or quadratic shock change. That none of these variables explained substantive variance in the sleep-aggression relationships (mediation *B*s -.02 to.02, all *p*s>.05) indicates that mood *changes* that were more proximal to aggression did not explain the sleep-aggression relationship.

Second, we computed analyses to evalute support for our expectation that resource depletion as a function of emotional processing would diminish inhibitory control. If correct, ERP indices of emotional processing (which presumably depletes inhibitory resources) would explain the relationship between inhibitory control processing and our aggression outcome. To test this explanation, we computed a multiple regression analysis incorporating a theorized ERP

index of inhibitory processing (No-go minus Go at frontocentral P3) in the first step, and a theorized frontocentral or parietal ERP index of emotional processing (Emotional minus Neutral P3, Negative minus Neutral P3, Positive minus Neutral P3) in the second step, with laboratory aggression scores as the dependent variable. This analysis revealed that the beta coefficient for the index of inhibitory processing was not changed before and after (*b*s ranging from -.08 and -.07, *p*s>.05 across steps) including the emotional processing index on Step 2 (Emotional minus Neutral P3, *b*s=-.09 to -.11, *p*>.05; Negative minus Neutral P3, *b*s=-.09 tp -.10, *p*>.05; or Positive minus Neutral P3, *b*s=-.08 to -.11, *p*>.05). In fact, neither variable was significantly associated with aggressive behavior. Further, P3 indices of inhibitory and emotional processing were not correlated (*r*=.04, *p*=.69) at the zero-order level, suggesting that emotional processing did not impact inhibitory control, at least at the level of ERP indices. Building upon our Aim 3 analyses, which found that these processes did not explain the sleep-aggression relationship, these regression results fail to support the idea that effects of emotional contexts (e.g., such as resource depletion) on inhibitory control processing explain sleep-aggression relationship.

Discussion

Although short sleep duration has long been theorized to increase the risk of engaging in aggressive behavior (Kamphuis et al., 2012), studies examining this relationship yield conflicting findings, and the psychological processes that may explain this relationship are not well understood. The present study was the first to examine relationships between naturally-occurring sleep loss and aggression in the laboratory. We found that shorter sleep during a three-day period predicted subsequently *greater* lab aggressive behavior, with patterns of change in aggressive behavior across the experiment also differing as a function of sleep duration. This study was also the first to examine potential psychological explanations of this relationship. Interestingly, despite showing increased no-go P3 amplitude during emotional versus neutral word blocks in particular, lower sleepers' behaviors (RT and commission errors) were similar across emotional conditions, indicating that the higher P3 amplitude apparent at lower levels of sleep did not translate to better performance within emotional conditions. Moreover, although ERP indices of inhibitory and emotional processing related to sleep and aggression, albeit in somewhat different patterns, these mechanisms did not explain the sleep-aggression link. These results have important implications for elucidating the relationship between sleep and aggression.

Aim 1: Linking Sleep Duration with Aggression

This is the first laboratory-based study to find that shorter sleep (the loss of several hours of sleep) predicts *greater* laboratory aggressive behavior (operationalized via average shock intensity), even after controlling for potential confounds such as gender and substance use. This

finding diverges from sleep deprivation studies with *extreme* degrees of sleep loss (24-33hrs) that have linked sleep deprivation with *less* aggressive behavior than rested controls or have found *no* sleep-aggression relationship (Cote et al., 2013; Vohs et al., 2011). In the context of these findings, our results suggest that the sleep-aggression relationship varies as a function of the *degree* of sleep loss. Theoretically and empirically, varying levels of sleep loss (ranging from several hours to extreme sleep loss) differentially influence cognitive and affective mechanisms that are in turn linked with aggression (Krizan & Herlache, 2016). However, the heightened levels of fatigue, amotivation, and reductions in cortical arousal found in extreme sleep loss (Alhola & Polo-Kantola, 2007; Cote, Milner, Osip, Baker, & Cuthbert, 2008) could be more influential than these mechanisms, resulting in the reduced aggression (or even the absence of a clear relationship with aggression) found in the extant literature. In contrast, only several hours of sleep loss may be associated with *higher* levels of aggression since the comparatively greater energy levels allow them to manifest cognitive and emotional disruptions into aggression behavior, which could explain the pattern of results identified in our study. Indeed, our finding that shorter sleep related to greater current aggressive tendencies (consistent with a review by Bozzay & Verona, in preparation), but *not* a lifetime history of aggressive behavior, provides some external validity to these results, since sleep duration fluctuates over the lifespan.

Further consistent with this interpretation, *changes* in our index of aggression across the experiment differed as a function of degree of sleep duration. In simple slopes analyses, at lower durations of sleep (<6 hours), shock intensity followed an ‘n’-shape, increasing more sharply in middle blocks, then declining near initial shock levels at the end of the experiment. In contrast, comparatively longer sleepers (>8 hours) maintained similar (and lower) shock levels across the course of the blocks. That there was a brief “burst” of aggression among shorter sleepers may

reflect lapses in engagement of executive functions observed under conditions of shorter sleep duration (<7 hours; Altena, Micoulaud-Franchi, Geoffroy, Saanz-Arigita, Bioulac, & Philip, 2016) that are integral to behavior regulation (e.g., attentional control; stimuli categorization; emotional regulation; Delgado et al., 2008; Heller, Fox, Wing, McQuisition, Vack, & Davidson, 2015). Although these bursts of higher aggression do not appear to be driven by the consumption of cognitive resources by emotional processes, the inconsistent recruitment of executive functions for other reasons may explain this "aggressive burst." Supporting this interpretation, we found that changes in aggression across blocks disappeared after controlling for physiological anxiety symptoms. Anxiety, like shorter sleep, has been linked with executive function deficits (e.g., deficits sustaining attention; Grillon, Robinson, Mathur, & Ernst, 2016), potentially as the result of several factors (e.g., biases towards threatening stimuli; rumination or racing thoughts; Robinson, Vytal, Cornwell, & Grillon, 2013). However, it is unclear whether our findings (and potentially, executive functioning deficits) are attributable to anxiety rather than sleep, given the bidirectional relationship between sleep and anxiety (Alvaro, Roberts, & Harris, 2013). Nevertheless, in the context of this interpretation, the finding that longer sleepers administered lower and more consistent shock levels across the block may reflect more intact or consistent utilization of executive control functions.

Finally, that relationships between sleep duration and *self-reported* daily angry affect (e.g., hostility, irritability) and other indices of negative affect (e.g., distress, scared) did not emerge in our study has implications for understanding relationships between sleep and affect more broadly. One potential explanation for this lack of findings is that our primary measure of sleep duration was *predominantly* objective in nature,[16] and objective duration has not been

[16] Subjective reports of sleep duration were used in the absence of objective data, but subjective data were predominantly used to cross-validate the objective data.

linked with next day negative affect in the literature (see Konjarski et al., 2018 for a review). Notably, however, *subjective* sleep duration, which has been moreso linked with next-day negative affect, also did not relate to daily mood ratings, although this finding is inconsistent in the literature (e.g., 5 of 10 studies finding a significant relationship; Konjarski et al., 2018; relationships in our study fluctuated in interpretation and substantively in size across days). It may be that we did not find a sleep-daily affect relationship because such relationships are more pronounced in certain samples (e.g., those higher on anxiety), or are better explained by relationships with variables that fluctuate day-to-day (e.g., environmental factors, relationship issues, level of activity) that may or may not relate to sleep. Partially supporting this interpretation, we did find that worse sleep related to stronger overall negative affect in the laboratory, when *all* participants were exposed to the same laboratory mood manipulations. Another possibility is that momentary ratings of affect, such as those used in our laboratory session, better capture sleep-affect relationships than a single daily rating of affect. Nevertheless, given the established role of affective states in promoting aggression, and that this literature is in its early stages, research is needed to more fully elucidate the nature of the relationship between sleep and affect.

Aim 2: Linking Sleep Duration with Response Inhibition and Emotional Processing

This was also the first study to examine relationships between sleep duration and theorized electrophysiological indices of emotional processing and inhibitory control. Overall, we found that sleep duration in our study did not predict overall P3 amplitude. This result diverges from research linking worse sleep with smaller P3 amplitude, although such findings emerge in the litearature at quite extreme degrees of sleep restriction (36 hours of sleep deprivation, Gosselin, Koninck, & Campbell, 2005; 18 hours of sleep loss, Morris, So, Lee,

Lash, & Becker, 1992; observed after 3 hours of sleep for 4 consecutive nights, Choudhary, Kishanrao, Dhanvijay, & Alam, 2016). The heightened fatigue and reduced cortical arousal that is found at extreme levels of sleep loss (i.e., several nights of missed sleep), but not within several hours of sleep loss, may explain why our results diverged from the literature (Alhola & Polo-Kantola, 2007; Cote et al., 2008). Moreover, this study is the first to examine associations between sleep duration and overall N2 amplitude, finding no effect. In the context of the literature, our results suggest that *overall* decrements in theorized attentional processes (P3 and N2) appear to emerge at extreme, but not naturalistic levels of sleep loss.

However, we did find that the allocation of attentional resources towards inhibitory cues varied as a function of sleep duration, captured by the significant three-way interaction (Sleep Duration x Trial Type x Word Category) on frontocentral P3 amplitude that emerged in our analyses. Consistent with other research (albeit at extreme levels of sleep loss and measured behaviorally; Anderson & Platten, 2011), lower sleepers showed reduced P3 to No-go cues (in comparison to Go cues), suggestive of reduced inhibitory processing. Moreover, we found that there was *reduced* No-Go P3 during neutral word blocks among low sleepers in particular, whereas Go/No-Go differentiation during emotional word blocks was *similar* across levels of sleep duration. These results are consistent with typical attention bias effects towards more *salient* stimuli (or, increased emotional processing) found in the broader literature, and indicate that biases towards emotional stimuli are evident across a range of sleep durations (including at extreme levels of sleep loss; Yoo et al., 2007; Gujar et al., 2011). It is worth noting that this pattern of results was the *opposite* of what we would have expected (i.e., increased rather than decreased No-Go P3 to emotional words) had our resource depletion hypothesis been supported. Instead of depleting cognitive resources, this salience bias may be even more enhanced under

conditions of low sleep. This in turn could facilitate *enhanced* differentiation of inhibitory cues within emotional relative to neutral conditions. Such cognitive adjustments at lower levels of sleep could focus attention on decision-making within contexts that are more salient, and potentially more critical to survival.

Interestingly, however, these biases towards emotional words at lower levels of sleep did not translate to performance improvements in these conditions. Specifically, while lower sleepers made more commission errors overall (which supports our interpretation of the reduced No-Go versus Go P3 as reduced inhibitory processing), they also made *similar* degrees of commission errors across emotional and neutral conditions, and showed slower reaction time to emotion words relative to neutral. This is an interesting finding, suggesting that lower sleepers did not perform better for emotional versus neutral conditions, *despite* showing increased No-Go P3 during emotional blocks. In other words, poor sleepers expended *more effort in differentiation inhibitory cues* in emotional conditions *to perform at the same level of accuracy* as they did in neutral conditions. While such efforts were sufficient to enable lower sleepers to perform similarly across conditions on the relatively simple task used in this study, these biases could translate to differences in behavior within more complex real-world scenarios, although additional research is needed to deduce the extent to which this may be the case. Nevertheless, since emotional information is more salient (and potentially more complex) than neutral information, our results may reflect that processing of emotional information (or, processing of non-emotional information when emotional information is present) is less efficient at lower levels of sleep. Another possibility is that other factors that were not examined in this study (e.g., sleepiness, fatigue) that are modulated by sleep duration play a role in impulsive action.

That effects related to emotional words were more apparent in frontocentral regions may indicate that shorter sleep is related to greater allocation of attentional processes to salient stimuli (e.g., emotional stimuli), but not to other processes such as memory processing (e.g., context updating; Polich, 2007) that are more strongly linked to the parietally-maximal P3. It is also interesting that sleep duration did not modulate our index of earlier inhibitory processing (parietal No-Go N2); it may be that this component of inhibition is not sensitive to sleep duration, or that effects of sleep are only apparent at more marked degrees of sleep loss. However, additional research is needed examining this component more frontally (to be consistent with most inhibition research) to confirm our results.

Aim 3: Explaining the Sleep-Aggression Relationship

Finally, this was the first study to examine whether our hypothesized electrophysiological and behavioral indices of cognitive and affective processing link sleep duration with aggressive behavior. Unexpectedly, neither the indices of response inhibition and emotional processing, nor their interplay, explained the sleep-aggression link. This is interesting since, at the zero-order level, our results suggested sleep duration and aggression tapped into the same processes, albeit in different ways. Specifically, sleep duration was predominantly associated with greater processing of emotional stimuli (nearly moderate effects), and to a lesser extent with inhibitory processing, whereas our measure of aggression (specifically, quadratic shock change) was primarily associated with inhibitory processing under negative emotional conditions (a result consistent with Verona & Bresin, 2015).

One interpretation of this pattern of effects is that sleep duration and indices of response inhibition and emotional processing explain *independent variance* in aggressive behavior. Indeed, relationships between sleep and aggression, and between indices of inhibitory and

emotional processing and aggression, remained relatively unchanged from their zero-order associations in the expanded models (i.e., with indirect effects). Diverging from dominant theories in the aggression literature implicating inhibitory control as a primary mechanism that drives the sleep-aggression relationship (e.g., Krizan & Herlache, 2016), our results suggest that inhibitory processing under negative emotional conditions specifically could comprise a pathway to aggression that is *unrelated* to sleep duration.

However, it is also possible that changes in cognitive functioning observed under conditions of lower sleep duration (e.g., emotional and inhibitory processes) modulate processes that are more proximal to aggressive behavior. For example, the comparably greater processing of emotional stimuli and decrements in inhibitory control we found at lower levels of sleep could contribute to decreased emotion regulation (which have been observed under conditions of extreme sleep loss; Thomsen, Mehlsen, Christensen, & Zachariae, 2003). In turn, within negative emotional (or threatening) contexts in particular, such dysregulation could translate to greater aggression risk (Roberton, Daffern, & Bucks, 2012). Our results preliminarily show partial support for such an idea. That is, our results suggested that *changes* in negative affect across the experiment did not explain variance in the sleep-aggression relationship; however, they did show that shorter sleep was associated with greater overall negative affect during the laboratory session, implicating more longstanding difficulties regulating emotions or broader tendencies towards negative affect under stressful conditions in reduced sleep, which theoretically could increase the risk of engaging in aggression. However, additional research is needed to test whether this may be the case at the psychophysiological level (e.g., emotion regulation paradigm and the Late Positive Potential).

It is also possible, however, that the processes we examined in this study are closely aligned with the processes that do link sleep and aggression, but that we are unable to detect this relationship due to methodological limitations. For example, while the components examined in this study theoretically represent our latent cognitive constructs of interest (i.e., emotional and inhibitory processing), it is possible that the ERPs only capture parts of what it means to have problems with inhibitory control or emotional processing, and that what ERPs capture in this study does not overlap with what drives aggression in the laboratory. Indeed, psychophysiological measurements often do not correspond to behavior (e.g., MacLeod & Donaldson, 2017). Future research employing alternative means of operationalizing these processes, particularly in innovative ways that may more closely correspond to theorized aggressive processes (i.e., measuring these processes at the brain level in the context of a stress induction) may be useful in elucidating the extent to which this may be the case.

Limitations

Our results should be interpreted in the context of several limitations. First, we did not experimentally manipulate the degree of sleep loss in this study, precluding inferences about the causality of sleep on aggression. Additional research is needed to ascertain whether this relationship persists when shorter durations of sleep (e.g., several hours of sleep loss) are induced using experimental methods, or whether the relationship that emerged in this study may be due to other explanatory processes (e.g., comorbid psychopathology, broad emotional dysregulation). Second, we did not measure physiological aspects of sleep (e.g., REM sleep phase) that research suggests could contribute to sleep-aggression relationships (e.g., Fantini, Corona, Clerici, & Ferini-Strambi, 2005). Methods such as polysomnography are needed in subsequent studies to elucidate whether particular features of sleep, sleep duration, and/or a

combination of these factors may contribute to aggression. Third, we were unable to conduct source localization in this study due to limitations of our EEG equipment; additional research utilizing methods with more regional precision (e.g., fMRI) is needed to identify specific brain circuits that may be impacted by sleep loss that underlie these processes. Fourth, it is possible that our choice of aggression paradigm design (which differed from other aggression paradigms chosen in the sleep literature, such as the point-stealing task and a reaction time aggression game; Cote et al., 2013; Vohs et al., 2011) could have contributed to our results that differed from the broader sleep-aggression experimental literature. Fifth, despite methodological strengths of our aggression paradigm (e.g., relationships with external measures of aggression; relationships with indices of aggression following provocation in the task; option to "not aggress"), the use of this task may not be fully comparable to the contexts in which aggression manifests in the real world. Sixth, as there are individual differences in the duration of sleep that is "needed" for adequate rest and rejuvenation, it is possible that the amount of sleep linked with increased risk of aggression varies at the individual level, a question we were unable to examine in this study. Additional research is needed to investigate the extent to which this may be the case.

Strengths and Conclusions

Nevertheless, this study offers an important contribution to the literature. We examined relationships that have been theorized in the literature (e.g., Krizan & Herlache, 2016) between sleep, indices of inhibitory and emotional processing, and aggression using a unique, diverse sample of community participants with a range of sleep durations and aggressivity. We utilized a methodologically rigorous approach, including using concordance of three days of objective sleep duration and sleep diary data to improve the stability and generalizeability of our sleep

estimates to broader sleep duration patterns. We used well-tested laboratory paradigms to measure indices of inhibitory and emotional processing in real-time, and mapped our sleep and psychophysiological data to aggressive behavior observed in the laboratory, and self-reported measures of aggression.

Our results have important implications for understanding relationships between sleep, cognitive processes related to behavior, and aggression. Our study is the first to find that shorter sleep predicts greater aggressive behavior observed in the laboratory, with patterns of change in aggressive behavior across the experiment also differing as a function of sleep duration. We found that lower sleep is linked with increased inhibition processing towards emotional versus neutral information, but that this does not translate to better performance overall, such that worse sleepers work harder in emotional contexts in order to perform comparably to neutral contexts. Although the cognitive processes examined in this study did not explain variance in the sleep-aggression relationship, it is possible that other mechanisms (e.g., mechanisms linked with the experience and regulation of emotion) that are more proximally related to aggression could explain this link, although additional research is needed to examine whether this is the case. Our study provides the first quasi-experimental evidence that sleep is related to aggression; if additional research is able to find mechanisms linking sleep and aggression, this could support sleep as an upstream intervention point for reducing aggression risk.

References

Agargun, M. Y., Tekeoglu, I., Gunes, A., Adak, B., & Kara, H. Ercan M.(1999). Sleep quality and pain threshold in patients with fibromyalgia. *Comprehensive Psvchiatry*, *4*(3), 226-228.

Aiken, L. S., West, S. G., & Reno, R. R. (1991). *Multiple regression: Testing and interpreting interactions*. Sage.

Algom, D., Chajut, E., & Lev, S. (2004). A rational look at the emotional stroop phenomenon: a generic slowdown, not a stroop effect. *Journal of experimental psychology: General*, *133*(3), 323.

Alhola, P., & Polo-Kantola, P. (2007). Sleep deprivation: Impact on cognitive performance. *Neuropsychiatric disease and treatment*, *3*(5), 553.

Altena, E., Micoulaud-Franchi, J. A., Geoffroy, P. A., Sanz-Arigita, E., Bioulac, S., & Philip, P. (2016). The bidirectional relation between emotional reactivity and sleep: From disruption to recovery. *Behavioral neuroscience*, *130*(3), 336.

Alvaro, P. K., Roberts, R. M., & Harris, J. K. (2013). A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. *Sleep*, *36*(7), 1059-1068.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Anderson, C., & Platten, C. R. (2011). Sleep deprivation lowers inhibition and enhances impulsivity to negative stimuli. *Behavioural brain research*, *217*(2), 463-466.

Anestis, M. D., Soberay, K. A., Gutierrez, P. M., Hernández, T. D., & Joiner, T. E. (2014). Reconsidering the link between impulsivity and suicidal behavior. *Personality and Social Psychology Review*, *18*(4), 366-386.

Archer, J. (2004). Sex differences in aggression in real-world settings: a meta-analytic review. *Review of General Psychology*, *8*(4), 291.

Ashworth, A., Hill, C. M., Karmiloff-Smith, A., & Dimitriou, D. (2015). The importance of sleep: attentional problems in school-aged children with Down syndrome and Williams syndrome. *Behavioral sleep medicine*, *13*(6), 455-471.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test–retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of psychosomatic research*, *53*(3), 737-740.

Banks, S., Van Dongen, H. P., Maislin, G., & Dinges, D. F. (2010). Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. *Sleep*, *33*(8), 1013-1026.

Barclay, N.L., & Ellis, J.G. (2013). Sleep-related attentional bias in poor versus good sleepers is independent of affective valence. *Journal of Sleep Research, 22*(4), 414-421.

Barclay, N. L., & Gregory, A. M. (2013). Quantitative genetic research on sleep: a review of normal sleep, sleep disturbances and associated emotional, behavioural, and health-related difficulties. *Sleep Medicine Reviews*, *17*(1), 29-40.

Bekker, E. M., Kenemans, J. L., & Verbaten, M. N. (2004). Electrophysiological correlates of attention, inhibition, sensitivity and bias in a continuous performance task. *Clinical Neurophysiology*, *115*(9), 2001-2013.

Belenky, G., Wesensten, N. J., Thorne, D. R., Thomas, M. L., Sing, H. C., Redmond, D. P., ... & Balkin, T. J. (2003). Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: A sleep dose-response study. *Journal of sleep research*, *12*(1), 1-12.

Berman, M. E., McCloskey, M. S., Fanning, J. R., Schumacher, J. A., & Coccaro, E. F. (2009). Serotonin augmentation reduces response to attack in aggressive individuals. *Psychological Science*, *20*(6), 714-720.

Bettencourt, B., Talley, A., Benjamin, A. J., & Valentine, J. (2006). Personality and aggressive behavior under provoking and neutral conditions: a meta-analytic review. *Psychological bulletin*, *132*(5), 751.

Bixler, E. (2009). Sleep and society: an epidemiological perspective. *Sleep Medicine*, *10*, S3-S6.

Blagrove, M., Alexander, C., & Horne, J. A. (1995). The effects of chronic sleep reduction on the performance of cognitive tasks sensitive to sleep deprivation. *Applied Cognitive Psychology*, *9*(1), 21-40.

Boles, S. M., & Miotto, K. (2003). Substance abuse and violence: A review of the literature. *Aggression and violent behavior*, *8*(2), 155-174.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological review*, *108*(3), 624.

Boyne, K., Sherry, D. D., Gallagher, P. R., Olsen, M., & Brooks, L. J. (2013). Accuracy of computer algorithms and the human eye in scoring actigraphy. *Sleep and Breathing*, *17*(1), 411-417.

Bradley, M. M., & Lang, P. J. (1999). *Affective norms for English words (ANEW): Instruction manual and affective ratings* (pp. 1-45). Technical report C-1, the center for research in psychophysiology, University of Florida.

Brower, K. J. (2003). Insomnia, alcoholism and relapse. *Sleep medicine reviews*, *7*(6), 523-539.

Brown, G. L., Ebert, M. H., Goyer, P. F., Jimerson, D. C., Klein, W. J., Bunney, W. E., & Goodwin, F. K. (1982). Aggression, suicide, and serotonin: Relationships of CSF amine metabolites. *The American journal of psychiatry*.

Brown, J. W., & Braver, T. S. (2005). Learned predictions of error likelihood in the anterior cingulate cortex. *Science*, *307*(5712), 1118-1121.

Brown, G. L., Ebert, M. H., Goyer, P. F., Jimerson, D. C., Klein, W. J., Bunney, W. E., & Goodwin, F. K. (1982). Aggression, suicide, and serotonin: Relationships of CSF amine metabolites. *The American Journal of Psychiatry*.

Buss, A. H. (1961). The psychology of aggression. New York: Wiley.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of personality and social psychology*, *63*(3), 452.

Buss, A. H., & Warren, W. L. (2000). The Aggression Questionnaire. Los Angeles, CA: Western Psychological Services

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193-213.

Buysse, D. J., Yu, L., Moul, D. E., Germain, A., Stover, A., Dodds, N. E., ... & Pilkonis, P. A. (2010). Development and validation of patient-reported outcome measures for sleep disturbance and sleep-related impairments. *Sleep, 33*(6), 781-792.

Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep*, *35*(2), 287-302.

Carretié, L., Hinojosa, J. A., Albert, J., López-Martín, S., De La Gándara, B. S., Igoa, J. M., & Sotillo, M. (2008). Modulation of ongoing cognitive processes by emotionally intense words. *Psychophysiology*, *45*(2), 188-196.

Carretié, L., Martín-Loeches, M., Hinojosa, J. A., & Mercado, F. (2001). Emotion and attention interaction studied through event-related potentials. *Journal of cognitive neuroscience*, *13*(8), 1109-1128.

Cherek, D. R., Moeller, F. G., Schnapp, W., & Dougherty, D. M. (1997). Studies of violent and nonviolent male parolees: I. Laboratory and psychometric measurements of aggression. *Biological psychiatry*, *41*(5), 514-522.

Choudhary, A. K., Kishanrao, S. S., Dhanvijay, A. K. D., & Alam, T. (2016). Sleep restriction may lead to disruption in physiological attention and reaction time. *Sleep Science*, *9*(3), 207-211.

Coccaro, E. F., Berman, M. E., & Kavoussi, R. J. (1997). Assessment of life history of aggression: development and psychometric characteristics. *Psychiatry research*, *73*(3), 147-157.

Coccaro, E. F., Berman, M. E., Kavoussi, R. J., & Hauger, R. L. (1996). Relationship of prolactin response to d-fenfluramine to behavioral and questionnaire assessments of aggression in personality-disordered men. *Biological psychiatry*, *40*(3), 157-164.

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry*, *62*(2), 168-178.

Cooney, N. L., Litt, M. D., Morse, P. A., Bauer, L. O., & Gaupp, L. (1997). Alcohol cue reactivity, negative-mood reactivity, and relapse in treated alcoholic men. *Journal of Abnormal Psychology*, *106*(2), 243.

Cohen, P., Cohen, J., West, S. G., & Aiken, L. S. (2002). Applied multiple regression/correlation analysis for the behavioral sciences . Hillsdale: Lawrence Earlbaum Associates. *Inc. Google Scholar*.

Cote, K. A., McCormick, C. M., Geniole, S. N., Renn, R. P., & MacAulay, S. D. (2013). Sleep deprivation lowers reactive aggression and testosterone in men. *Biological Psychology*, *92*(2), 249-256.

Cote, K. A., Milner, C. E., Osip, S. L., Baker, M. L., & Cuthbert, B. P. (2008). Physiological arousal and attention during a week of continuous sleep restriction. *Physiology & behavior*, *95*(3), 353-364.

Cote, K. A., Milner, C. E., Smith, B. A., Aubin, A. J., Greason, T. A., Cuthbert, B. P., ... & Duffus, S. E. (2009). CNS arousal and neurobehavioral performance in a short-term sleep restriction paradigm. *Journal of sleep research*, *18*(3), 291-303.

Cross, C. P., Copping, L. T., & Campbell, A. (2011). Sex differences in impulsivity: a meta-analysis. *Psychological Bulletin*, *137*(1), 97.

Curtin, J. J. (2011). PhysBox: The psychophysiology toolbox. An open source toolbox for psychophysiological data reduction within EEGLAB.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation--a possible prelude to violence. *Science*, *289*(5479), 591-594.

De Houwer, J., & Tibboel, H. (2010). Stop what you are not doing! Emotional pictures interfere with the task not to respond. *Psychonomic Bulletin & Review*, *17*(5), 699-703.

Delgado, M. R., Nearing, K. I., LeDoux, J. E., & Phelps, E. A. (2008). Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron*, *59*(5), 829-838.

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9-21.

Delplanque, S., Silvert, L., Hot, P., & Sequeira, H. (2005). Event-related P3a and P3b in response to unpredictable emotional stimuli. *Biological psychology*, *68*(2), 107-120.

Demos, K. E., Hart, C. N., Sweet, L. H., Mailloux, K. A., Trautvetter, J., Williams, S. E., ... & McCaffery, J. M. (2016). Partial sleep deprivation impacts impulsive action but not impulsive decision-making. *Physiology & Behavior*, *164*, 214-219.

Dias-Ferreira, E., Sousa, J. C., Melo, I., Morgado, P., Mesquita, A. R., Cerqueira, J. J., ... & Sousa, N. (2009). Chronic stress causes frontostriatal reorganization and affects decision-making. *Science*, *325*(5940), 621-625.

Dinges, D. F., & Kribbs, N. B. (1991). Performing while sleepy: Effects of experimentally-induced sleepiness.

Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., ... & Pack, A. I. (1997). Cumulative sleepiness, mood disturbance and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. *Sleep: Journal of Sleep Research & Sleep Medicine*.

Dolcos, F., LaBar, K. S., & Cabeza, R. (2004). Dissociable effects of arousal and valence on prefrontal activity indexing emotional evaluation and subsequent memory: an event-related fMRI study. *Neuroimage, 23*(1), 64-74.

Donkers, F. C., & Van Boxtel, G. J. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and cognition*, *56*(2), 165-176.

Doran, S. M., Van Dongen, H. P. A., & Dinges, D. F. (2001). Sustained attention performance during sleep deprivation: evidence of state instability. *Archives italiennes de biologie*, *139*(3), 253-267.

Edinger, J. D., & Fins, A. I. (1995). The distribution and clinical significance of sleep time misperceptions among insomniacs. *Sleep: Journal of Sleep Research & Sleep Medicine*.

Evenson, K. R., Goto, M. M., & Furberg, R. D. (2015). Systematic review of the validity and reliability of consumer-wearable activity trackers. *International Journal of Behavioral Nutrition and Physical Activity*, *12*(1), 159.

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta psychologica*, *101*(2), 267-291.

Fantini, M. L., Corona, A., Clerici, S., & Ferini-Strambi, L. (2005). Aggressive dream content without daytime aggressiveness in REM sleep behavior disorder. *Neurology, 65*(7), 1010-1015.

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods*, *39*(2), 175-191.

Ferguson, C. J., & Rueda, S. M. (2009). Examining the validity of the modified Taylor competitive reaction time test of aggression. *Journal of Experimental Criminology*, *5*(2), 121.

Ferrara, M., & De Gennaro, L. (2001). How much sleep do we need?. *Sleep medicine reviews*, *5*(2), 155-179.

Fichtenberg, N. L., Putnam, S. H., Mann, N. R., Zafonte, R. D., & Millard, A. E. (2001). Insomnia screening in postacute traumatic brain injury: utility and validity of the Pittsburgh Sleep Quality Index. *American Journal of Physical Medicine & Rehabilitation*, *80*(5), 339-345.

Franzen, P. L., Buysse, D. J., Dahl, R. E., Thompson, W., & Siegle, G. J. (2009). Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. *Biological psychology*, *80*(3), 300-305.

Fritz, M. S., & MacKinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological science*, *18*(3), 233-239.

Gaines, J., Vgontzas, A. N., Fernandez-Mendoza, J., Basta, M., Pejovic, S., He, F., & Bixler, E. O. (2015). Short-and Long-Term Sleep Stability in Insomniacs and Healthy Controls. *Sleep*, *38*(11), 1727-1734.

Glenn, C. R., Blumenthal, T. D., Klonsky, E. D., & Hajcak, G. (2011). Emotional reactivity in nonsuicidal self-injury: Divergence between self-report and startle measures. *International Journal of Psychophysiology*, *80*(2), 166-170.

Goldstein, J. H., Davis, R. W., & Herman, D. (1975). Escalation of aggression: Experimental studies. *Journal of Personality and Social Psychology*, *31*(1), 162.

Goldstein, R. Z., Tomasi, D., Alia-Klein, N., Zhang, L., Telang, F., & Volkow, N. D. (2007). The effect of practice on a sustained attention task in cocaine abusers. *Neuroimage*, *35*(1), 194-206.

Gosselin, A., De Koninck, J., & Campbell, K. B. (2005). Total sleep deprivation and novelty processing: implications for frontal lobe functioning. *Clinical neurophysiology*, *116*(1), 211-222.

Grandner, M. A., Kripke, D. F., YOON, I. Y., & Youngstedt, S. D. (2006). Criterion validity of the Pittsburgh Sleep Quality Index: Investigation in a non-clinical sample. *Sleep and biological rhythms*, *4*(2), 129-136.

Grillon, C., Robinson, O. J., Mathur, A., & Ernst, M. (2016). Effect of attention control on sustained attention during induced anxiety. *Cognition and emotion*, *30*(4), 700-712.

Gujar, N., Yoo, S. S., Hu, P., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *Journal of Neuroscience*, *31*(12), 4466-4474.

Haack, M., & Mullington, J. M. (2005). Sustained sleep restriction reduces emotional and physical well-being. *Pain*, *119*(1), 56-64.

Hajcak, G., MacNamara, A., & Olvet, D. M. (2010). Event-related potentials, emotion, and emotion regulation: an integrative review. *Developmental neuropsychology*, *35*(2), 129-155.

Handley, I. M., Lassiter, G. D., Nickell, E. F., & Herchenroeder, L. M. (2004). Affect and automatic mood maintenance. *Journal of Experimental Social Psychology*, *40*(1), 106-112.

Harrison, Y., & Horne, J. A. (2000). The impact of sleep deprivation on decision making: a review. *Journal of experimental psychology: Applied*, *6*(3), 236.

Harrison, Y., Horne, J. A., & Rothwell, A. (2000). Prefrontal neuropsychological effects of sleep deprivation in young adults--a model for healthy aging. *Sleep*, *23*(8), 1067-1073.

Haynes, P. L., Bootzin, R. R., Smith, L., Cousins, J., Cameron, M., & Stevens, S. (2006). Sleep and aggression in substance-abusing adolescents: results from an integrative behavioral sleep-treatment pilot program. *SLEEP-NEW YORK THEN WESTCHESTER-*, *29*(4), 512.

Heller, A. S., Fox, A. S., Wing, E. K., McQuisition, K. M., Vack, N. J., & Davidson, R. J. (2015). The neurodynamics of affect in the laboratory predicts persistence of real-world emotional responses. *Journal of Neuroscience*, *35*(29), 10503-10509.

Horne, J. A. (1993). Human sleep, sleep loss and behaviour: Implications for the prefrontal cortex and psychiatric disorder. *The British Journal of Psychiatry*.

Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral disinhibition and the development of substance-use disorders: Findings from the Minnesota Twin Family Study. *Development and psychopathology*, *11*(04), 869-900.

Ireland, J. L., & Culpin, V. (2006). The relationship between sleeping problems and aggression, anger, and impulsivity in a population of juvenile and young offenders. *Journal of Adolescent Health*, *38*(6), 649-655.

Junker, A., Bjørngaard, J. H., Gunnell, D., & Bjerkeset, O. (2014). Sleep problems and hospitalization for self-harm: a 15-year follow-up of 9,000 Norwegian adolescents. the Young-HUNT study. *Sleep*, *37*(3), 579.

Kahn-Greene, E. T., Killgore, D. B., Kamimori, G. H., Balkin, T. J., & Killgore, W. D. (2007). The effects of sleep deprivation on symptoms of psychopathology in healthy adults. *Sleep medicine*, *8*(3), 215-221.

Kahn-Greene, E. T., Lipizzi, E. L., Conrad, A. K., Kamimori, G. H., & Killgore, W. D. (2006). Sleep deprivation adversely affects interpersonal responses to frustration. *Personality and Individual Differences*, *41*(8), 1433-1443.

Kawada, T. (2008). Agreement rates for sleep/wake judgments obtained via accelerometer and sleep diary: a comparison. *Behavior research methods*, *40*(4), 1026-1029.

Keene, O. N. (1995). The log transformation is special. *Statistics in medicine*, *14*(8), 811-819.

Konjarski, M., Murray, G., Lee, V. V., & Jackson, M. L. (2018). Reciprocal relationships between daily sleep and mood: A systematic review of naturalistic prospective studies. *Sleep medicine reviews*, *42*, 47-58.

Kopp, B., Mattler, U., Goertz, R., & Rist, F. (1996). N2, P3 and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography and clinical neurophysiology*, *99*(1), 19-27.

Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of general psychiatry*, *59*(2), 131-136.

Krizan, Z., & Herlache, A. D. (2016). Sleep disruption and aggression: Implications for violence and its prevention. *Psychology of Violence*, *6*(4), 542.

Kronholm, E., Partonen, T., Laatikainen, T., Peltonen, M., Härmä, M., Hublin, C., ... & Valve, R. (2008). Trends in self-reported sleep duration and insomnia-related symptoms in Finland from 1972 to 2005: a comparative review and re-analysis of Finnish population samples. *Journal of sleep research*, *17*(1), 54-62.

Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biological psychology*, *84*(3), 437-450.

Lemola, S., Schwarz, B., & Siffert, A. (2012). Interparental conflict and early adolescents' aggression: Is irregular sleep a vulnerability factor?. *Journal of adolescence*, *35*(1), 97-105.

León, A. G., Reyes, G. A., Vila, J., Pérez, N., Robles, H., & Ramos, M. M. (2002). The Aggression Questionnaire: A validation study in student samples. *The Spanish Journal of Psychology*, *5*(1), 45-53.

Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation a review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*, *18*(2), 84-88.

Lezak, M. D. (2004). *Neuropsychological Assessment*. Oxford University Press, USA.

Linehan, M. M. (1993). *Skills training manual for treating borderline personality disorder*. Guilford Press.

Littlefield, A. K., Sher, K. J., & Steinley, D. (2010). Developmental trajectories of impulsivity and their association with alcohol use and related outcomes during emerging and young adulthood I. *Alcoholism: Clinical and Experimental Research*, *34*(8), 1409-1416.

Lund, H. G., Reider, B. D., Whiting, A. B., & Prichard, J. R. (2010). Sleep patterns and predictors of disturbed sleep in a large population of college students. *Journal of adolescent health*, *46*(2), 124-132.

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate behavioral research*, *39*(1), 99-128.

MacLeod, C. A., & Donaldson, D. I. (2017). Investigating the Functional Utility of the Left Parietal ERP Old/New Effect: Brain Activity Predicts within But Not between Participant Variance in Episodic Recollection. *Frontiers in human neuroscience*, *11*, 580.

Massey Jr, F. J. (1951). The Kolmogorov-Smirnov test for goodness of fit. *Journal of the American statistical Association*, *46*(253), 68-78.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds? Coherence among emotion experience, behavior, and physiology. *Emotion*, *5*(2), 175.

Mauss, I. B., Troy, A. S., & LeBourgeois, M. K. (2013). Poorer sleep quality is associated with lower emotion-regulation ability in a laboratory paradigm. *Cognition & emotion*, *27*(3), 567-576.

McCall, C., & McCall, W. V. (2012). Comparison of actigraphy with polysomnography and sleep logs in depressed insomniacs. *Journal of sleep research*, *21*(1), 122-127.

McKenna, F. P., & Sharma, D. (1995). Intrusive cognitions: An investigation of the emotional Stroop task. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *21*(6), 1595.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the penn state worry questionnaire. *Behaviour research and therapy*, *28*(6), 487-495.

Miller, G. A., Lutzenberger, W., & Elbert, T. (1991). The linked-reference issue in EEG and ERP recording. *Journal of Psychophysiology*.

Montgomery-Downs, H. E., Insana, S. P., & Bond, J. A. (2012). Movement toward a novel activity monitoring device. *Sleep and Breathing*, *16*(3), 913-917.

Morris, A. M., So, Y., Lee, K. A., Lash, A. A., & Becker, C. E. (1992). The P300 event-related potential. The effects of sleep deprivation. *Journal of occupational medicine.: official publication of the Industrial Medical Association*, *34*(12), 1143-1152.

Mullins-Sweatt, S. N., Lengel, G. J., & Grant, D. M. (2013). Non-suicidal self-injury: The contribution of general personality functioning. *Personality and Mental Health*, *7*(1), 56-68.

Muzur, A., Pace-Schott, E. F., & Hobson, J. A. (2002). The prefrontal cortex in sleep. *Trends in Cognitive Sciences*, *6*(11), 475-481.

Naumann, E., Bartussek, D., Diedrich, O., & Laufer, M. E. (1992). Assessing cognitive and affective information processing functions of the brain by means of the late positive complex of the event-related potential. *Journal of Psychophysiology*, *6*, 285-285.

Nieuwenhuis, S., & Yeung, N. van den Wildenberg, & Ridderinkhof, KR (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience*, *3*(1), 17-26.

Palomba, D., Angrilli, A., & Mini, A. (1997). Visual evoked potentials, heart rate responses and memory to emotional pictorial stimuli. *International journal of psychophysiology*, *27*(1), 55-67.

Paquet, J., Kawinska, A., & Carrier, J. (2007). Wake detection capacity of actigraphy during sleep. *SLEEP-NEW YORK THEN WESTCHESTER-*, *30*(10), 1362.

Paterson, J. L., Dorrian, J., Ferguson, S. A., Jay, S. M., Lamond, N., Murphy, P. J., ... & Dawson, D. (2011). Changes in structural aspects of mood during 39–66h of sleep loss using matched controls. *Applied Ergonomics*, *42*(2), 196-201.

Pedersen, W. C., Vasquez, E. A., Bartholow, B. D., Grosvenor, M., & Truong, A. (2014). Are you insulting me? Exposure to alcohol primes increases aggression following ambiguous provocation. *Personality and Social Psychology Bulletin*, *40*(8), 1037-1049.

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences*, *13*(4), 160-166.

Pfefferbaum, A., Ford, J. M., Weller, B. J., & Kopell, B. S. (1985). ERPs to response production and inhibition. *Electroencephalography and clinical neurophysiology*, *60*(5), 423-434.

Pigeon, W. R., Pinquart, M., & Conner, K. (2012). Meta-analysis of sleep disturbance and suicidal thoughts and behaviors.

Polich, J. (2007). Updating P300: an integrative theory of P3a and P3b. *Clinical neurophysiology*, *118*(10), 2128-2148.

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior research methods, instruments, & computers*, *36*(4), 717-731.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior research methods*, *40*(3), 879-891.

Putilov, A. A. (2015). Can sleepiness be evaluated quickly, directly, objectively, and in absolute terms?. *Somnologie-Schlafforschung und Schlafmedizin*, *19*(3), 193-204.

Qi, J. L., Shao, Y. C., Miao, D., Fan, M., Bi, G. H., & Yang, Z. (2010). The effects of 43 hours of sleep deprivation on executive control functions: event-related potentials in a visual go/no go task. *Social Behavior and Personality: an international journal*, *38*(1), 29-42.

Randler, C., & Vollmer, C. (2013). Aggression in Young Adults—A Matter of Short Sleep and Social Jetlag? *Psychological reports*, *113*(3), 754-765.

Reynolds, A. C., & Banks, S. (2010). Total sleep deprivation, chronic sleep restriction and sleep disruption. *Prog Brain Res*, *185*, 91-103.

Ritter, D., & Eslea, M. (2005). Hot sauce, toy guns, and graffiti: A critical account of current laboratory aggression paradigms. *Aggressive Behavior*, *31*(5), 407-419.

Roberton, T., Daffern, M., & Bucks, R. S. (2012). Emotion regulation and aggression. *Aggression and violent behavior*, *17*(1), 72-82.

Robinson, O. J., Vytal, K., Cornwell, B. R., & Grillon, C. (2013). The impact of anxiety upon cognition: perspectives from human threat of shock studies. *Frontiers in human neuroscience*, *7*, 203.

Rossa, K. R., Smith, S. S., Allan, A. C., & Sullivan, K. A. (2014). The effects of sleep restriction on executive inhibitory control and affect in young adults. *Journal of Adolescent Health*, *55*(2), 287-292.

Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, *18*(4), 288-302.

Salthouse, T. A. (2009). When does age-related cognitive decline begin? *Neurobiology of aging*, *30*(4), 507-514.

Scharf, R. J., Demmer, R. T., Silver, E. J., & Stein, R. E. (2013). Nighttime sleep duration and externalizing behaviors of preschool children. *Journal of Developmental & Behavioral Pediatrics*, *34*(6), 384-391.

Semiz, Ü. B., Algül, A., Başoğlu, C., Ateş, M. A., Ebrinc, S., Cetin, M., ... & Günay, H. (2008). The Relationship Between Subjective Sleep Quality and Aggression in Male Subjects with Antisocial Personality Disorder.

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., ... & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *The Journal of clinical psychiatry*.

Sheridan, A., Murray, L., Cooper, P. J., Evangeli, M., Byram, V., & Halligan, S. L. (2013). A longitudinal study of child sleep in high and low risk families: Relationship to early maternal settling strategies and child psychological functioning. *Sleep medicine*, *14*(3), 266-273.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological methods*, *7*(4), 422-445.

Smith, J. L., Johnstone, S. J., & Barry, R. J. (2008). Movement-related potentials in the Go/NoGo task: the P3 reflects both cognitive and motor inhibition. *Clinical Neurophysiology*, *119*(3), 704-714.

Sprague, J., & Verona, E. (2010). Emotional conditions disrupt behavioral control among individuals with dysregulated personality traits. *Journal of Abnormal Psychology*, *119*(2), 409.

Staniloiu, A., & Markowitsch, H. (2012). Gender differences in violence and aggression–a neurobiological perspective. *Procedia-social and behavioral sciences*, *33*, 1032-1036.

Sterpenich, V., Albouy, G., Boly, M., Vandewalle, G., Darsaud, A., Balteau, E., ... & Rauchs, G. (2007). Sleep-related hippocampo-cortical interplay during emotional memory recollection. *PLoS Biol*, *5*(11), e282.

Tedeschi, J. T., & Quigley, B. M. (1996). Limitations of laboratory paradigms for studying aggression. *Aggression and Violent Behavior*, *1*(2), 163-177.

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination—relationship with negative mood and sleep quality. *Personality and Individual Differences*, *34*(7), 1293-1301.

Vallières, A., & Morin, C. M. (2003). Actigraphy in the assessment of insomnia.

Van Cauter, E., Spiegel, K., Tasali, E., & Leproult, R. (2008). Metabolic consequences of sleep and sleep loss. *Sleep Medicine*, *9*, S23-S28.

van der Zweerde, T., Lancee, J., Slottje, P., Bosmans, J., Van Someren, E., Reynolds, C., ... & van Straten, A. (2016). Cost-effectiveness of i-Sleep, a guided online CBT intervention, for patients with insomnia in general practice: protocol of a pragmatic randomized controlled trial. *BMC psychiatry*, *16*(1), 85.

Van Dongen, H. P., Maislin, G., Mullington, J. M., & Dinges, D. F. (2003). The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep

physiology from chronic sleep restriction and total sleep deprivation. *SLEEP-NEW YORK THEN WESTCHESTER-*, *26*(2), 117-129.

Van Dongen, H., Rogers, N. L., & Dinges, D. F. (2003). Sleep debt: Theoretical and empirical issues. *Sleep and Biological Rhythms*, *1*(1), 5-13.

Van Veen, V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology & behavior*, *77*(4), 477-482.

Verbruggen, F., & De Houwer, J. (2007). Do emotional stimuli interfere with response inhibition? Evidence from the stop signal paradigm. *Cognition and emotion*, *21*(2), 391-403.

Verona, E., & Bresin, K. (2015). Aggression proneness: Transdiagnostic processes involving negative valence and cognitive systems. *International Journal of Psychophysiology*, *98*(2), 321-329.

Verona, E., & Kilmer, A. (2007). Stress exposure and affective modulation of aggressive behavior in men and women. *Journal of abnormal psychology*, *116*(2), 410.

Verona, E., Patrick, C. J., & Lang, A. R. (2002). A direct assessment of the role of state and trait negative emotion in aggressive behavior. *Journal of Abnormal Psychology*, *111*(2), 249.

Verona, E., Sadeh, N., & Curtin, J. J. (2009). Stress-induced asymmetric frontal brain activity and aggression risk. *Journal of Abnormal Psychology*, *118*(1), 131.

Verona, E., Sprague, J., & Sadeh, N. (2012). Inhibitory control and negative emotional processing in psychopathy and antisocial personality disorder. *Journal of abnormal psychology*, *121*(2), 498.

Vogler, N., Perkinson-Gloor, N., Brand, S., Grob, A., & Lemola, S. (2014). Sleep, aggression, and psychosocial adjustment in male prisoners. *Swiss journal of psychology*.

Vohs, K. D., Glass, B. D., Maddox, W. T., & Markman, A. B. (2010). Ego depletion is not just fatigue: evidence from a total sleep deprivation experiment. *Social Psychological and Personality Science, 2*(2), 166-173.

Volkow, N. D., Tomasi, D., Wang, G. J., Telang, F., Fowler, J. S., Wang, R. L., ... & Swanson, J. M. (2009). Hyperstimulation of striatal D2 receptors with sleep deprivation: Implications for cognitive impairment. *Neuroimage*, *45*(4), 1232-1240.

Vyazovskiy, V. V., & Delogu, A. (2014). NREM and REM Sleep Complementary Roles in Recovery after Wakefulness. *The Neuroscientist*, *20*(3), 203-219.

Vyazovskiy, V. V., & Harris, K. D. (2013). Sleep and the single neuron: the role of global slow oscillations in individual cell rest. *Nature Reviews Neuroscience, 14*(6), 443-451.

Walker, M. P., & van Der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological bulletin*, *135*(5), 731.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of personality and social psychology*, *54*(6), 1063.

Werner, H., Molinari, L., Guyer, C., & Jenni, O. G. (2008). Agreement rates between actigraphy, diary, and questionnaire for children's sleep patterns. *Archives of pediatrics & adolescent medicine*, *162*(4), 350-358.

Wong, M. M., Brower, K. J., Fitzgerald, H. E., & Zucker, R. A. (2004). Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. *Alcoholism: Clinical and Experimental Research, 28*(4), 578-587.

Wyble, B., Sharma, D., & Bowman, H. (2008). Strategic regulation of cognitive control by emotional salience: A neural network model. *Cognition and Emotion, 22*(6), 1019-1051.

Yatani, H., Studts, J., Cordova, M., Carlson, C. R., & Okeson, J. P. (2002). Comparison of sleep quality and clinical and psychologic characteristics in patients with temporomandibular disorders. *Journal of orofacial pain*, *16*(3).

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychological review*, *111*(4), 931.

Yokomaku, A., Misao, K., Omoto, F., Yamagishi, R., Tanaka, K., Takada, K., & Kohyama, J. (2008). A study of the association between sleep habits and problematic behaviors in preschool children. *Chronobiology international*, *25*(4), 549-564.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—a prefrontal amygdala disconnect. *Current Biology*, *17*(20), 877-878.

Zhao, X., Lynch, J. G., & Chen, Q. (2010). Reconsidering Baron and Kenny: Myths and truths about mediation analysis. *Journal of consumer research*, *37*(2), 197-206.

Appendix A.

General Recruitment

(USF IRB #Pro00027233)

USF Psychology researchers seeking adults (18-30 years old) to participate in a 3-hour study on the effects of emotions on decision-making. The study consists of two sessions that are 2.5 hours each, several days apart. Payment: $35 (and transportation costs) and opportunity to participate in another paid study. Call 888-8888 or email usfeblab@gmail.com.

Appendix B.

Targeted Recruitment

(USF IRB #Pro00027233)

USF Psychology researchers seeking adults (18-30 years old) who have a history of anger or aggression problems to participate in a study on the effects of emotions on decision-making. The study consists of two sessions that are 2.5 hours each, several days apart. Payment: $35 (and transportation costs) and opportunity to participate in another paid study. Call 888-8888 or email usfeblab@gmail.com.

Appendix C.

Example General Flyer

Participants needed for paid research!

(USF IRB #Pro00027233, Principal Investigator Edelyn Verona, Ph.D.)

Looking for adults (18-30 years) to participate in a research study on the effects of emotion on decision-making and interpersonal judgments.

For completing a study that is two sessions of 2.5 hours each, you will be paid $35 (plus $10 as bonus for attending first-time scheduled appointment and extra $5 for transportation).

By participating, you will help us improve our understanding of emotions and decision-making.

Call 888-8888 or email usfeblab@gmail.com. Say that you are calling about the "emotion and behavior" study.

Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com
Emotion & Behavior Study: usfeblab@gmail.com

Appendix D.

Example Specific Flyer

Participants needed for paid research!

(USF IRB #Pro00027233, Principal Investigator Edelyn Verona, Ph.D.)

Looking for adults (18 to 30 years) with a history of problems controlling their anger and aggression to participate in a research study on the effects of emotion on decision-making and interpersonal judgments.

For completing a study that is two sessions of 2.5 hours each, you will be paid $35 (plus $10 as bonus for attending first-time scheduled appointment and extra $5 for transportation).

By participating, you will help us improve our understanding of emotions and decision-making.

Call 888-8888 or email usfeblab@gmail.com. Say that you are calling about the "emotion and behavior" study.

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Emotion & Behavior Study: usfeblab@gmail.com

Appendix E.

SCREENING PROCEDURES

USF PARTICIPANTS: PRE-SCREENING CONSENT FOR PHONE SCREENING SCRIPT

Introduction:

Hello, my name is [_____]. I am a member of the research team at the University of South Florida.

Thank you for your interest in our research study! We are conducting a study on individuals, some of whom may have problems with aggression or anger.

First, can I ask your age? [[Ask first to see if they meet age eligibility, if not we should not continue with the rest of the script to save the participant the time and effort]]

Participant Age (must be between 18 and 30 years): _____ years

If participants do not meet age inclusion criteria: I regret to inform you that you do not meet the requirements to participate in this study, but we do want to thank you for contacting us and expressing interest in this project. Thank you for your time!

If participants do meet age inclusion criteria: OK, thank you. We are studying the effects of emotions on decision-making in a study with two sessions that last about 2.5 hours each. The study involves answering question and interviews about your emotions and behaviors and completing computer tasks. During the task, you will have an electrode cap placed on your head to record your EEG (brain) waves. During this task, you will also experience minor shocks. These shocks may be irritating, but they are neither painful nor harmful. If you experience major discomfort during the study and wish to discontinue participation, you may do so at any time. You receive $35 for participating (plus $10 bonus for showing up the first time you are scheduled and $5 transportation reimbursement). You will also have the opportunity to participate in another paid study.

You will also have the opportunity to participate in another study be asked to complete an interpersonal judgment task, in which you and another participant will rotate roles of being an employee and supervisor. During this task, you may be asked to provide feedback via shocks as to the correctness of the employee's responses on a memory task.

Finally, you will also be asked to provide contact information for 1-2 close friends/relatives or romantic partners. Study personnel will contact these individuals to ask them questions about your recent behaviors. Allowing us to contact your friends/relatives/significant others will improve our way of measuring your emotions and behaviors. This task takes another 1-1.5 hours and you will be paid $35, plus $10 bonus if you complete both tasks in the same session or within one week of each other.

How does this sound? Do you want to continue with questions to see if you are eligible for the study?

Before you can enroll in the study, we need to ask you a few questions to determine if you meet certain requirements to participate. All information discussed will be confidential. You may refuse to answer any question and stop this interview at anytime. I will begin with the questions, would you like to continue with the screening questions?

Gender: M F

Is English your native language?

_____

If not, can you read and write in English? YES NO

In your lifetime, have you used any types of drugs (e.g., marijuana, cocaine, heroin, meth) If they have, ask which drugs and how often?

_____

_____

Have you used any types of drugs (e.g., marijuana, cocaine, heroin, meth) in the past month? If they have, ask which drugs and how often?

_____

_____

Have you drunk alcohol in the past month? If they have, ask how much and how often?

_____

_____

Do you have a history of problems controlling your anger and/or aggression? YES NO

If YES, please describe the nature of these problems?

_____

_____

_____

Have you ever been diagnosed or are you currently diagnosed with a psychological disorder, such as depression, schizophrenia, or bi-polar disorder? YES NO

If YES, please describe what you have or are diagnosed with and when you received this diagnosis?

_____

_____

_____

Are you currently taking any psychiatric medications for mental health reasons? YES NO

If YES, please what are you taking and why are taking it?

_____

_____

_____

Do you have any question or concerns regarding your participation in this study? YES NO

For use by study personnel only:

Does this participant meet criteria for this study? YES NO

If participants meet inclusion/eligibility criteria: You meet the requirements to participate in this study. Would you like to be scheduled to attend your first session?

Yes: May I get your name, telephone number (home and cell), and email address? How would you prefer to be contacted? Available dates to begin sessions at your institution are [Dates] at [Times], which option would be work best with your schedule? Thank you for volunteering to participate in this study, and we will see you at the session scheduled on [Date] at [Time] for approximately 3 hours where you will be provided more information and asked to sign a consent form.

Do you have questions for me at this time? If you later decide you have any questions, please contact the research team at ()_____. Thank you.

No: Thank you for your time. Please contact us at ()_____, if you change your mind about being a participant in this study. The information we have collected from you today will not be used and will be kept confidential.

If participants do not meet inclusion criteria: I regret to inform you that you do not meet the requirements to participate in this study, but we do want to thank you for contacting us and expressing interest in this project. We will destroy all information you have shared with us during this phone call. Thank you for your time!

Appendix F.

IRB Approval

USF
UNIVERSITY OF SOUTH FLORIDA

RESEARCH INTEGRITY AND COMPLIANCE
Institutional Review Boards, FWA No. 00001669
12901 Bruce B. Downs Blvd., MDC035 • Tampa, FL 33612-4799
(813) 974-5638 • FAX (813) 974-7091

5/15/2017

Melanie Bozzay
Psychology
[redacted]
Tampa, FL 33647

RE: **Expedited Approval for Initial Review**
IRB#: Pro00030534
Title: Linking Sleep and Aggression: The Role of Response Inhibition and Negative Emotional Processing

Study Approval Period: 5/15/2017 to 5/15/2018

Dear Mrs. Bozzay:

On 5/15/2017, the Institutional Review Board (IRB) reviewed and **APPROVED** the above application and all documents contained within, including those outlined below.

Approved Item(s):
Protocol Document(s):
Sleep and Behavior Protocol_v2.0_5.8.17_Clean.docx

Consent/Assent Document(s)*:
Consent Form Community Participants_v2.0_5.8.17_Clean.docx.pdf
Consent Form SONA Participants_v2.0_5.8.17_Clean.docx.pdf

*Please use only the official IRB stamped informed consent/assent document(s) found under the "Attachments" tab. Please note, these consent/assent documents are valid until the consent document is amended and approved.

It was the determination of the IRB that your study qualified for expedited review which includes activities that (1) present no more than minimal risk to human subjects, and (2) involve only procedures listed in one or more of the categories outlined below. The IRB may review research through the expedited review procedure authorized by 45CFR46.110 and 21 CFR 56.110. The research proposed in this study is categorized under the following expedited review category:

(4) Collection of data through noninvasive procedures (not involving general anesthesia or sedation) routinely employed in clinical practice, excluding procedures involving x-rays or microwaves. Where medical devices are employed, they must be cleared/approved for marketing.

(5) Research involving materials (data, documents, records, or specimens) that have been collected, or will be collected solely for nonresearch purposes (such as medical treatment or diagnosis).

(7) Research on individual or group characteristics or behavior (including, but not limited to, research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices, and social behavior) or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies.

As the principal investigator of this study, it is your responsibility to conduct this study in accordance with IRB policies and procedures and as approved by the IRB. Any changes to the approved research must be submitted to the IRB for review and approval via an amendment. Additionally, all unanticipated problems must be reported to the USF IRB within five (5) calendar days.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638.

Sincerely,

John A Schinka, Ph.D.

John Schinka, Ph.D., Chairperson
USF Institutional Review Board

Appendix G.

Sleep Diary

Complete in Morning

Day of Week: _____

I went to bed last night at: _____ AM / PM

I got out of bed this morning at: _____ AM / PM

Last night I fell asleep: ☐ Easily ☐ After some time ☐ With difficulty

I woke up during the night ____ times and for _____ minutes

Last night I slept a total of: _____ hours and _____minutes

My sleep was disturbed by *(list mental or physical factors including noise, lights, pets, allergies, temperature, discomfort, stress, etc.)*:

When I woke up for the day, I felt: ☐ Refreshed ☐ Somewhat refreshed ☐ Fatigued

Yesterday, I consumed caffeinated drinks in the:

☐ Morning ☐ Afternoon ☐ Evening ☐ NONE

I consumed _____ caffeinated drinks yesterday

I took a nap yesterday: Yes / No

If yes, I napped for _____ minutes

During the day yesterday, how likely was I to doze off while performing daily activities:

☐ No chance ☐ Slight chance ☐ Moderate chance ☐ High chance

Throughout the day yesterday, my mood was:

☐ Very pleasant ☐ Pleasant ☐ Unpleasant ☐ Very unpleasant

Approximately 2-3 hours before going to bed, I consumed:

Alcohol: Yes/No

A heavy meal: Yes/No

Caffeine: Yes/No

In the hour before going to bed, my bedtime routine included *(list activities including reading a book, using electronics, taking a bath, doing relaxation exercises, etc.):*

I wore the Fitbit all day yesterday: Yes/No

I wore the Fitbit while sleeping last night: Yes/No

Indicate the extent to which you felt this way over the past day:

1	2	3	4	5
Very Slightly or Not at All	A Little	Moderately	Quite a Bit	Extremely

1. Distressed

2. Upset

3. Scared

4. Hostile

5. Irritable

6. Alert

7. Nervous

8. Afraid

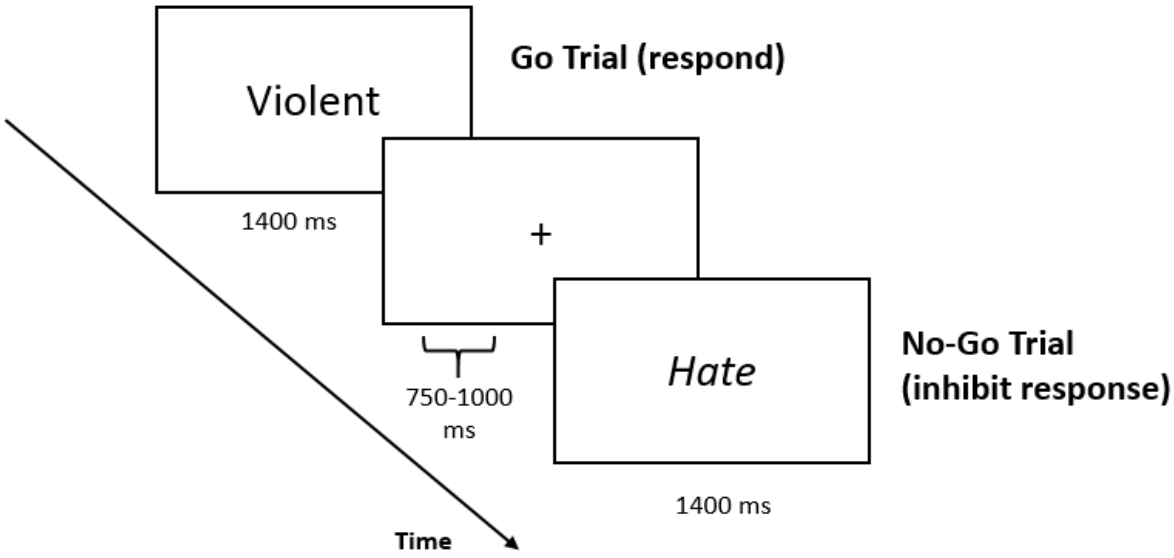
Use this scale to answer the next several questions:

1	2	3	4	5	6	7
None at all	Slight, a very mild urge	Mild urge	Moderate urge	Strong urge, but easily controlled	Strong urge and difficult to control	Strong urge and would have engaged in the behavior

1. At its most severe point, how strong was your urge to hurt yourself yesterday?
    a. Did you hurt yourself yesterday?
    b. If yes, how many times?
2. At its most severe point, how strong was your urge to drink alcohol yesterday?
    a. Did you drink alcohol yesterday?
    b. If yes, how many drinks?
3. At its most severe point, how strong was your urge to use drugs yesterday?
    a. Did you use drugs yesterday?
    b. If yes, what did you use?
    c. If yes, how much did you use?
4. At its most severe point, how strong was your urge to be aggressive yesterday?
    a. Were you physically or verbally aggressive towards someone else yesterday?
    b. If yes, what did you do?
        i. Yelled at someone
            1. How many times?
        ii. Hit or fought with someone physically
            1. How many times?

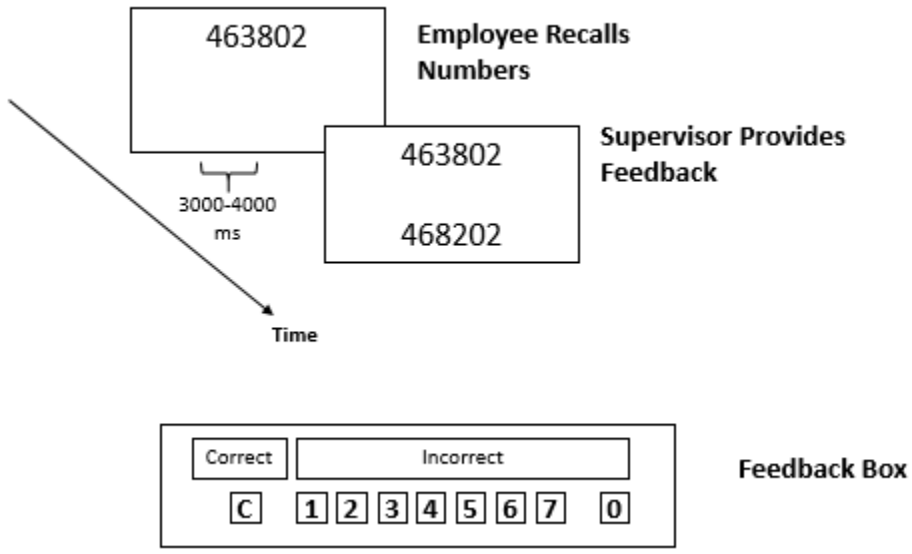
Appendix H.

Emotional Go/No-Go Task



Appendix I.

Phase 2 of the Aggression Paradigm



Appendix J.

POST-STUDY QUESTIONNAIRE
("Supervisor" Version)

Instructions: This form is used to evaluate the experimental procedure. Please answer the following questions, and feel free to be as honest as you can. Your responses to these items will have no bearing on your experimental credits or compensation for participation.

OVERALL EXPERIENCE

1. Please rate your overall lab experience today on the following dimensions

Boring	1	2	3	4	5	6	7	*Exciting*
Uninteresting	1	2	3	4	5	6	7	*Interesting*
Easy	1	2	3	4	5	6	7	*Difficult*
Uninvolving	1	2	3	4	5	6	7	*Involving*
Wasteful	1	2	3	4	5	6	7	*Useful*

2. Please rate your perceptions of the lab personnel (i.e., the lab experimenter(s) that conducted your session).

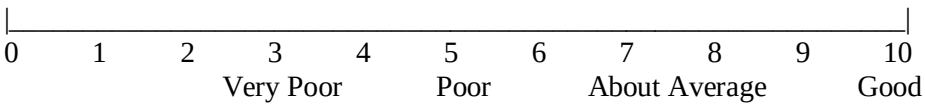
lazy	1	2	3	4	5	6	7	*hardworking*
unprofessional	1	2	3	4	5	6	7	*professional*
irresponsible	1	2	3	4	5	6	7	*responsible*
not helpful	1	2	3	4	5	6	7	*helpful*
inefficient	1	2	3	4	5	6	7	*efficient*
lethargic	1	2	3	4	5	6	7	*energetic*
untrustworthy	1	2	3	4	5	6	7	*trustworthy*
reserved	1	2	3	4	5	6	7	*outgoing*

3. If you interacted with any other participants as part of this experiment, please give your reactions of the other participant(s).

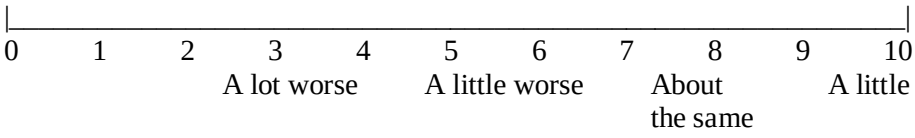
Unattractive	1	2	3	4	5	6	7	*Attractive*
Incompetent	1	2	3	4	5	6	7	*Competent*
Rude	1	2	3	4	5	6	7	*Friendly*
Immature	1	2	3	4	5	6	7	*Mature*
Unlikable	1	2	3	4	5	6	7	*Likable*

EMPLOYEE-SUPERVISOR TASK

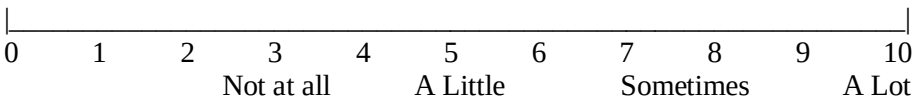
4. What was your impression of the other participant's (employee's) performance during the digit-span task?



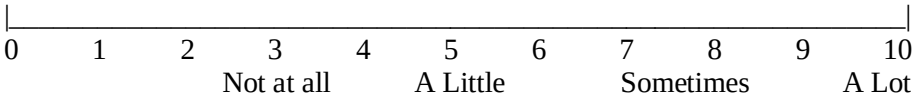
5. How well do you think you could have done on the digit span task that the other participant (employee) worked on during the task?



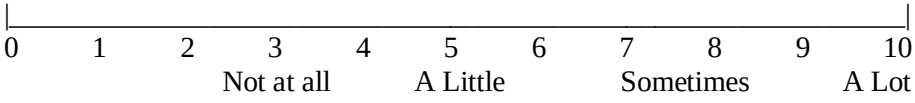
6. To what extent did you utilize the shock levels to encourage better performance in the employee?



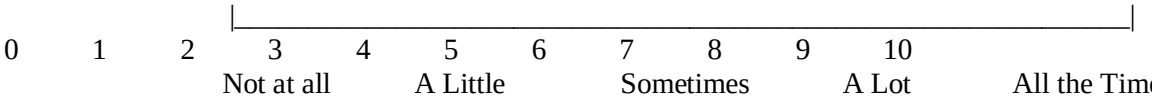
7. To what extent did you choose higher shock levels because you were upset at the employee or about the employee's performance?



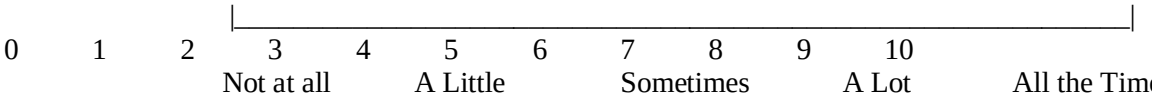
8. To what extent did you choose the no shock button because you did not want to hurt the employee?



9. To what extent did you choose the no shock button because you did not think this would help the employee perform better?



10. To what extent did you choose the no shock button because you were not upset at the employee?



GENERAL QUESTIONS AND CONCERNS

11. Had you heard of this research project before from friends or other people? Please explain.

_____
_____
_____

12. Did you have any concerns about the experimenter's explanation for the purpose of the study? Please explain.

_____
_____
_____

13. Did the experimental procedures involve deception or deviate in any way from what you were told? If so, please describe the exact nature of the deceptions or deviations.

_____
_____
_____

14. Describe any unusual or potentially harmful aspects of this experiment (if any).

_____
_____
_____

15. What do you think we were trying to investigate in this study?

_____
_____
_____

16. Indicate how this experiment might be improved to make it more valuable for the participants.

_____
_____
_____

Appendix K.

Post-Study Interview

1. What was your overall experience of the different tasks?

2. The interpersonal judgment task or writing the essay:
    - Did you find it difficult writing the essay?
    - Would you have rather been in the rater role?

3. Did you enjoy or not enjoy doing the experiment with another participant?

Employee-Supervisor Task:

4. How did you determine which button or shock levels to administer in the employee-supervisor task? Did you have a pattern or strategy?

5. Did you notice a difference in your pattern of responding or decision-making from one block to another?

6. Did you ever become annoyed or distressed at the employee's performance? When did this happen? What did you do when you felt annoyed at him/her?

7. Anything unusual about the experiment that made you wonder about things? Anything out of the ordinary?

Appendix L.

Participant Demographics Form

Participant Information Sheet

Instructions: Please fill out or circle the following information about yourself. All responses are completely confidential.

I. Current Information:

1. **Zip Code: _____**
2. **Age: _____**
3. **Sex:**
   0 = female

   1 = male

   2= Transgender (male to female)

   3= Transgender (female to male)

   4=Other (please describe) _____

4. **Ethnicity:**
   1 = Hispanic
   2 = Not-Hispanic
5. **Race:**
   1 = Caucasian (White) 5 = Pacific Islander or Hawaiian

   2 = African American (Black) 6 = Native American

   3 = Asian descent 7 = Mixed ethnicity

   4 = Latino/a 8 = Middle Eastern/North African

   9 = Other: _____

6. **Household income (if you are a full time student or dependent, select your parent/guardian household income):**
   1 = less than $15,000 4 = $45,001 – 60,000

2 = $15,000-30,000

3 = $30,001 – 45,000

5 = $60,001 – 75,000

6 = Over $75,000

7. **What is your occupation (job)?**
    1. Service worker or laborer, for example maid, bellhop, janitor, stock handler, farm laborer, car washer, entry-level factory work, unemployed for long periods of time
    2. Untrained worker, for example restaurant help (busboy, waiter/waitress), bartender, cook, waste management (garbage collector), gardener, parking attendants
    3. Machine operators and semi-skilled worker, for example machine operator / excavation, painter, barber, bus driver, chauffer, child care worker, hairstylist/beautician, health or nurse aide/assistant, butcher, roofer, taxicab driver, truck driver, non-commissioned soldier, housekeeper
    4. Skilled manual worker, for example tenant farmers, small business owner, flight attendant, plumber, carpenter, decorator detective, dry wall/carpet installer, electrician, firefighter, machinist, mail carrier, mechanic, police/law enforcement, receptionist, tailor, welder, jeweler, meter reader, repairmen
    5. Clerical and sales worker, for example secretary, bank teller, bookkeeper, recreation worker, library attendant, bill account collector
    6. Technician and semi-professional, for example medium-size farm owner, advertising agent, dental hygienist, legal secretary, foremen, photographer, sheriff, occupational therapist, construction inspector, air traffic controller
    7. Manager and other professional, for example actor or entertainer, computer programmer, funeral director, office/sales manager (not retail), public relations, insurance adjustor, realtor, reporter, social worker, elementary or middle school teacher, vocational counselor
    8. Administrator and technical professional, for example district manager of large business, accountant, professional clergy, chiropractor, pharmacist, registered nurse, high school principal or high school teacher, computer analyst, airplane pilot, author /editor
    9. Executive and major professional, for example the chairperson, (vice-)president, owner or treasurer of large business, corporation, or farm; lawyer, judge, doctor, college professor, engineer, architect dentist, commissioned officer (major, lieutenant, commander)
    10. Homemaker
    11. Other (specify) _____ (e.g., retired)

8. **How did you hear about our study?**

    1 = Flyer in community (coffee shop, supermarket, etc.)

    2 = Newspaper Ad

    3 = Craigslist Ad

4 = Contacted via mail, email, and/or phone from our research team

5 = Participated in previous research study

6 = Heard about it from a friend or relative

Who told you about the study (e.g. friend, sister, etc.)? _____

7= Other (Please describe): _____

9. **Are you currently taking any medications for a psychological condition (for example, depression, schizophrenia, anxiety)?**
   1 = Yes

   0 = No

   If yes, what are the medications and/or what are they for?

   1 _____

   2 _____

   3 _____

   4 _____

   5 _____

10. **In the last <u>48 hours</u>, have you used any types of drugs (e.g., marijuana, cocaine, heroin, meth, pain pills)**
    1 = Yes
    0 = No

    If yes, which drugs did you take?

    1 _____

    2 _____

    3 _____

    4 _____

    5 _____

11. **In the last <u>48 hours</u>, have you drunk alcohol?**
    1 = Yes
    0 = No

    If yes, how much did you drink (if you drank more than one type of drink, please indicate how much you drank of each type)?

    1 _____

    2 _____

    3 _____

    4 _____

    5 _____

12. **Do you smoke (i.e., cigarettes, e-cigarettes)?**
    1 = Yes
    0 = No

    If yes:

    What do you smoke (please circle)? Cigarette E-Cigarettes Both

    How frequently do you smoke?

    _____

    How much do you smoke each day (e.g., one pack)?

    _____

    When was the last time you smoked?

    _____

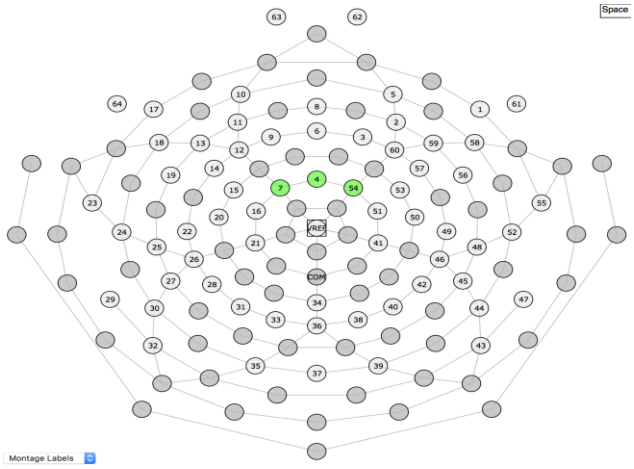
    Will you need smoke breaks during the study?

    1 = Yes
    0 = No

Appendix M.

Montages used in data analyses:

Frontocentral:



Parietal: