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Repetitively counting sheep: Sleep as a moderator of executive function performance on obsessive-compulsive symptoms

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REPETITIVELY COUNTING SHEEP: SLEEP AS A MODERATOR OF EXECUTIVE FUNCTION PERFORMANCE ON OBSESSIVE-COMPULSIVE SYMPTOMS

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ABSTRACT

As a leading cause of disability worldwide, Obsessive-Compulsive Disorder (OCD) is associated with considerable costs on individual and economic levels. According to a U.S. national comorbidity survey, approximately 28% of individuals experience obsessive-compulsive (OC) symptoms in their lifetime. As with most psychiatric disorders, sleep disturbances are highly prevalent in individuals with OCD and have been linked to greater severity of OC symptoms and poorer treatment response. Similarly, deficient executive functioning (EF) has been demonstrated in OCD, with research evidencing a connection between EF impairment and OCD course, symptom severity, and treatment response. Sleep difficulties are also implicated in impaired EF, as the primary brain region responsible for EF (i.e., the prefrontal cortex) seems to be particularly vulnerable to inadequate sleep. Given high dropout rates and residual symptoms following OCD treatment, a better understanding of these relations (OCD, EF, and sleep) might contribute to improved treatment success. The current study examined associations among these constructs, hypothesizing that sleep impairment would moderate the relationship between EF performance and OC symptom severity. A nonclinical sample of university undergraduates and community members ($N = 91$; $M_{age} = 25.87$; $SD = 12.50$; 86.8% White; 68.1% female) completed a series of online self-report measures and computerized cognitive performance tasks. Though, as expected, both sleep and depressive symptoms significantly predicted OC symptom severity, EF performance was not associated with other variables of interest at even the basic correlational level. Extant literature points to enumerable factors (e.g., clinical symptom levels, use of OC-relevant stimuli in EF tasks,

comorbid disorders, medication effects, etc.) potentially contributing to the EF-OCD relationship, particularly where sleep is concerned. Perhaps, EF deficits emerge once symptoms have reached clinical severity, which only a small portion of the current sample endorsed. Limited symptom variance, remote data collection, and videoconferencing methodology also likely contributed to null findings. Future research should extend this study to an in-person, laboratory paradigm using clinical samples. As a relatively unstudied area with potential to better understand the experience and course of OCD, continued research is needed to investigate specific emotional and behavioral elements impacting the EF-OCD relationship with co-occurring sleep factors.

LIST OF ABBREVIATIONS AND SYMBOLS

ACC	Anterior Cingulate Cortex
ADHD	Attention-Deficit/Hyperactivity Disorder
CSTC	Cortico-Striatal-Thalamic-Cortical
DASS-21	Depression, Anxiety, Stress Scale-21
DOCS	Dimensional Obsessive-Compulsive Scale
DSP	Delayed Sleep Phase
EF	Executive Function
NREM	Non-Rapid Eye Movement
OC	Obsessive-Compulsive
OCD	Obsessive-Compulsive Disorder
OFD	Orbitofrontal Cortex
PEBL	Psychology Experiment Building Language
PFC	Prefrontal Cortex
PSQI	Pittsburgh Sleep Quality Index
PTSD	Post-Traumatic Stress Disorder
REM	Rapid Eye-Movement
SWS	Slow-Wave Sleep
VST	Victoria Stroop Test

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CHAPTER 1

INTRODUCTION

Obsessive-Compulsive Disorder

Obsessive-Compulsive Disorder (OCD) is a psychiatric disorder comprised of obsessions and/or compulsions experienced as time consuming (> 1 hour per day), distressing, and functionally impairing across one or more settings [i.e., social, occupational, domestic, etc.; American Psychiatric Association (APA), 2013]. Symptoms of OCD include intrusive and excessive thoughts, images, or impulses (i.e., obsessions), which elicit emotional distress and often lead the afflicted to perform ritualistic behaviors (i.e., compulsions) aimed at decreasing that distress (APA, 2013). OCD has a mean age at onset of 19.5 years, with approximately a quarter of cases beginning by age 14. Gender differences, however, have revealed that males typically have a younger age at onset, with about 25% experiencing initial symptoms prior to age 10 (APA, 2013). This disorder is relatively common (Sadock et al., 2014; Toobaei et al., 2015) and is observed worldwide across various cultures and socioeconomic statuses (Goodman et al., 2000). Although the estimated lifetime prevalence of OCD is reportedly between 2 - 3% (Fettes et al., 2017), results from a United States nationally representative sample of those experiencing obsessive-compulsive (OC) symptoms in their lifetime appears to be much higher (28.2%; Ruscio et al., 2010).

Rated as one of the top ten leading causes of disability worldwide [The World Health Organization (WHO), 2017], OCD can be quite debilitating and has been associated with

substantial costs on both individual and economic levels. For instance, those with OCD often report diminished quality of life and considerable functional impairment, particularly regarding occupational, social, and domestic/family roles. According to the National Institute of Mental Health (2017), approximately 51% of adults with OCD experience severe functional impairment, followed by about 35% with moderate and 14% with mild impairment. In fact, findings from an epidemiological study by Ruscio et al. (2010) assessing for 12-month and lifetime OCD prevalence ($N = 2073$) indicated that most 12-month respondents primarily fell within moderate to severe impairment ranges based on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). Additionally, 65% of 12-month respondents reported severe role impairment on the Sheehan Disability Scales (SDS; Leon et al., 1997). Furthermore, lifetime respondents reported spending an average of 5.9 hours and 4.6 hours per day occupied by or involved in obsessions and compulsions, respectively. In addition to the functional burden experienced by the individual, economic burdens are apparent as well. A 1990 epidemiological survey estimated the direct and indirect costs of OCD accounted for yearly expenses of about $8.4 billion in the U.S. This figure comprised approximately 6% of the total cost for all mental illness nationwide. Moreover, indirect costs related to U.S. productivity loss due to those suffering with OCD were an estimated $6.2 billion that year (DuPont et al., 1995).

Unfortunately, although the overall impact is significant, many do not seek treatment until 10 - 11 years following symptom onset (Hollander et al., 1997; Stengler et al., 2013). Additionally, although individuals may seek professional help, this often leads to non-effective interventions, with evidence suggesting an average 17-year period between symptom onset and successful treatment (Hollander et al., 1997) in those suffering from OCD. In consequence, research has shown low remission rates in the absence of effective treatment, often resulting in a

chronic course of the disorder (Lovell et al., 2017). As OCD is highly comorbid with other psychiatric disorders, co-occurring symptoms tend to further complicate its course. Some of the more prevalent comorbidities include: anxiety disorders (76%), mood disorders (63%), impulse-control disorders (56%), and substance-use disorders (39%; DSM-5, 2013; Ruscio et al., 2010).

Along with high comorbidity rates, OCD is a heterogeneous disorder with much disagreement regarding its pathophysiology. Neurobiological research has begun aiming at parsing apart this complicated disorder and its neurological complexities (Maia et al., 2008). Such research investigating treatment effectiveness for OCD has revealed evidence of changes in neurological network functioning (Moody et al., 2017), suggesting the contribution of particular brain regions to symptom expression and maintenance. Although the exact pathophysiological nature of OCD is still unclear, there is general consensus regarding dysfunction of distinct neural loops of the cortico-striatal-thalamic-cortical (CSTC) circuits (Fettes et al., 2017). These circuits originate in particular areas of the cortex (e.g., dorsolateral prefrontal cortex, anterior cingulate cortex/ventromedial prefrontal cortex, lateral orbitofrontal cortex), pass through parts of the basal ganglia in the striatum (e.g., nucleus accumbens, caudate nucleus, putamen), as well as the thalamus, before terminating through feedback projections in the cortex (Fettes et al., 2017).

Although a number of corticostriatal pathways have been implicated in OCD, the CSTC circuitry most often suggested is that involving the orbitofrontal loop (Fettes et al., 2017). This particular loop consists of the orbitofrontal cortex (OFC), the anterior cingulate cortex (ACC), and the caudate nucleus of the basal ganglia (Chamberlain et al., 2005). The OFC is an area of the prefrontal cortex (PFC) responsible for higher-order cognition (e.g., planning, decision-making, response inhibition, learning, focusing attention), affect regulation, and behavior (Stalnaker et al., 2015). Similarly, functions of the ACC involve mediating cognitive influences

on emotion and affect regulation, as well as the management of social behavior (e.g., empathy, impulse control, decision-making; Stevens et al., 2011). The caudate nucleus is implicated in learning, especially in memory storage and processing, using information from prior experiences to influence future behavior and decision-making. This structure in particular seems to be involved in the performance of repetitive, stereotyped behaviors in studies with non-human animals (Chamberlain et al., 2005).

Neuroimaging studies demonstrate hyperactivity in striatal components of CSTC circuits, which are responsible for driving compulsive behavior, and hypoactivity in cortical structures of the PFC, responsible for inhibiting behavioral responses. This neurological hyperactivity is seen both during rest and during symptom provocation in those with OCD (Bhikram et al., 2017), and thus, suggests anatomical abnormalities that are specific to this disorder (Brambilla et al., 2002).

However, neurological abnormalities are only one component of the overall etiological picture of OCD, which is more likely a contribution of a myriad of factors. According to the biopsychosocial model, the development and experience of psychological disorders are understood through the interplay of the following factors: 1) biological (e.g., physical health, genetic vulnerabilities, neurological functioning), 2) psychological (e.g., thoughts, beliefs, perceptions), and 3) environmental (e.g., cultural factors, social interactions). In addition to the neuroanatomy specific to OCD, other biological factors include those associated with genetics and pediatric infections. For instance, twin studies have revealed moderate heritability of the disorder (Abramowitz et al., 2009), with a concordance rate of 57% for monozygotic twins and 22% for dizygotic twins. Additionally, OCD is twice as likely to occur in first-degree relatives of adults with OCD, the likelihood of which increases 10-fold when symptom-onset occurs in childhood or early adolescence (APA, 2013). Regarding pediatric infections, there are also

instances when childhood OCD occurs as a result of streptococcal infection due to inflammation of the basal ganglia (Abramowitz et al., 2009).

Psychological factors refer to the thoughts, beliefs, and perceptions about oneself, others, one's environment, and one's experiences (Abramowitz et al., 2009). Related to OCD, contemporary cognitive theories propose that obsessions and compulsions result from specific types of dysfunctional beliefs which have been empirically demonstrated: 1) perfectionism and intolerance of uncertainty, 2) overemphasis of and the need to control one's thoughts, and 3) exaggerated responsibility and overestimation of threat (Taylor & Jang, 2011). It is suggested that the strength of such dysfunctional beliefs contribute to the likelihood a person will develop obsessions and compulsions (Abramowitz et al., 2009). For example, most individuals experience thought intrusions on a regular basis, but it is the appraisal of such an intrusion as personally significant, harmful or threatening, unacceptable, or as something the person is responsible for, which results in the development of obsessions. Thus, compulsions arise in attempts to neutralize, remove, or prevent obsession-related outcomes (Abramowitz et al., 2009).

Lastly, the biopsychosocial model suggests that environmental causes consist of domains such as interpersonal relationships, stressors, and early life experiences. Traumatic events, like physical and sexual abuse in childhood, are cited as risk factors for the development of OCD (APA, 2013). In addition, interpersonal and social aspects may considerably impact the expression and severity of OCD. In particular, the manner in which family members and meaningful others (e.g., friends, teachers, coworkers) respond to the individual can serve to both increase or decrease symptom distress (March & Mulle, 1998). In fact, twin studies investigating adults with OCD suggest that 53 – 73% of the total variance in symptoms is due to the unique contribution of environmental factors (van Grootheest et al., 2007).

Much research has investigated and highlighted the contributions of each biopsychosocial component, and it is generally accepted that the influences of these elements on the overall etiology of OCD are multifaceted (Abramowtiz & Reuman, 2020; Taylor & Jang, 2011). However, Taylor and Jang (2011) were the first to test the integration of genetic-environmental and cognitive-behavioral perspectives into a single empirically supported model of OCD etiology. Their results suggested that genetic and environmental factors impact dysfunctional beliefs and OC symptoms, and that dysfunctional beliefs also impact symptoms. While preliminary, these findings provide additional support for the direct and indirect influences each etiological element has on OCD symptomatology (Taylor & Jang, 2011), suggesting continued exploration of factors contributing to the development and experience of the disorder is needed.

Sleep Disturbance

Although typically thought of as a period of inactivity, research illuminates the crucial function of sleep in overall health and wellbeing. Adequate sleep is a key component to maintaining both physical and mental health, as it supports proper immune function, metabolism, cognitive processes, as well as positive mood and adaptive emotion regulation (Nagy et al., 2020; Nienstedt & Alic, 2013). Inadequate sleep, however, has been associated with increased risk for a number of chronic medical illnesses, increased mortality rates, as well as many psychiatric illnesses (Munafo et al., 2018; Nienstedt & Alic, 2013; Tsuno et al., 2005).

Based on recommendations by the National Sleep Foundation (2015), the average adult aged 18 – 64 requires between seven and nine hours of sleep per night. However, approximately 30% of adults in the U.S. report typically obtaining less than the recommended amount [i.e., ≥7 hours per night; APA, 2013; Centers for Disease Control and Prevention (CDC), 2020]. Sleep is considered insufficient when it is of short duration (i.e., <5 – 6 hours per night), fragmented (i.e.,

short nocturnal sleep interruptions prevent the physical consolidation of sleep), or when a person experiences selective sleep stage deprivation (i.e., loss of certain physiological sleep stages; Paavonen et al., 2016). Sleep disturbance, or primary insomnia, is defined as one's dissatisfaction with the quality and quantity of sleep which causes considerable distress and daily functional impairment. Sleep disturbances can occur at various points throughout the sleep period and manifests as difficulties with sleep onset (i.e., initial insomnia; >30 minutes), sleep maintenance (i.e., middle insomnia), and early morning awakenings (i.e., late insomnia). Sleep disturbances have also been associated with feeling tired and unrested the next day (i.e., nonrestorative sleep; APA, 2013).

Approximately one third of U.S. adults experiences some form of insomnia-related symptoms (APA, 2012). A 2019 National Health Interview Survey (NHIS) reported that approximately 20% of the U.S. population suffer from chronic insomnia, with about 36% reporting at least one symptom of insomnia in the last 12 months. Furthermore, about 10 – 15% of people experience daytime impairment (e.g., social interactions, work performance) such as irritability and alterations in mood, low energy and fatigue, and cognitive impairment (APA, 2012).

Sleep deprivation or obtaining an inadequate amount of sleep for a given individual is termed sleep deprivation (Nienstedt & Alic, 2013). Those experiencing sleep deprivation are at a greater risk of developing diabetes, hypertension, cardiovascular disease (Nienstedt & Alic, 2013), obesity, cancer (Munafo et al., 2018), as well as immune system decline (Banks & Dinges, 2007). Sleep deprivation has also been shown to result in delayed reaction times (e.g., involved in driving or hand-eye coordination tasks) and can negatively affect decision-making and impulse control (Nienstedt & Alic, 2013). As such, inadequate sleep has been associated

with increased mortality rates, with individuals obtaining less than five hours of sleep per night being at a 15% increased chance of death from all causes (e.g., chronic health conditions, human-error-related accidents; Munafo, et al., 2018). In fact, human-error-related accidents have reported estimated annual economic costs between $43 and $56 billion (Goel et al., 2013). Additionally, a 2004 report by the U.S. Surgeon General determined that approximately 70 million Americans are impacted by sleep-related disturbances, constituting $16 billion in health-care costs and $50 billion in productivity loss annually (Quan, 2005).

In addition to physical health, sleep has been implicated in proper emotional and psychological functioning. Regarding mental health, even moderate sleep deprivation can lead to an increased experience of and reaction to stress and can have negative consequences on mood (Chang, Pien, Duntley, & Macones, 2010; Nienstedt & Alic, 2013). Furthermore, poor sleep plays a role in many psychiatric disorders (e.g., major depression, anxiety disorders, schizophrenia, substance use disorders; Gillin, 2007; Perkins et al., 2000; Tsuno et al., 2005) and has been cited as a risk factor for suicidality (Turvey et al., 2002). Specifically, research shows that individuals with chronic insomnia are at a six times greater chance of developing an anxiety disorder and a forty times increased chance of experiencing major depression (Ford & Kamero, 1989; Johnson et al., 2006). Sleep seems to be involved in one's capacity for adaptive emotion regulation (i.e., the ability to control or modulate emotions in contextually appropriate manners; Suveg & Zeman, 2004), a transdiagnostic factor demonstrated in numerous emotional disorders. Also, studies have found poor sleep quality to be related to emotion regulation difficulties not only in clinical populations, but in healthy controls as well (Baum et al., 2014; Cox et al., 2016).

Moreover, sleep and emotional difficulties can have a reciprocal relationship, with symptoms of each exacerbating the other and perpetuating a cycle of sleep debt (i.e., collective

amount of inadequate sleep over time; Munafo et al., 2018). Specific symptoms related to emotional disorders, such as worry and rumination, have recently been linked with sleep disturbance (Cox et al., 2016; Nota & Coles, 2015) in both healthy and clinical populations (Borders et al., 2014; Cox et al., 2016; Kertz & Woodruff-Borden, 2011). Findings from a 2015 study demonstrated the cyclical nature of sleep and worry in that sleep quality predicted subsequent worry frequency and duration, while frequency of evening-time worry also predicted subsequent sleep quality (Thielsch et al., 2015). Another type of perseverative thought is that of obsessional thinking, as demonstrated in disorders such as OCD. In fact, obsessive thought patterns have been suggested as potential contributors to sleep impairment (Cox & Olatunji, 2016). Consequently, research has recently begun investigating the function of sleep within this disorder and how impaired sleep contributes to symptom maintenance, severity (Benca et al., 1992; Schubert & Coles, 2013; Turner et al., 2007), and treatment response (Ivarsson & Skarphedinsson, 2015) in those with OCD.

Sleep and OCD

As in other psychopathologies, sleep disturbance is highly prevalent in those with OCD, with some studies demonstrating reported sleep difficulties in 65% – 70% of clinical samples (Nordahl et al., 2014, 2018). However, research studying sleep in OCD is fairly new, and the exact relation between the two is still unclear. Contributing to this complex association are the high rates of comorbidity between OCD and other disorders, all of which also seem to be associated with sleep impairment. Previously, it was thought that sleep disturbances in OCD were primarily a facet of negative affectivity associated with various comorbidities, such as mood and anxiety disorders (Cox & Olatunji, 2016). For instance, depression is highly comorbid with both sleep difficulties and OCD, and many of the sleep problems demonstrated with OC

symptoms are also seen in those with depression. In fact, research has demonstrated sleep patterns in OCD with comorbid depression as being more similar to patterns of those with major depression (Bobdey et al., 2002), while sleep patterns of OCD patients without comorbid depression more closely resemble normal sleep patterns (Bobdey et al., 2002; Diaz-Román, Perestelo-Pérez, & Buela-Casal, 2015). These findings suggest that, perhaps, secondary depression could be responsible for the sleep disturbances seen in OCD. Moreover, comorbid anxiety is also highly prevalent in those with insomnia and OCD, and some have posited that potential mediating effects of physiological hyperarousal and general anxiety may explain the association between poor sleep and OCD (Timpano et al., 2014). Despite these findings, however, there is also evidence suggesting that sleep difficulties in OCD patients remain even after controlling for depression and anxiety (Cox & Olatunji, 2016; Timpano et al., 2014). In a nationally representative sample, Cox and Olatunji (2016) found a positive association between OC symptom severity and severity of sleep impairment which was not accounted for by comorbid depression or anxiety. Furthermore, findings demonstrated that even minor sleep disturbance (i.e., not clinical insomnia) increased the likelihood of experiencing obsessive-compulsive symptoms. These findings seem to rule out the concept of sleep impairment simply being a result of negative affectivity, but rather, sleep disturbances seen in OCD are likely unique to the disorder (Cox & Olatunji, 2016).

Further evidence of the uniqueness of sleep patterns in OCD can been seen in the actual sleep architecture and circadian rhythm disturbances demonstrated by polysomnographic tests. Sleep architecture refers to the organizational structure of sleep (Harding et al., 2008), which is made up of two types of sleep: non-rapid eye-movement (NREM; i.e., 75 – 80% of total sleep time) and rapid eye-movement (REM) sleep (20 – 25% of total sleep time; Carskanon &

Dement, 2005). NREM is broken down further into three sleep stages (i.e., stages 1, 2, and 3), each denoting increasing levels of sleep depth. Stage 3 is also referred to as slow-wave sleep (SWS), which mostly takes place during the beginning of the night (Harding et al., 2008). NREM and REM sleep alternate in a cyclical fashion with each NREM-REM cycle lasting for approximately 90 – 120 minutes (Carskanon & Dement, 2005). During a typical night of sleep, a sleep period begins with a short phase of stage 1 sleep, then on to stages 2 and 3, before entering REM sleep. From here, a person will cycle between NREM stages and REM throughout the remainder of the night, with REM sleep increasing as the night progresses. Although not fully understood, research has determined that adequate cycling through each of the sleep phases is important for optimal cognitive, emotional, and physiological function (Carskanon & Dement, 2005). However, irregular sleep architecture (e.g., abnormal cycling, absence of sleep stages) has been associated with disordered sleep (Carskanon & Rechtschaffen, 2005), psychiatric disorders (Benca et al., 1992), as well as learning and memory impairment (Tucker et al., 2006; Mednick, Nakayama, & Stickgold, 2003).

Polysomnographic studies have revealed characteristic sleep cycling patterns in those with major depression, consisting of decreased SWS, faster onset of REM, longer REM duration, and higher frequency of rapid eye-movements per REM period (Palagini et al., 2013). Similar patterns of reduced SWS and abnormal REM distribution have been noted in many psychiatric disorders (Benca et al., 1992; Palagini et al., 2013), and previous studies have demonstrated such sleep architecture in patients with OCD as well (Gaillard et al., 1984; Walsleben et al., 1990). However, these studies did not account for potential comorbidities associated with OCD, particularly comorbid depression (Paterson et al., 2013). More recently, in a meta-analysis on sleep patterns related to OCD, Nota et al. (2015) noted differences between the sleep stages of

OCD patients compared to other psychopathology and healthy controls. Unlike patterns of *increased* REM and *reduced* SWS seen in those with depression and other disorders, findings demonstrated the reverse was true in the OCD group. Specifically, sleep architecture in the OCD with secondary comorbid depression group mimicked that of sleep seen in major depression; however, those with OCD without cooccurring disorders had *lower* percentages of REM sleep and *higher* SWS than healthy controls. Interestingly, the authors remarked that the pattern seen in the OCD patients was consistent with those experiencing sleep deprivation, noting that attentional and emotional regulation difficulties following disrupted sleep are also commonly seen in individuals with OCD.

Similar to having distinctive sleep architecture, a 14-study review by Paterson et al. (2013) found that those with OCD demonstrated altered sleep patterns consisting of less sleep duration, reduced sleep efficiency, difficulty falling asleep, and worse subjective sleep quality compared to healthy controls. Furthermore, more severe OC symptoms were linked to more sleep disturbances, a finding which has since been confirmed by Raines et al. (2015) and Cox and Olatunji (2016). Additionally, OCD seems to be characterized by later bedtimes than those of healthy controls. Specifically, research has demonstrated an association between later bedtimes with greater severity of OC symptoms (Nota & Coles, 2015), as well as subjective severity of insomnia symptoms (Cox & Olatunji, 2016; Raines et al., 2015).

Related to delayed sleep time, OCD appears to be associated with specific aspects of circadian rhythm and sleep-wake cycles. For instance, studies have shown a high prevalence of delayed sleep phase (DSP) in those with OCD (Nota et al., 2015, Schubert & Coles, 2013), which has also been associated with greater severity of OC symptoms (Benca et al., 1992; Schubert & Coles, 2013; Turner et al., 2007). DSP is a subtype of circadian rhythm sleep-wake

disorder characterized by a pattern of delayed sleep onset and awakening by two or more hours beyond conventional sleep-wake times (APA, 2013; Mukhopadhyay et al., 2008). In a sample of 187 individuals suffering from severe, enduring OCD, 17.6% met diagnostic criteria for DSP, even after controlling for comorbid depression (Mukhopadhyay et al., 2008). Similarly, Coles et al.'s 2012 study investigating sleep habits and OC symptoms in a sample of 266 undergrads found that those with later bedtimes had significantly more OC symptoms, scores of which were consistently higher across OCD domains, despite controlling for negative affectivity. In addition, they found individuals who went to bed later had significantly reduced sleep duration and higher levels of negative affectivity than those with typical bedtimes (i.e., habitually going to bed before 1:00 a.m.). Habitual and abnormally late, or delayed, bedtimes can result in individuals going to bed later in the circadian phase. As such, they are likely subjected to different levels of evening lighting, and in turn, a phase shift is perpetuated.

Evidence pointing to the high prevalence of DSP in OCD is perhaps due to the behavioral aspects of the OC symptoms themselves. For instance, Coles et al. (2012) highlighted that many diagnosed with OCD wait to perform compulsions and/or rituals at night to prevent embarrassment from family members or avoid potential interruptions (Coles & Sharkey, 2011; Coles et al., 2012). Likewise, some have obsessional nighttime thinking which may disrupt sleep onset and maintenance (Cox & Olatunji, 2016). Studies have reported that with such a sleep-wake shift as seen in DSP, individuals are more likely to experience negative thought patterns, such as rumination and worry, thereby propagating a cycle of continued sleep disruption and difficulty going to sleep (Turner et al., 2007). For example, overall experience of perseverative and intrusive thoughts, as seen in rumination and worry (Thomsen et al, 2003), has been linked to the inability to fall asleep (i.e., sleep onset latency). Overall, processes such as increased

attention to thoughts, poor cognitive control, and impaired response inhibition, which have been shown to maintain perseverative thoughts, may relate to trouble falling asleep (Thomsen et al., 2003).

Not only do sleep and OC symptoms seem to reciprocally impact the severity and impairment of the other, but it also appears that each plays a role regarding treatment response; however, the findings are mixed. Some studies have indicated impaired sleep is associated with reduced OCD treatment outcomes (Ivarsson & Skarphedinsson, 2015). Additionally, there is evidence suggesting sleep disturbances must be specifically targeted during the treatment of OCD in order for insomnia symptoms to improve (Kallestad et al., 2012). On the contrary, other findings suggest secondary sleep disturbances improve when the primary disorder (i.e., OCD) is effectively treated (Harvey, 2001). In a study by Nordahl and colleagues (2018) assessing the effectiveness of treatment in patients with OCD and comorbid sleep disturbance, results showed comorbid sleep difficulties did not impair treatment outcome. In fact, individuals with higher degrees of sleep disturbances at pre-treatment had better outcomes on OCD symptoms at post-treatment. Additionally, they posited that skills learned throughout effective OCD treatment appeared to generalize to insomnia symptoms as evidenced by reduction of sleep disturbances from pre- to post-test which were maintained at 6-month follow up. Regardless of the equivocal findings related to the impact sleep has on OCD treatment outcome, and vice versa, evidence continues to support a connection between the two.

Although much is still poorly understood with regard to sleep and OCD, research is beginning to highlight this important and unique relation. Each seems to considerably affect the other, and it appears a positive relationship exists between the severity of OC symptoms and sleep disturbances. Additionally, some studies suggest poor sleep may interfere with patients'

response to OCD treatment. Despite impaired sleep being common across psychiatric disorders, it also seems to have a distinct association with OCD as evidenced by higher prevalence of DSP and sleep architectural patterns more closely resembling those seen in sleep deprivation. As sleep deprivation has been linked to cognitive deficits, specifically deficits in executive functioning, which are commonly seen in individuals with OCD, such findings are notable and likely critical in understanding this complex connection.

Executive Function

Although a broad term encompassing a variety of complex processes, executive function (EF) generally refers to higher-order cognitive processes that regulate and integrate basic lower-level thoughts and actions to achieve optimal daily performance in novel situations (Cox et al., 2015; Norman et al, 2000; Roopesh et al., 2013; Snyder et al., 2015). Utilizing EFs is an effortful process, which enables the overriding of automatic, instinctual responses to navigate and function in one's environment effectively, efficiently, and appropriately. As such, proper EF use is necessary for overall quality of life (Brown & Landgraf, 2010), including physical and mental health and development, as well as optimal occupational, academic, and social functioning (Diamond, 2013). With regard to physical health, research has shown that poorer EFs are associated with obesity and overeating, substance abuse, and reduced adherence to treatment (Diamond, 2013). Related to mental health, many mental disorders, such as depression, addiction, attention-deficit/hyperactivity disorder (ADHD), schizophrenia, post-traumatic stress disorder (PTSD), and obsessive-compulsive disorder (OCD) have been linked to impaired EFs (Snyder, et al., 2015). EF difficulty has also been implicated in school readiness (Morrison & Chein, 2011) and academic success (Borella et al., 2010), job success (e.g., productivity; Bailey,

2007) adherence to public safety (e.g., crime, violence, recklessness; Denson et al., 2011), and the ability to get along with others (e.g., coworkers, friends, spouse; Eakin et al., 2004).

Various conceptualizations have been offered in efforts to understand and study EF; however, the most commonly used model of adult EF is the three-factor model by Miyake and Friedman (2012). The three-factor model, which provides an organizational framework for the regulation of complex human cognition (Miyake et al., 2000), includes the most basic and primary EFs: updating (i.e., working memory), set-shifting (i.e., cognitive flexibility, attention switching, or task shifting), and response inhibition (i.e., inhibitory control). From these three core EFs stem higher-order EFs like decision-making, planning, reasoning, and problem solving (Aidman et al., 2019; Diamond, 2013).

Updating

Updating, or sometimes referred to as working memory, is the cognitive process of monitoring and coding external information such that extraneous input might be discarded and replaced with task-relevant information (Miyake et al., 2000). Contrary to other memory processes of information storage and maintenance, updating allows for the active manipulation of pertinent information in working memory. Working memory enables one to work with information that is no longer perceptually present by holding said information in one's mind to relate it to other information over time (Diamond, 2013). This EF is essential for such things as performing mental math, understanding written and spoken language, turning instructions into action, combining new information with thoughts and plans, mentally reorganizing lists and tasks, and the ability to conceive relations between objects or concepts that are seemingly unrelated, to name a few. One's abilities to make decisions, reason, plan, and be creative would be impossible without the capacity for updating (Diamond, 2013).

Set-Shifting

Set-shifting, also referred to as attention switching, is the capacity to change focus among various aspects of a stimulus upon change in feedback (Olley et al., 2007; Toobaei et al., 2015). This EF allows for humans to actively adapt to the current situational context or present task demands (Déak & Narasimham, 2003; Miyake et al., 2000; Poljac et al., 2010). Furthermore, proper set-shifting requires the ability to inhibit cognitive interference from previous stimuli to disengage from a former task and turn attention toward a new task. This helps with transitioning efficiently when switching tasks, is involved in seeing objects from different spatial perspectives, and helps humans to mentally shift from their own point-of-view and consider situations from another's perspective (Diamond, 2013). This concept is closely related to the function of cognitive flexibility, which is the flexible use of cognitive strategies (Sternheim et al., 2014). By utilizing this EF, individuals are able to think creatively, engage in different problem-solving strategies when former strategies are no longer effective, empathize with others, and take advantage of novel and unanticipated opportunities (Diamond, 2013).

Response Inhibition

Response inhibition is the ability to suppress situationally inappropriate and unwanted automatic or dominant responses (Chamberlain et al., 2005; Miyake et al., 2000). This EF includes the controlling of thoughts, behavior, attention, and emotion in order to respond in a contextually appropriate and adaptive manner (Diamond, 2013). For example, response inhibition enables individuals to remain on-task despite distractions or inhibit the urge to quit a task prematurely, resist the urge to lash out when someone has upset him/her, and adhere to social norms (e.g., waiting one's turn, respecting others' personal boundaries, following rules or the law). Additionally, inhibition allows individuals to consider all the facts before jumping to

conclusions or further assess a situation rather than accepting the first answer/solution that comes to mind. In this way, humans are better able to interact and get along with others, make smarter and safer decisions, as well as plan and problem-solve more effectively (Diamond, 2013).

The aforementioned core EFs are the building blocks for higher-order EFs, such as reasoning, problem solving, and decision-making, which are important to overall physical, mental, and social functioning (Aidman et al., 2019; Diamond, 2013). However, when physical and emotional needs are not adequately met, EF impairment may occur and even mimic EF disorders such as ADHD (e.g., poor attentional control, impulsivity, forgetfulness; Diamond, 2013). Indeed, EF appears to be especially susceptible to adverse emotional and behavioral factors such as stress, sadness, limited physical exercise, and even sleep difficulties (Diamond, 2013), subsequently leading to further functional impairments.

EF and Sleep

Much research has been dedicated to the cognitive consequences of sleep deprivation, and the effects of inadequate sleep on cognitive processes vary. Of particular interest are the effects sleep has on EF. EFs, the most basic of which include response inhibition, attentional shifting, and working memory capacity (Aidman et al., 2019), are primarily associated with the PFC of the brain. Research suggests that as sleep debt increases, executive performance decreases. This is especially true with regard to performance of these most basic EFs, particularly during times of total sleep restriction and circadian dips (e.g., early morning and midday). EF declination can be seen even after just one 24-hour period of total sleep restriction and continues to worsen as phases of inadequate sleep persist (Aidman et al., 2019).

The impact of insufficient sleep on EF is partially explained by the effects sleep restriction has on the PFC. Though various brain regions are affected by inadequate sleep,

evidence suggests that the PFC is particularly sensitive to the effects of such (Diamond, 2013; Wilckens, Woo, Kirk, Erickson, & Wheeler, 2014). A meta-analysis by Lim and Dinges (2010) highlighted consistencies in PFC functioning of young adults to that of older adults who were experiencing normal, age-related PFC decline. For instance, one study investigated the effects of 36 hours of total sleep deprivation in young adults (i.e., $M = 23$ years old) using tests to measure PFC function. Findings revealed similar PFC functioning as seen in normal, healthy, middle-aged adults (i.e., average age of 60; Harrison & Horne, 2000). Such findings are supported by fMRI studies which have demonstrated changes in activity levels in PFC regions (i.e., lateral and medial PFC) following periods of sleep deprivation as compared to non-sleep deprived subjects, suggesting that the PFC likely compensates for effects of low alertness and fatigue (Chee & Choo, 2004; Drummond et al., 2000). Additionally, reductions of metabolic activity in the PFC have been shown after just one night of sleep loss (i.e., 24 hours of sleep deprivation; Thomas et al., 2000). Taken together, while reduced cognitive speed and accuracy performance are certainly expected after a night of poor sleep, the PFC appears to be especially vulnerable to effects of sleep deprivation (Lim & Dinges, 2010), thus, impacting EF necessary for adequate daily functioning.

With regard to the three-factor model, poor sleep (i.e., sleep deprivation, restriction, debt, normative variations) has been implicated in the impairment of all three primary EFs. A study by Olson et al. (2016), investigating the effects of sleep debt on updating performance found that greater sleep debt was associated with increased updating impairment as demonstrated through a decision-making task. Specifically, the less sleep individuals had prior to task performance, the more their decisions were influenced by more recent task-related information, as opposed to incorporating information occurring over extended periods of time; thereby, impacting the

overall effectiveness of their decisions. Similarly, attentional aspects appear to be impacted by inadequate sleep, with excessive sleepiness being linked to more errors on tasks involving set-shifting (Grant & Chamberlain, 2018). Sleep-related attentional deficits also include diminished allocation of attentional resources, increased mind wandering, and limited cognitive awareness of mind wandering (Grant & Chamberlain, 2018; Poh et al., 2016). Effects of sleep deprivation have also been demonstrated with regard to components involved in set-shifting: reaction time and accuracy, known as shift costs (Heuer et al., 2004). In particular, sleep deprivation results in greater shift costs such as increased reaction time and reduced accuracy in tasks involving set-shifting (Heuer et al., 2004). Furthermore, research has also shown increased impulsivity and impaired response inhibition associated with insufficient sleep, as well as increased risky decision-making (Grant & Chamberlain, 2018). Findings from the Grant and Chamberlain study (2018) suggest that self-reported sleepiness seems to be associated with symptoms consistent with gambling disorder, ADHD, and internet addiction, as well as maladaptive decision-making (i.e., gambling more points on a gambling task).

EF impairment seems to result not only for those experiencing sleep deprivation and total restriction, but also with regard to normal variations in sleep quality (e.g., sleep continuity, total sleep time). For instance, in a study by Wilckens et al. (2014) examining such sleep parameters across different ages, results indicated greater sleep continuity (i.e., amount and distribution of sleep versus wakefulness, including sleep onset and maintenance, during a particular sleep period) as associated with better cognitive performance overall. Particularly, sleep continuity was significantly related to improved working memory and inhibitory control in young adults. Additionally, total sleep time was associated with working memory in young adults, in that very short or very long total sleep time predicted working memory declination.

As the three core EFs serve to regulate and control more complex cognitive processes needed for daily interaction and survival, such findings have important implications across various settings (Diamond, 2013; Lewis & Carpendale., 2009). Another important aspect affected by both EF and sleep that impacts every day functioning is that of emotional processing (Levens & Gotlib, 2010). Sleep disturbance as it relates to EF may have negative subsequent effects on emotional processes. Research has demonstrated that even one night of sleep loss can impair top-down executive control from the frontal lobe, particularly, connectivity between the PFC and the amygdala. This leads to increased reactivity of the amygdala (i.e., responsible for emotion perception and controlling aggression), which can lead to deficient executive control over perseverative and repetitive thought phenomena. Additionally, a hyperactive amygdala may result in attentional bias toward negative and potentially threatening environmental stimuli (Yoo et al., 2007). This negative attentional bias toward threat, along with perseverative thought processes (e.g., rumination, worry, obsessions) are hallmarks of many mood and anxiety disorders.

EF and Psychopathology

The majority of psychiatric disorders have been associated with some form of EF deficiency (Snyder et al., 2015). In fact, studies have shown that diminished EF predicts several psychiatric transdiagnostic risk factors (Ruscio et al., 2007) such as emotion dysregulation (McRae, et al., 2012), worry (Snyder et al., 2015), and rumination (Demeyer et al., 2012). Some have suggested that EF impairment may actually be an intermediate transdiagnostic risk factor for the development of psychopathology as it has been demonstrated in numerous psychiatric disorders such as schizophrenia, major depression, bipolar disorder, PTSD, ADHD, as well as

various anxiety disorders (e.g., panic disorder, social phobia, generalized anxiety disorder; Snyder et al., 2015).

Although to differing extents, evidence of impairment in the three primary EFs of the three-factor model are displayed in each of the above-mentioned disorders. According to a review by Snyder et al. (2015), individuals with schizophrenia appear to have the greatest EF impairment comparatively, with large effect sizes related to updating, set-shifting, and response inhibition. While similar deficits can be seen in mood disorders, such as major depression, small to medium effect sizes are shown on tasks involving these three EFs. Likewise, bipolar disorder has been associated with moderate effect sizes for set-shifting and inhibition; however, research is limited with regard to updating in this disorder. The study of updating seems to be lacking with regard to PTSD and ADHD, as well. Nevertheless, PTSD has been associated with diminished inhibition and medium effects of set-shifting, while those with ADHD show small to medium effect sizes in set-shifting and response inhibition. Lastly, with respect to EF in anxiety disorders, research is fairly limited, and findings are mixed (Snyder et al., 2015), with some evidence of set-shifting difficulty in disorders such as panic disorder, generalized anxiety disorder, and social phobia. There is also evidence suggesting EF deficits contribute to threat attentional bias in those with anxiety, which serves to maintain their anxiety (Heeren et al., 2013).

EF and OCD

Another disorder associated with EF impairment is OCD, with findings from neuropsychiatric studies providing evidence of such (Toobaei et al., 2015). EF impairment is, therefore, a fundamental symptom of the disorder (Moritz et al., 2009). Common EF impairments seen in OCD include deficits with response inhibition, memory (i.e., non-verbal

memory, spatial working memory, verbal memory), attention (e.g., set-shifting), fluency, decision-making, processing speed, and planning (Toobaei et al., 2015). A study by Kashyap et al. (2013) examined EF with regard to level of insight in OCD patients, as poor insight has been associated with poorer prognosis of the disorder. In addition, some researchers have posited that poor insight may be due to deficient neurological functioning. Following a series of cognitive tests (e.g., Stroop, Wisconsin Card Sorting Test, Iowa Gambling Task, Tower of Hanoi Test) assessing various EFs (i.e., planning, set-shifting, decision-making, verbal memory, conflict resolution and response inhibition, fluency, working memory), findings indicated particular impairments associated with levels of OCD insight. More specifically, poor insight was related to larger deficits in conflict resolution and response inhibition, fluency, and verbal memory. In turn, the investigators suggested those with poorer insight may have trouble processing conflicting information, as well as remediating their memory through updating. Furthermore, poor insight in OCD may be associated with failure to access corrective feedback in order to adjust faulty thoughts and beliefs.

Further evidence of EF impairment in OCD was seen in an investigation by Roopesh et al. (2012) comparing executive performance of a sample of medication naïve, non-depressed OCD participants to a group of healthy controls. Areas assessed included planning and problem-solving, response inhibition, attention and concentration, concept formation, set-shifting, and working memory. Results indicated that OCD patients performed significantly worse on tasks requiring organizational strategies and set-shifting, but groups did not differ regarding memory recall and response inhibition. Other studies, however, have revealed deficits in non-verbal memory (Abramovitch et al., 2005), set-shifting (Panendes et al., 2005), and response inhibition (Abramovitch et al., 2013; Chamberlain et al., 2005) for individuals diagnosed with OCD.

Additionally, the heterogeneous nature of OCD likely contributes to mixed and inconsistent research findings regarding EFs specific to OCD. While a number of EF deficits have been cited as being associated with OCD (Abramovitch et al., 2013; Penandés et al., 2005), other studies have found evidence to the contrary (Bohne et al., 2008; Krishna et al., 2011), suggesting comparable performance to control groups. Still, others assert any EF impairment observed in OCD is better explained by moderating factors (Moritz et al., 2009), including medication effects (selective serotonin reuptake inhibitors (SSRIs; Kuelz et al., 2004), symptom presentation (Rasmussen et al., 2016), and high rates of comorbidities (Moritz et al., 2001).

With respect to medication, SSRIs are commonly prescribed to those with OCD (Skandali et al, 2018). As OCD is also highly comorbid with anxiety disorders, these patients may also be prescribed benzodiazepines (Moritz et al., 2001). Importantly, SSRIs and benzodiazepines have been shown to slow down one's reaction time and processing speed (Kuelz et al., 2004). A review by Kuelz et al. (2004) highlighted why this is problematic. For example, while some studies assessing attention in individuals with OCD found slower information processing than healthy controls (Schmidtke et al., 1998; Basso et al., 2001; Moritz et al., 2001, 2002), most of these studies included patients who were medicated with SSRIs or benzodiazepines (Basso et al., 2001, Moritz et al., 2001, 2002). On the contrary, studies finding comparable processing speeds between OCD participants and controls either consisted of nonmedicated participants (Cohen et al., 1996; Sieg et al., 1999), or medication was not reported (Berthier et al., 1996). Furthermore, a study investigating cognitive performance associated with the administration of an acute SSRI revealed distinct EF impacts. Specifically, participants demonstrated reductions in learning and cognitive flexibility, but enhanced response inhibition

(Skandali et al., 2018). As such, it is important to consider how medication effects may impact EF in these individuals, and subsequently, interpretations of research findings.

Another aspect likely influencing inconsistent findings of EF impairment in OCD is the fact that the disorder contains various symptom dimensions (i.e., contamination/checking, symmetry/ordering/arranging/counting, sexual/religious, aggression, somatic, hoarding/collecting, miscellaneous; Rosario-Campos et al., 2006). Research highlights that certain OC symptom dimensions may be characterized by distinct neurological functions and mechanisms (Mataix-Cols et al., 2005), evidence of which has been demonstrated in a number of different EFs. In 2006, Lawrence and colleagues conducted a study investigating decision-making and set-shifting associated with different OCD dimensions. Findings revealed that, in general, the OCD group did not differ from controls with regard to decision-making; however, they did demonstrate worse performance on set-shifting tasks. Interestingly, distinct symptom-domain deficits emerged, with the presence and severity of hoarding symptoms being associated with reduced decision-making ability and symmetry/order symptoms being linked to deficient set-shifting. (Lawrence et al., 2006).

Similarly, Leopold and Backenstrass (2015) conducted a meta-analysis examining the neuropsychological functioning of OCD patients presenting as “washers” (i.e., symptoms related to contamination/washing) and “checkers” (i.e., symptoms associated with harm avoidance and doubt), finding that those with checking symptoms (e.g., repeated ensuring that doors are locked, stoves are turned off, some harmful act was not committed, etc.) showed greater impairment in the majority of cognitive domains tested as measured by various neurocognitive tasks (e.g., Stroop Test, Go/No-Go, Trail Making Test-B, spatial N-Back task). Specifically, large effects were found in the domains of planning/problem-solving and response inhibition, moderate

effects in set-shifting, and small effects in sustained attention, encoding, verbal and nonverbal memory, and processing speed. In addition, Bragdon et al., (2018) conducted a meta-analysis comparing cognitive functioning in those with symptoms in symmetry/ordering (i.e., symptoms of incompleteness) with those endorsing obsessing/checking (i.e., symptoms of harm avoidance) dimensions. Results indicated large effects sizes associated with poorer attention and working memory in the symmetry/ordering group as compared to the obsessing/checking participants.

Inconsistent findings have also been shown related to response inhibition. Many studies have demonstrated impaired response inhibition for individuals with OCD (Chamberlain et al., 2005; de Wit et al., 2012; Penadés et al., 2007). However, there are others that show no differences in response inhibition between OCD and control groups (Bohne et al., 2008; Krishna et al., 2011), suggesting reduced inhibition may be associated with some OCD dimensions, but not all (Ramussen et al., 2016). Taken together, these studies suggest neurological differences associated with OCD presentation type which may be differentially associated with particular EF impairments.

As previously mentioned, EF impairment is associated with a number of psychiatric disorders (e.g., anxiety disorders, mood disorders, impulse-control disorders, substance-use disorders; DSM-5; Ruscio et al., 2007, 2010). Due to high rates of comorbidity of other disorders in those with OCD, it is likely that failing to properly control for the effects of such could have led to misinterpretations of EF impairment in OCD patients (Moritz et al., 2001). As evidence has demonstrated that depression can influence performance on non-verbal memory assessments (Krishna et al., 2011) and set-shifting abilities (Moritz et al., 2001), much speculation has been devoted to whether impairments seen in OCD are better attributed to commonly occurring comorbid depressive symptoms. Again, findings seem to be conflicting, with some studies

demonstrating that controlling for depression yields non-significant differences in EF impairment between OCD and control groups (Basso et al., 2001; Moritz et al., 2001, 2003). In contrast, more recent studies have demonstrated EF deficits in OCD irrespective of depressive symptoms, as OCD patients with low levels of depressive symptoms display similar EF impairment as those without depressive symptoms (Snyder et al., 2015).

Though findings with respect to EF deficits in OCD are equivocal due to impacts of commonly prescribed medications, heterogeneity of symptoms, and moderating effects of comorbidities, evidence supporting an association between the two remains. The aforementioned information outlines a variety of specific EF deficits in OCD, the development of which can all be reduced to the three core EFs: updating, set-shifting, and response inhibition (Diamond, 2013; Miyake et al., 2000). Each of these primary EFs have been proposed as potential endophenotypes of OCD (Cavedini et al., 2010; Chamberlain et al., 2007; de Wit et al., 2012), and research shows that individuals with OCD demonstrate small but significant effect sizes related to response inhibition and set-shifting, with large effect sizes in regard to updating (Snyder et al., 2015). Therefore, for the sake of parsimony, specific EF impairment as seen in OCD symptomatology will be explained using the three-factor model (Miyake et al., 2000) as an organizational framework.

Updating in OCD

A great deal of evidence is suggestive of memory impairment in those with OCD. It has been asserted that repetitive checking behavior, as seen in checking compulsions, is a product of the inability to update one's memory with corrective information in order to modify faulty beliefs and obsessions (Chamberlain et al., 2005; Kashyap et al., 2013). Those with checking symptoms often report doubt related to the accuracy of their recall ability and whether or not

they performed a particular action already (e.g., unplugging appliances, locking doors; Rachman & Shafran, 1998; Woods et al., 2002). A meta-analysis by Woods et al. (2002) reviewed the literature on memory impairment in OCD, including studies consisting of those with an OCD diagnosis endorsing high checking compulsions. Results indicated that "checkers" had worse performance on working memory tasks (e.g., Digit Span from the Wechsler Adult Intelligence Scale) than "non-checkers." Additionally, Kashyap et al. (2013) speculated that failure to use corrective information (i.e., evidence that contradicts beliefs) to update memories may be associated with OCD patients who have poor insight into their disorder. Stated otherwise, updating impairment may prevent the revision of deeply held irrational beliefs, which ultimately maintains poor insight. This is important considering that poor insight has been connected with increased symptom severity, a greater number of symptoms, and a longer and more chronic course of illness (Kashyap et al., 2012). However, as with most of the literature investigating EF in OCD, findings are equivocal at present. For instance, some researchers suggest that working memory deficits may be secondary to OCD symptoms (Abramovitch et al., 2013; Woods et al., 2002) such as when obsessional thoughts pose distractions while performing memory-related tasks (Woods et al., 2002).

From a neurological perspective, further support for updating deficits in OCD is demonstrated through shared neurological regions implicated in each. For example, updating is often associated with the dorsolateral prefrontal cortex (DLPFC) of the brain (Chamberlain et al., 2005, Miyake et al., 2000) and reduced activity in this area is typically related to working memory deficits. Neuroimaging studies have suggested that in the brains of those with OCD, this region often appears smaller in size, with one study reporting a negative association between symptom severity and DLPFC volume (Vahabzadeh & McDougle, 2014). Furthermore, the

DLPFC appears to be hyperactive in individuals with OCD (de Vries et al., 2013; Vahabzadeh & McDougle, 2014). Brain imaging results from de Vries et al. (2013) demonstrated that those with OCD, compared with non-OCD patients, exhibited compensatory hyperactivity in brain regions associated with working memory on visuospatial N-back tasks. The experiment involved four conditions: a control group and three groups varied by increasing levels of working memory load. As expected, participants in the control group exhibited increased activation in working memory neural networks dependent on level (high versus low) of task load (i.e., as demonstrated by length of time delay since stimuli presentation). However, those in the OCD group displayed hyperactivation at low level task loads and performance impairment at high loads. That is, for OCD patients, it appears as though hyperactivity in working memory neural networks function to compensate for working memory deficits at lower task demands, but performance declines as task load increases. The authors speculated this compensatory hyperactivation occurs as a result of an inability to inhibit extraneous stimuli leading to impairment in working memory, an effect which was also observed in OCD patients' unaffected first-degree relatives (de Vries et al., 2013).

Taken together, updating appears to be involved in OCD symptom presentation and may even differentially impact certain symptom domains (i.e., checking vs. nonchecking; Woods et al., 2002). Moreover, one's memory updating ability seems to be involved in individuals' levels of insight regarding their obsessions and compulsions, which may indirectly contribute to overall outcome expectancies and treatment response (Kashyap et al., 2012). Also, the DLPFC, the brain region associated with updating, has been highly implicated in pathophysiology of OCD. Specifically, this area seems to be smaller in OCD patients' brains and becomes hyperactive during tasks requiring even low working memory capacity (de Vries et al., 2013). Taken

together, while the understanding of how updating is connected to OCD is still imprecise, the evidence maintains support for such a connection.

Set-Shifting in OCD

In OCD, set-shifting deficits have been associated with perseveration (Miyake et al., 2000), a mechanism commonly underlying obsessional thinking and compulsive behaviors (Chamberlain et al., 2005). For instance, those with OCD aim to alleviate intrusive thoughts through the rigid use of strategies, whether mentally or behaviorally (compulsions), despite repeated feedback of strategy ineffectiveness over time (Sternheim et al., 2014). While findings investigating set-shifting abilities in OCD are inconsistent (Abramovitch et al., 2013), several studies have found that individuals with OCD tend to do poorer on neuropsychological tests measuring set-shifting (e.g. WCST; Kashyap et al., 2013), with greater perseverative errors than controls (Lawrence et al., 2006). Similarly, in a sample of 288 undergraduate students, results from Sternheim et al. (2014) demonstrated a negative relationship between severity of OC symptoms and cognitive flexibility, with higher OC symptoms associated with more total and perseverative errors on a neurocognitive measure of set-shifting (i.e., Berg Card Sorting Task). Additionally, it is important to consider the nature of the tasks used in studying this construct specific to OCD. For instance, Morein-Zamir et al. (2010) investigated the effects of OCD-relevant stimuli (as opposed to neutral stimuli) on task shifting (i.e., set-shifting) and response inhibition performance using a stop-signal task. Participants were instructed to press a particular key on a keyboard in the presence of target stimuli and would occasionally be provided an external signal to stop (i.e., inhibit the preponent response to press the key). Not only were they instructed to stop in the presence of the tone, they were also directed to press a different key with their non-dominant hand instead (i.e., task switch, set-shift). Although inhibition was impaired

regardless of stimuli meaning, findings demonstrated that using emotionally valanced, OCD-relevant stimuli (versus neutral) negatively impacted the OCD group's ability to shift their attention in order to comply with new task instructions.

Regarding the brain regions associated with set-shifting, the frontal lobes and the ACC have been implicated. Specifically, impaired frontal lobe functioning has been demonstrated in perseverative and repetitive behavior, while the ACC is involved in the regulation of emotions and social behavior, as seen in decision-making (Vahabzadeh & McDougle, 2014). There is also evidence that set-shifting difficulty may involve the DLPFC, as it plays a role in the allocation of attentional resources and attention shifting. As previously mentioned, hyperactivity in the DLPFC has been demonstrated in those with OCD, which may be responsible for the overallocation of attentional resources to obsessional thoughts and failure to disengage from obsession-oriented tasks (Chamberlain et al., 2005; de Vries et al., 2013; Vahabzadeh & McDougle, 2014). In a review by Szczepanski and Knight (2014) on human behavior as a result of lesions to the PFC, results indicated that lesions to the DLPFC caused perseverative errors (Barceló & Knight, 2002) and deficient flexibility of rule application. That is, in studies using the WCST to measure set-shifting, patients with PFC lesions were unable to sort cards according to new rules and continued to sort cards based on original instructions, similar to deficits seen in OCD (Shallice & Burgess, 1991).

The ability to shift one's attention appears to be related to the perseverative thoughts and behaviors characteristic of those with OCD (Lawrence et al., 2006; Sternheim et al., 2014). The limited capacity for cognitive flexibility likely maintains OCD symptoms and interferes with one's ability to disengage from maladaptive thoughts and strategies. This is particularly true for emotionally valanced stimuli (Morein-Zamir et al., 2010), which may be attributable to

dysfunction of the frontal lobes, ACC, and the DLPFC regions of the OCD brain (Chamberlain et al., 2005; de Vries et al., 2013; Vahabzadeh & McDougle, 2014).

Response Inhibition in OCD

Though findings are mixed, with some research failing to find group differences in response inhibition related to OC symptoms (Alilo et al., 2011; Moritz et al., 2002; Toobaei et al., 2015), others maintain that impaired capacity to inhibit responses underpins the inability to resist compulsive behaviors demonstrated in OCD (Chamberlain et al., 2005; Kalanthroff et al., 2016). According to a study investigating motor inhibition (assessed by a stop-signal task) in depressed and non-depressed OCD patients using neutral and OCD-relevant stimuli, results indicated inhibitory impairments in both groups compared to controls. Notably, OCD groups did not significantly differ from one another, suggesting response inhibition deficits were not attributable to depressive symptoms. Moreover, this study demonstrated inhibition impairments regardless of stimulus type (i.e., neutral or OCD-relevant; Morein-Zamir et al., 2010). Significant correlations between inhibition performance and particular subscales of a psychometrically sound OCD instrument, the Padua Inventory (Burns et al., 1996), were also found. Specifically, higher endorsements on the contamination and harm to others subdomains accounted for this relation suggesting that certain subtypes of OCD may be particularly vulnerable to response inhibition deficits (Morein-Zamir et al., 2010).

Neurologically, evidence for impaired response inhibition in OCD lies in the neural circuitry involved with the OFC and caudate nucleus (Fineberg et al., 2010). Specifically, the OFC is responsible for inhibitory control and the caudate nucleus plays a role in compulsive behavior. In a review by Fineberg et al. (2010), the literature points to dysfunction in the neural networks involving each of these structures, in that those with OCD experience *hypo*activation of

the OFC and *hyper*activation of the caudate nucleus. The OFC is also highly implicated in decision-making, particularly with regard to evaluation of a reward. Therefore, lesions to this area of the brain result in difficulty with inhibiting dominant responses, especially those related to behaviors that were previously rewarded but no longer are (Szczepanski & Knight, 2014). Such inhibition deficits, in turn, interfere with one's ability for reversal learning, thereby, maintaining ritualistic impulses, as seen in compulsive behaviors. Studies have also suggested reduced activity of the ACC in individuals with OCD, which is suspected to be responsible for diminished ability to suppress inappropriate repetitive and ritualistic behavior in response to obsessions (Chamberlain et al., 2005; Kalanthroff et al., 2016). Furthermore, the DLPFC, which sends projections to both the OFC and the caudate nucleus is involved in response inhibition, as well. A review of the literature (Szczepanski & Knight, 2014) showed that lesions to the DLPFC results in difficulty inhibiting prepotent responses as demonstrated on tasks assessing planning and problem solving (i.e., Tower of Hanoi, Tower of London; Goel & Grafman, 1995), each of which require inhibition of dominant responses. As abnormalities in this area for individuals with OCD have been cited (Vahabzadeh & McDougle, 2014), the DLPFC likely plays a part in response inhibition deficits for these individuals.

Reduced response inhibition in OCD has been speculated as underlying compulsive and ritualistic behaviors common to the disorder (Chamberlain et al., 2005; Kalanthrof et al., 2016). In OCD, dysfunctional inhibition does not seem to be the result of co-occurring depressive symptoms and may differentially impact OCD subdomains (Morein-Zamir et al., 2010). Additionally, reduced response inhibition appears to occur across contexts (i.e., OCD-relevant versus neutral stimuli), which supports suggestions of impaired response inhibition as an endophenotype of the disorder (Morein-Zamir et al., 2010). This is further evidenced in a study

demonstrating such deficiencies in not only those with OCD, but also in non-OCD first degree relatives (Chamberlain et al., 2007). Support for genetic markers can also be found in neuroimaging studies implicating abnormal functioning of specific brain structures associated with response inhibition (i.e., OFC, ACC, DLPFC) in individuals with OCD.

In sum, much research has been devoted to investigating EF in OCD as impaired EF has been cited as fundamental to the disorder (Toobaei et al., 2015). As an integral part of daily functioning in general, reduced EF has been associated with poor patient insight into OCD, as well as poorer expected outcomes. For instance, deficient EF may impact, or be impacted by, symptom severity, duration of the disorder, as well as continued maintenance of symptoms (Kashyap et al., 2013). Unfortunately, EF encompasses a multitude of cognitive processes, making it a complex topic for study, a complexity of which is further compounded when studying such a heterogeneous disorder like OCD. Therefore, findings are mixed, with some studies finding evidence of significant associations between particular EFs and OCD (Abramovitch et al., 2013; Penadés et al., 2005), and others finding comparable functioning of those same EFs to that of healthy controls (Bohne et al., 2008; Krishna et al., 2011). However, such inconsistencies are likely the consequences of improper controlling of OCD covariates such as participant medication (Kuelz et al., 2004), symptom presentation (Rasmussen et al., 2016), and comorbid psychiatric disorders (Moritz et al., 2001).

Despite limited understanding of precisely how these two constructs are related, there remains a multitude of evidentiary support for the existence of such a connection. In attempt to parse this connection apart, researchers have investigated a number of specific EFs in the disorder, all of which are rooted in the three primary EFs as outlined in the three-factor model: updating, set-shifting, and response inhibition (Diamond, 2013; Miyake et al., 2000). Suggested

as endophenotypes of OCD (Cavendini et al., 2010; Chamberlain et al., 2007; de Wit et al., 2012), impairment of each of these three core EFs has been evidenced in relation to specific OC symptoms (Chamberlain et al., 2005; Kashyap et al., 2013; Lawrence et al., 2006; Sternheim et al., 2014), as differentially associated to certain symptom domains (Lawrence et al., 2006; Morein-Zamir et al., 2010), and as demonstrated by abnormal functioning of neural structures involved in the pathophysiology of OCD (de Vries et al., 2013; Fineberg et al., 2010; Szczepanski & Knight, 2014; Vahabzadeh & McDougle, 2014). Consequently, continued research to clarify the association between impaired EF and OCD is vital to understanding the clinical picture and treatment of OCD.

OCD, Sleep, and Executive Function

Overall Review of Relations

OCD is a relatively common psychiatric disorder which has been observed worldwide and spans across cultures and socioeconomic statuses (Goodman et al., 2000; Sadock et al., 2014; Toobaei et al., 2015). As one of the leading causes of disability and economic costs in the world (WHO, 2017), OCD impacts not only the individuals who experience it, but also their families, friends, coworkers, and communities at large (DuPont et al., 1995). Indeed, OCD is a quite debilitating disorder that affects one's overall quality of life, including domestic, social, and academic/occupational functioning. While OCD impacts individuals to varying degrees, it has been reported that approximately 51% of adults with OCD fall into the category of *severe* impairment [National Institute of Mental Health (NIMH), 2017].

Unfortunately, OCD is subject to a great deal of heterogeneity, as well as high rates of comorbidities (e.g., depression, anxiety); therefore, its clinical, neurological, and etiological pictures are not entirely clear (APA, 2013; DSM-5, 2013; Maia et al., 2008; Ruscio et al., 2010).

Although there is a lack of clarity regarding the pathophysiology of OCD, there is general consensus of anatomical abnormalities within CSTC loops specific to the disorder. Primary areas of focus within these loops include the OFC of the PFC, the ACC, and the caudate nucleus. From a biopsychosocial standpoint, research has demonstrated various biological, psychological, and environmental events that are common for those with OCD, the interplay of which seems to occur to differing degrees. For instance, OCD is considered moderately heritable (Abramowitz et al., 2009), has empirical support for specific types of dysfunctional beliefs (e.g., perfectionism, overestimation of threat; Taylor & Jang, 2011), and the onset of symptoms is often associated with the occurrence of life stressors (e.g., interpersonal conflict, negative familial reactions to symptoms, trauma; APA, 2013; March & Mulle, 1998; van Grootheest et al., 2007).

A factor that can have biological, psychological, and environmental impacts is the quantity and quality of sleep an individual obtains. Insufficient sleep can lead to a number of physical (e.g., high blood pressure, obesity, immune system compromise, etc.) and emotional consequences (e.g., irritability, low mood, impulsivity, etc.; Munafo et al., 2018; Nagy et al., 2020; Nienstedt & Alic, 2013; Tsuno et al., 2005), and sleep disturbance has been linked to the majority of mood and anxiety disorders (Gillin, 2007, Perkins et al., 2000; Tsuno et al., 2005). Recently, research has begun highlighting its influence on OCD, as well. Although sleep disturbance is highly prevalent in those with OCD, the exact nature of this relation remains unclear, with findings suggesting the influences of each on the other are likely reciprocal. In addition, though some researchers suggest that sleep difficulties are attributable to co-occurring disorders in those with OCD (Bobdey et al., 2002, Cox & Olatunji, 2016; Munafo et al., 2018), others assert a relationship that is unique to OCD itself (Cox & Olatunji 2016; Nota et al., 2015; Paterson et al., 2013; Schubert & Coles, 2013).

The unique influence of sleep in OCD is demonstrated in studies indicating a positive association between sleep impairment and OC symptom severity (Cox & Olatunji, 2016), as well as sleep patterns and architecture that is distinct from those seen in other psychiatric disorders (Nota et al., 2015; Paterson et al., 2013). For instance, evidence suggests that obsessional thinking or compulsive rituals may prolong sleep onset, thereby impacting one's circadian rhythms (e.g., sleep-wake disorders developing as a result of delayed sleep onset; APA, 2013; Mukhopadhyay et al., 2008). Such findings support the increased prevalence of DSP in OCD, the presence of which has also been associated with greater OC symptom severity (Mukhopadhyay et al., 2008; Nota et al. 2015; Schubert & Coles, 2013). With regard to sleep architecture, some studies have emphasized the difference in the sleep architecture of those with OCD compared to those with comorbid depression and healthy controls. That is, SWS and REM sleep patterns present in an opposite fashion as seen in depression and other mental disorders and are more closely aligned with those seen in sleep deprivation. As such, researchers are beginning to posit that sleep disturbance is not a mere result of comorbid depression or other negative affectivity in OCD sufferers (Nota et al., 2015).

Neurologically speaking, sleep is a necessary restorative process that functions to remove toxins from the brain and maintain proper neural functioning (Cox et al., 2016; Nagy et al., 2020; Nienstedt & Alic, 2013). In particular, the PFC of the brain appears to be especially vulnerable to sleep deprivation (Diamond, 2014; Lim & Dinges, 2010; Wilckens et al., 2014). This PFC-specific impact has considerable implications for EFs, which are housed in the PFC and are responsible for the modulation of complex thoughts and behaviors (Cox et al., 2016; Norman et al., 2000; Roopesh et al., 2013; Snyder et al., 2015). As such, studies have found an inverse

relation between sleep and EF, in that as sleep deprivation increases, EF performance declines (Aidman et al., 2019).

Executive functioning is quite complex and encompasses a great deal of cognitive processes. According to the three-factor model, all higher-order EFs develop from the capacity for three basic EFs: updating (i.e., replacing of irrelevant information with new task-relevant information in working memory), set-shifting (i.e., changing attentional focus based on relevant feedback), and response inhibition (i.e., suppressing situationally inappropriate, dominant responses; Diamond, 2013; Miyake et al., 2000). Notably, research has demonstrated an association of sleep deprivation and reduced functioning in all three core EFs (Grant & Chamberlain, 2018; Heuer et al., 2004; Olson et al., 2016;).

Furthermore, as one of the responsibilities of EF is to regulate emotional processes, it makes sense that sleep indirectly influences emotional experiences (Levens & Gotlib, 2010; McRae et al., 2012; Snyder et al., 2015). Moreover, impaired EF has been shown to be involved in the majority of psychiatric disorders (e.g., depression, anxiety, schizophrenia, ADHD) and has even been cited as predicting transdiagnostic risk factors (e.g., emotion dysregulation, rumination, worry; Ruscio et al., 2007). Diminished EF has also been suggested as an intermediate risk factor for the development of psychopathology (Snyder et al., 2015). In relation to sleep, insufficient sleep can result in the reduced capacity to appropriately allocate attentional resources needed for a task at hand, update one's memory and filter out irrelevant information, and inhibit contextually unwanted behavior (Vahabzadeh & McDougle, 2014), all of which are EF deficits demonstrated in those with OCD (Abraovitch et al., 2013; Chamberlain et al., 2005; Penandés et al., 2005).

According to Snyder et al. (2015), findings have demonstrated large effect sizes related to updating, as well as small but significant effect sizes in set-shifting and response inhibition in individuals with OCD. Moreover, each of the three primary EFs have evidence to support their potential as endophenotypes of OCD, and each has been connected to neural regions implicated in the pathophysiology of the disorder (Chamberlain et al., 2005; Fineberg et al., 2010; Kalanthroff et al., 2016; Miyake et al., 2000; Szczepanski & Knight, 2015; Vahabzadeh & McDougle, 2014). For example, the DLPFC seems to be smaller and hyperactive in those with OCD, an area which plays a part in updating, set-shifting, and response inhibition (Vahabzadeh & McDougle, 2014). Hypoactivation of the OFC (i.e., resulting in impulsivity) and hyperactivation of the caudate nucleus (i.e., resulting in compulsivity) are involved in both set-shifting and response inhibition (Fineberg et al., 2010). In addition, the ACC, which regulates emotions and social behaviors is also involved in all three core EFs (Chamberlain et al., 2005; Kalanthroff et al., 2016). With regard to specific OC symptoms, researchers suspect that dysfunctional updating is involved in symptoms such as repetitive checking and poor insight due to the inability to modify irrational beliefs and obsessions (Chamberlain et al., 2005; Kashyap et al., 2013). Diminished set-shifting is thought to contribute to perseverative thoughts and behavioral rigidity despite feedback over time that such behaviors are ineffective (Chamberlain et al., 2005; Lawrence et al., 2006; Sternheim et al., 2014). Additionally, response inhibition is seen in the inability to resist engaging in compulsive behaviors (Chamberlain et al., 2005; Kalanthrof et al., 2016).

Although the overall picture remains uncertain, much research points to the connection between EF and OCD. Specifically, EF has been indirectly associated with the overall course of the disorder, including symptom severity, number of symptoms, chronicity, and treatment

response (Kashyap et al., 2013). In addition, research is beginning to highlight the significance and uniqueness of sleep in OCD, as it has been linked to similar difficulties, such as increased symptom severity and reduced treatment response. Therefore, given that insufficient sleep is particularly impactful on the area of the brain responsible for EF and also appears involved in the course of OCD, it reasonably follows that these three constructs are likely connected. Of particular interest is the effect both impaired EF and poor sleep have on the treatment of OCD.

Clinical Treatment Implications

The gold-standard treatment for OCD is an aspect of Cognitive Behavior Therapy (CBT) referred to as Exposure and Response Prevention (ERP). ERP is designed to incrementally and systematically expose individuals to their obsessional-triggered fear while simultaneously preventing them from engaging in the compulsion aimed at reducing emotional discomfort. In doing so, it is suggested that these individuals have the opportunity to learn that their discomfort will likely decrease on its own without the use of the compulsion(s), thereby severing the association between the compulsion and reduction of discomfort. While this treatment has been proven effective, it has an approximate 60 – 80% success rate for those who are motivated and compliant with intensive therapy (Foa & Steketee, 1979; Goodman et al., 2000; Riggs & Foa, 1993). Unfortunately, overall, only 40 – 50% of patients improve with ERP as treatment refusal and dropout is fairly common (Abramowitz et al., 2018; Goodman et al., 2000). Though improvement is seen in about half of patients, this does not mean they are fully relieved of their symptoms, as many continue to experience residual symptoms and even relapse (Abramowitz et al., 2018; Goodman et al., 2000). This leaves a great deal of room for treatment improvement and the hope of answering the question of why so many either fail to complete treatment or continue to have residual difficulties.

Research has suggested that EF may be a key component to the success of and response to CBT (Hybel et al., 2017), and there is some evidence supporting proper EF as a predictor of CBT outcome (D'Alcante et al., 2012; Hybel et al., 2017). This line of therapy requires the completion of homework assignments (i.e., practicing therapy skills on their own between sessions), cognitive restructuring (i.e., engaging in flexible thinking and generating alternative thoughts for given situations), monitoring emotional reactions, and tracking progress (Kircanski et al., 2011; Piacentini et al, 2007). Such tasks require proper EF function (Mohlman & Gorman, 2005) in order to effectively plan, attend to and incorporate feedback contrary to their obsessional beliefs, hold and manipulate relevant information in working memory, and inhibit automatic and dominant responses (Hybel et al., 2017). EF also impacts one's ability to preserve information in memory, as well as learn and relearn skills for proper treatment benefit (Geller et al., 2018; Yazdi-Ravandi et al., 2018).

Perhaps further contributing to the reason behind limited response to OCD treatment is that of the role and function of sleep involved both in the disorder as well as in EF functioning. Impaired sleep has been linked with reduced OCD treatment response, and negative associations between sleep deprivation and EF performance have been demonstrated. Therefore, investigating potential relations amongst these three constructs may serve to illuminate limitations in the clinical treatment of OCD and contribute to overall treatment enhancement.

Current Study

OCD is a heterogeneous disorder with a complex presentation, involving high rates of comorbidities and diversity across symptom domains. Contributing to such complexity is the involvement of EF deficits, which have been seen both as symptoms of the disorder, as well as a

complication for effective treatment. Furthermore, sleep has been shown to be impacted in OCD, interfere with effective OCD treatment, and associated with EF deficits.

Given the considerable impact sleep can have on proper EF, OCD symptom severity, and treatment outcome, as well as the vital role EF plays in OCD symptomatology and treatment response, the investigation of relations among these constructs is important. In addition, although literature has linked each of these constructs to the others, no study reviewed has yet to assess these three constructs together. The current study examined the relations among OC symptom severity, EF performance, and sleep impairment. The following hypotheses were tested:

Hypotheses

1. All variables of interest (EF performance, OC symptom severity, sleep impairment) would be significantly related.
    - H1a: EF performance would be negatively associated with OC symptom severity.
        a) Participants with poorer updating abilities would have higher levels of OC symptoms.
        b) Participants with poorer set-shifting abilities would have higher levels of OC symptoms.
        c) Participants with poorer response inhibition would have higher levels of OC symptoms.
    - H1b: Sleep impairment would be negatively correlated with EF performance.
        a) Those with greater sleep impairment would perform more poorly on a measure of updating ability.
        b) Those with greater sleep impairment would perform more poorly on a measure of set-shifting ability.

c) Those with greater sleep impairment would perform more poorly on a measure of response inhibition.

- H1c: Sleep impairment would be positively related to OC symptom severity.
    a) Individuals with greater sleep impairment would have greater OC symptom severity.

2. EF performance would predict OC symptom severity, controlling for impaired sleep and depressive symptoms.
    - H2a: Poorer updating abilities would predict participant level of OC symptom severity, above and beyond sleep impairment and presence of depressive symptoms.
    - H2b: Poorer set-shifting abilities would predict participant level of OC symptom severity, above and beyond sleep impairment and presence of depressive symptoms.
    - H2c: Poorer response inhibition abilities would predict participant level of OC symptom severity, above and beyond sleep impairment and presence of depressive symptoms.
3. Sleep impairment would moderate the effect of EF performance on OC symptoms.
    - H3a: Greater sleep impairment would moderate the relationship between poor updating abilities and OC symptom severity.
    - H3b: Greater sleep impairment would moderate the relationship between poor set-shifting abilities and OC symptom severity.
    - H3c: Greater sleep impairment would moderate the relationship between poor response inhibition abilities and OC symptom severity.

CHAPTER 2

METHOD

Participants

The current study included a nonclinical sample of undergraduate students and individuals from the community. Of the 141 individuals enrolled in the study, complete data (i.e., all self-report measures and EF tasks) were collected from 91 participants (M_{age} = 25.87, *SD* = 12.50). The majority of the sample identified as female (68.1%), White (86.8%) and non-Latinx (97.8%), single and never married (70.3%), and unemployed (44.0%). Complete demographic information can be found in Table 1. Study eligibility required participants: 1) be over the age of 18, 2) have access to the internet, and 3) have a device with video capabilities (e.g., laptop, webcam, cellphone). Additionally, participants were instructed to be located in a quiet environment with minimal traffic and interruptions during the time of the study. Following completion of the study, those recruited through Sona Systems as part of university course requirement were awarded research credit. A priori power analyses using G*Power (Faul et al., 2009) revealed a sample size of 89 would be needed to be fully powered to test all hypotheses and achieve a medium effect (*f* = .15).

Measures

Self-Report Measures

Demographics Questionnaire. The Demographics Questionnaire is a self-report form of basic demographic information. Information gathered included participant age, gender, race, ethnicity, religious affiliation, education level, employment status, marital status, household

income, and current use of over-the-counter or prescription medications at the time of the study. The demographics questionnaire was taken from a template of National Institutes of Health (NIH) demographics.

Pittsburgh Sleep Quality Index (PSQI; Buysse, 1989). The PSQI is an 18-item self-report questionnaire measuring overall quality of sleep and presence of sleep disturbances within the past month. Respondents rate how often they have experienced disturbances within seven areas of sleep (i.e., subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction) using a 4-point Likert-type scale (0:"Not during the past month" – 3: "Three or more times a week"). Adding scores on each of the seven components yields one global score, ranging from 0 – 21. Participant global scores were used in the current study analyses as a measure of overall sleep impairment. An overall score of 5 or more is indicative of a "poor" sleeper. The PSQI demonstrates sensitivity in differentiating "good" (< 5) from "poor" sleep, good internal consistency, and a reliability coefficient of $\alpha = 0.83$ (Smyth, 2008).

Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010). The DOCS is a self-report measure consisting of 20 items assessing symptoms within each of the four main dimensions of OCD: contamination/washing, harm obsessions/checking compulsions, symmetry/ordering, and unacceptable thoughts. Each symptom dimension includes five items measuring severity within the past month using the following parameters: 1) time occupied by obsessions/compulsions, 2) avoidance behavior, 3) distress associated with symptoms, 4) functional impairment, and 5) difficulty letting go of obsessions and resisting compulsions. Items are rated on a scale of 0 ("no symptom) to 4 ("extreme symptoms"). A total score is determined by summing the score of all items together, higher scores of which represent greater severity of

symptoms. Total scores were used as a measure of OC symptom severity. The initial validation study revealed a cutoff score of 18 as having a 78% diagnostic sensitivity and 78% specificity (Abramowitz et al., 2010). As such, the use of the DOCS with both clinical and nonclinical populations has been empirically supported. Regarding internal consistency, Cronbach's alphas for each subscale, as well as the total score, ranged from good to excellent (αs = 0.90 – 0.96). Additionally, test-retest coefficients (rs = 0.55 – 0.66) for the total and dimensional scores revealed acceptable reliability.

Depression, Anxiety, Stress Scale-21 (DASS-21; Lovibond & Lovibond, 1995). The DASS-21 is a 21-item self-report questionnaire used to assess and distinguish among symptoms on three scales: depression (i.e., symptoms related to dysphoric mood), anxiety (i.e., symptoms of physical arousal, panic, and fear), and stress (i.e., symptoms of tension, irritability, and tendency to overreact to stressful events). Responses to items are selected on a 4-point Likert-type scale indicating degree to which the items applied to the respondent within the past week. The scale ranges from 0 – 3, with 0 corresponding with "did not apply to me at all" and 3 being "applied to me very much or most of the time." The sum total of the depression scale was used in the present study to determine presence of depressive symptoms. Possible scores on the depression scale range from 0 – 42, with higher scores indicating more severe depressive symptoms. The DASS-21 has good internal consistency (αs = 0.87 – 0.94), as well as excellent concurrent validity when correlated with other measures of depression and anxiety (e.g., BAI, BDI, STAI-T; Antony, Bieling, Cox, Enns, & Swinson, 1998).

Executive Function Tasks

The current study measured EF through a series of computerized tasks using the Psychology Experiment Building Language (PEBL; Mueller & Piper, 2014), a free cross-platform software system used for running computer-based experiments and tests.

Dual N-Back Task (Jaeggi et al., 2008). The Dual N-Back task is a variation of the N-Back (Kirchner, 1958) cognitive test designed to measure a number of aspects related to working memory, including updating. The PEBL version of the Dual N-Back was used for this study. For this task, participants track concurrently presented visual and auditory stimuli and decide whether each current stimulus matches one presented a designated number (i.e., *n*) of items back. Each stimulus is presented for a span of three seconds, with auditory stimuli presented through computer speakers and visual stimuli displayed on a computer monitor. Auditory stimuli consist of a series of single consonant letters (A, C, G, K, Q, R, T, V), while visual stimuli consist of a blue square appearing in one of eight positions at random around a fixation cross. In the initial block, *n* is assigned the value of 1, meaning that correct responses require participants to press a designated key on the keyboard when the current stimulus matches the one presented 1 item back. For each subsequent block, *n*'s value increases by 1 item (e.g., 2-back, 3-back). With each visually presented stimulus, participants simultaneously hear a letter. They are to select the corresponding key (i.e., *left shift* key for auditory, *right shift* key for visual) for each matched stimulus, whether visually or auditorily presented. Updating ability was determined by an overall accuracy score for each block, consisting of number hits (correctly identified targets), misses (missed targets), and correct rejections (not pressing the key when an incorrect stimulus is presented), which was used in analyses for the present study. Since participants are required to maintain and update a continuously changing set of stimuli while responding to each item, the N-

Back task demonstrates good face validity as a test of working memory (Jaeggi et al., 2010; Kane et al., 2007). The N-Back also has high internal consistency ($\alpha = .96$).

Connections Test (Salthouse et al., 2000). The Connections Test is a version of the Trail Making Test (TMT; Reitan, 1958, 1992), a neuropsychological assessment measuring visual attention and set-shifting. For the Connections Test, participants are instructed to connect a 7x7 arrangement of circles containing either numbers or letters, using a designated pattern. The test consists of four conditions: 1) targets contain only numbers and connections are made in numerical order, 2) targets contain only letters and connections are made in alphabetical order, 3) targets contain both letters and numbers and connections are made in alternating numerical and alphabetical sequence (i.e., 1-A-2-B), and 4) targets are similar to the third condition but instructions are reversed in that participants must select letters before numbers in alphabetical and numerical sequence (i.e., A-1-B-2). Conditions 1 and 2 are termed "simple" sequences, and conditions 3 and 4 are referred to as "complex" sequences. All patterns require each successive connection between circles to be made with those adjacent to the previous circle (i.e., above, below, beside, diagonal) and must be completed as quickly as possible. As in the TMT, the Connections Test yields the time in which it takes to complete each pattern. Of particular interest for the current study was the switching process as captured by the complex sequences when participants were instructed to complete patterns that involve alternating from numbers to letters and letters to numbers, respectively. Specifically, the capacity of set-shifting can be measured by averaging completion times for sequence type (simple and complex) and calculating a derived ratio score of complex performance relative to simple performance (i.e., Complex/Simple). This derived ratio score has been demonstrated as a useful measure of set-shifting ability and has shown significant positive correlations ($r = .45$, $p < .05$) with other measures of this ability (i.e.,

alternating-switch tasks; Arbuthnott & Frank, 2000). A computerized version for the Connections Test through PEBL was administered.

Victoria Stroop Test (VST; Spreen & Strauss, 1998). The VST is a brief version of the Stroop task (Stroop, 1935), a standardized color-naming task designed to assess response inhibition. The VST involves the following three conditions: 1) naming the ink color of nonword stimuli (dots; control task; "Dot" condition), 2) naming of the ink color of neutral (noncolor words; "Word" condition), and 3) naming the ink color of color words printed in contrasting colors (e.g., yellow written in blue ink; "Interference" condition). In particular, the last task requires the overriding of automatic, dominant reading responses in order to respond with the more effortful color-naming responses. The VST consists of 24 items within each of the three tasks, which also include training tasks to assist the participant in establishing the relevant response to a specific task. The PEBL computerized version of this task was used. For this version of the VST, four colors (i.e., blue, red, green, yellow) correspond to keyboard numbers 1 – 4. Following the completion of the VST, number of errors and completion times are recorded for each condition. Response inhibition ability is measured by two interference scores (i.e., low and high interference). Low interference is derived by calculating the ratio between the completion time for the Word condition and the completion time for the Dot condition (i.e., Word/Dot). High interference is the ratio between completion times for the Interference condition and the Dot condition (i.e., Interference/Dot). The current study used the number of errors and the high interference ratio in analyses to determine impairment in response inhibition. Evidence supports the psychometrically soundness of the VST for measuring inhibition as it demonstrates excellent test-retest reliability (rs = 0.83 – 0.91) for time to complete each task

condition, as well as sensitivity to frontal lobe impairment (i.e., typically associated with reduced response inhibition; Spreen & Strauss, 1998).

Procedure

Both undergraduate students and individuals from the community were recruited for participation in the current study. A description of the study was posted on the University of Mississippi Department of Psychology's Sona Systems (i.e., online subject pool system for university research), as well as on various social media platforms and university research listservs. The description included information on delivery modality of the study (i.e., video conferencing via Zoom Video Communications Inc.; Zoom), eligibility criteria, and how to contact the researcher if interested (for social media and listserv recruits). Upon registering for the study, participants were emailed a list of study reminders (i.e., required internet access, video conferencing capabilities, and a quiet environment at the time of their study session), as well as a Zoom access link for their scheduled study session. Once in the Zoom meeting, informed consent and self-report questionnaires were completed electronically via Qualtrics, the university's survey platform.

Following completion of self-report measures, a PEBL file was shared with participants via email and directions for accessing the EF tasks were provided. The researcher remained on the Zoom call throughout the completion of these tasks to assist participants as needed. Once the study was complete, student participants were awarded course/extra credit.

Data Analysis

Preliminary Analyses

Statistical analyses were completed using SPSS Version 27, and alpha was set at $p < .05$. Data were collected from 141 participants. Initial data screening revealed 31 participants with

greater than 5% missing data which were subsequently excluded from further analyses. Testing for outliers using Mahalanobis distance resulted in the removal of 4 additional participants. Lastly, only those participants with complete responses for all study measures (i.e., PSQI, DOCS, DASS-21) and EF tasks (i.e., Connections, Dual N-back, and VST) were included in the analyses, resulting in a total of 91 participants ($N = 91$). Following statistical assumption checks and data cleaning, descriptive statistics, frequencies, and initial correlations were run for all study variables to determine whether depression covaried with each and should be controlled for in subsequent analyses.

CHAPTER 3

RESULTS

Primary Analyses

Correlational Analyses

Correlational analyses were run to identify relations among constructs of interest (i.e., sleep impairment, OC symptoms, depressive symptoms, set-shifting, updating, response inhibition). Although it was initially proposed that sleep impairment would be measured by general sleep habits over the past month (as measured by the PSQI) *and* amount of sleep participants obtained the night before testing, due to a low response rate for number of hours slept the night prior to testing, only general sleep was used. As a result of nonnormal distributions of several data, Spearman correlations were run to determine preliminary correlates. A summary of these findings can be found in Table 1. Contrary to Hypotheses 1a and 1b, EF performances were not significantly correlated with either OC symptom severity (Updating r = -.01, Set-Shifting r = -.002, Response Inhibition Efficiency r = .02, Response Inhibition Errors r = .06) or sleep impairment (Updating r = .06; Set-Shifting r = -.03, Response Inhibition Efficiency r = .04; Response Inhibition Errors r = .004). Regarding Hypothesis 1c, general sleep impairment was positively associated with OC symptom severity (r = .41, p < .01), suggesting that increased ongoing sleep disturbances are related to more severe levels of OC symptoms. Additionally, depressive symptoms were significantly correlated with both OC symptom severity (r = .61, p < .001) and general sleep impairment (r = .52, p < .01) in a positive direction. Results

suggest those with higher levels of depressive symptoms are more likely to also experience more severe OC symptoms and greater levels of general sleep impairment.

Hierarchical Multiple Regression Analyses

Hierarchical multiple regression analyses were used to assess Hypotheses 2a – 2c testing whether performance on EF tasks (i.e., updating, set-shifting, response inhibition) would predict participant severity of OC symptoms above and beyond sleep impairment and presence of depressive symptoms. Table 2 includes a summary of regression results. Specifically, to test Hypotheses 2a – 2c, general sleep impairment and depressive symptoms were initially entered into Step 1. Results indicated both general sleep impairment and depressive symptoms were significant predictors of OC symptom severity $F(2, 88) = 39.20$, $p < .001$, accounting for 47.10% of the overall model variance. These results suggest individuals experiencing greater general sleep impairment and depressive symptoms are more likely to experience higher levels of OC symptoms. Scores for EF tasks (i.e., updating, set-shifting, and response inhibition, respectively) were entered into Step 2. Results revealed none of the EFs to be unique predictors of OC symptom severity.

Moderation Analyses

Moderation analyses using the PROCESS macro version 4.1 (Hayes, 2022) for SPSS was used to test to test Hypotheses 3a – 3c stating that greater sleep impairment will moderate the effect of EF performance on OC symptom severity (see Figures 1 – 4). Specifically, OC symptoms were entered into the model as the outcome variable, while EF performance scores (i.e., updating, set-shifting, and response inhibition, respectively), general sleep quality scores, and interaction terms (i.e., Updating x General Sleep, Set-Shifting x General Sleep, Response Inhibition x General Sleep) were entered as predictor variables. Results did not support

moderation, and none of the EFs of interest were unique predictors of OC symptoms (see Table 4).

Hypothesis 3a. The overall model with updating, general sleep quality, and the updating x general sleep quality interaction term entered as predictors was significant and accounted for 27.31% of the variance, $F(3, 87) = 10.89$, $p < .001$. The updating x general sleep quality interaction term was not significant, $F(1, 87) = .42$, $p = .52$.

Hypothesis 3b. When predictor variables included set-shifting, general sleep quality, and set-shifting x general sleep quality, the overall model was significant, accounting for 27.09% of the variance, $F(3, 87) = 10.78$, $p < .001$. Again, the interaction term set-shifting x general sleep quality was not significant, $F(1, 87) = .14$, $p = .71$.

Hypothesis 3c. For response inhibition efficiency, general sleep quality, and response inhibition efficiency x general sleep, the overall model was also significant and 27.58% of the total variance was accounted for, $F(1, 87) = 11.04$, $p < .001$. Similarly, when response inhibition efficiency was replaced with the response inhibition error score, the overall model was also significant and accounted for 28.83% of the variance, $F(1, 87) = 11.75$, $p < .001$. However, the response inhibition efficiency x general sleep quality [$F(1, 87) = .52$, $p = .47$] and response inhibition errors x general sleep quality [$F(1, 87) = .96$, $p = .33$] interaction terms were not significant.

Post Hoc Analyses

EF Performance Between Medication and Non-Medication Groups

Approximately 52% ($n = 47$) of participants reported regularly taking medication at the time of testing. Because some medications have been shown to impact EFs, this study wanted to first explore whether performances on EF tasks were significantly different between medication

and non-medication groups. Specifically, post hoc independent samples t-tests were conducted to determine whether taking medication (prescribed and over-the-counter) had an effect on participant scores on updating, set-shifting, and response inhibition. Findings demonstrated no significant differences in scores between the two groups.

Correlational Analysis Among Clinically Elevated OC Symptoms, Sleep, and EF Performance

Factor analyses of the DOCS suggests a cutoff score of 18 is useful for determining nonclinical from clinical populations, correctly classifying 78% of those with clinical levels of OCD (Abramowitz et al., 2010). In the present study, 32% of participants (n = 30) endorsed symptoms at or exceeding the clinical threshold. Spearman correlations were run to explore whether clinical levels of OC symptoms were associated with remaining variables of interest (i.e., general sleep impairment, updating, set-shifting, response inhibition). Results indicated OC symptoms were positively related to sleep impairment (r = .59, p <.001), suggesting that as clinical OC symptoms increase in severity, quality of sleep worsens. Correlations of OC symptoms with updating (r = -.59, p < .001) and set-shifting (r = -.59, p < .001) revealed significant relationships between severity of symptoms and EF performances, suggesting that as OC symptom severity increases, updating declines but set-shifting performances improves. In relation to response inhibition, findings suggested clinical levels of OC symptoms were unrelated to both efficiency (r = .00; p < .99) and number of errors (r =.05, p = .81).

CHAPTER 4

DISCUSSION

As a highly prevalent and debilitating condition, OCD research has highlighted the need for continued study of this heterogeneous disorder. Contributing to the complexity of this disorder is the bidirectional relationship it shares with deficits in EF. Frequently co-occurring with both OCD and impaired EF is that of disrupted sleep (Munafo et al., 2018; Theilsch et al., 2015). However, at present, no study reviewed has examined associations among these three constructs together. Therefore, this study aimed to determine whether sleep impairment moderated the relationship between EF performance and severity of OC symptoms in a nonclinical community sample.

Surprisingly, at the basic correlation level, not all primary variables of interest were related. As expected, OC symptoms, sleep impairment, and depressive symptoms were all associated with each other. However, EF performance was not significantly related to any of the other investigated constructs. As the construct of EF is highly complex and involves a number of cognitive processes, findings regarding its associations with OCD, sleep difficulties, and depressive symptoms are mixed (Abramovitch et al., 2013; Bohne et al., 2008; Krishna et al., 2011; Penadés et al., 2005). Evidence suggests EFs are often differentially impacted by certain aspects of each of the aforementioned conditions, and EF deficits may even be better attributed to moderating factors unique to each.

For example, research has demonstrated the impact of insufficient sleep on EF, with a number of sleep factors uniquely influencing this relationship. Specifically, course and severity

of sleep difficulties, as well as individual differences (e.g., demographic and genetic factors) have been shown to influence this relationship (Ebisawa, 2007; Hirshkowitz et al., 2015). Regarding course of sleep impairment, evidence has suggested distinct EF deficits occur depending on whether an individual experiences acute (i.e., absence or reduction in the usual sleep time lasting from one or two days) or chronic (i.e., routinely disrupted sleep over an extended period of time) sleep disturbance. The present study used global PSQI scores to assess presence of sleep disturbances over the last month, representing longer-term, chronic sleep quality. Thus, it is possible that using measures of more proximal, acute sleep factors may have revealed a relation to EF performance that longer-term sleep patterns assessed by the PSQI did not.

Not only does length of time with sleep disturbances lead to differences in EF deficits, but it appears that the severity of sleep symptoms plays a role as well. Many studies examining cognitive performance related to sleep have focused on those with sleep disorders (Rana et al., 2018), making it difficult to generalize findings to non-clinical populations. The DSM-5 distinguishes disordered sleep from that of transient sleep disturbance by the amount of time with sleep impairment (e.g., > 3 months). In this study, participant global PSQI scores ranged from 2 – 16 with 81% of participants exceeding the clinical cutoff for impaired sleep. Despite a large portion of the sample endorsing clinical levels of sleep disturbances, the majority of these scores fell to the low end of the clinical range, restricting the sample variance. Furthermore, the PSQI only assesses the presence of symptoms within the last month, and it is unclear whether sleep disturbances in this sample are consistent with those seen in clinical sleep disorders. Perhaps, greater variance of clinical levels of sleep disturbance, in addition to a clinically sleep disordered

population would have demonstrated the expected effect between sleep impairment and EF performance.

Quality of sleep is also subject to a number of individual differences (e.g., age, gender, circadian rhythms; Ebisawa, 2007) and not everyone has the same sleep need (i.e., amount of sleep required to feel rested and alert; Hirshkowitz et al., 2015). One highly studied individual factor in both sleep and EF research is that of the effects of aging on sleep, with evidence supporting natural age-related decline in quality of sleep and EF. In fact, studies have shown aging can actually enhance susceptibility to sleep-related EF impairment, and research has aimed to parse apart normal, age-related EF impairment from that resulting from poor sleep (Rana et al., 2018). Studies have demonstrated sleep impacts EF differently based on age, with some research showing little to no EF impairment in young sleep-deprived adults compared to their older counterparts (Alhola & Polo-Kantola, 2007; Rana et al., 2018). Considering 87% of participants in the present study fell within the young adulthood age range (i.e., ages 18 – 35), with 66% under the age of 25, it is highly possible age-related factors influenced the lack of a significant relationship between poor sleep and EF performance.

Similarly, findings on EF in OCD have been inconsistent with some research asserting that any EF impairment in the disorder is better explained by moderating factors (e.g., medication effects, symptom presentation, comorbidities; Kuelz et al., 2004; Moriz et al., 2009; Rasmussen et al., 2016). For example, commonly prescribed medications to treat OCD and its comorbid disorders (e.g., SSRIs, benzodiazepines) have demonstrated impairments on reaction times, processing speeds, learning, cognitive flexibility, and response inhibition (Skandali et al., 2018; Kuelz et al., 2004). While the present study found no differences in EF performance between participants who regularly took medication (prescribed and over-the-counter) and those

who did not, participants reported mostly subclinical levels OC symptoms. As such, although medications were not distinguished by type (psychotropic vs. other), the number of participants experiencing clinical levels OC symptoms was underpowered to determine whether the use of psychotropic medications typically prescribed for OCD had an effect on EF performance in this sample.

In addition, EF seems to play a role in the clinical severity, course, and treatment response in OCD populations. For instance, updating impairment in OCD populations has been associated with poorer patient insight into the disorder, and poorer insight has been linked to greater severity, number of symptoms, and length of illness (Kashyap et al., 2013). In relation to length of illness, which has been associated with greater OC symptom severity, a study by Nakao et al. (2009) found EF deficits in a group with long-term (i.e., M = 20 years) OCD, but in not those with short-term OCD (i.e., M = 5 years). Other research has suggested particular EF deficits may come secondary to OC symptoms as a result of devoting cognitive resources to OC-related stimuli (e.g., distracting obsessional thoughts limiting working memory capacity; Abramovitch et al., 2013; Woods et al., 2002). Taken together, it is possible that EF deficits are only made manifest in more chronic, clinical presentations of the disorder after a substantial period of time. It may even mean that while some EFs are predictive of OC symptom severity as hypothesized in the present study, the effect may be much more subtle or function in a manner that the EF tasks used in this study were not sensitive enough to tap into. Regardless, the use of a nonclinical OC population in the present study likely influenced findings related to EF performance and OC symptoms. Specifically, DOCS total scores were used to indicate the severity of OC symptoms, with scores ranging from 0 – 49 and 32% (n = 30) of participants reporting symptoms exceeding the clinical threshold. Indeed, although post hoc correlational

analyses with this clinical subset of participants demonstrated an association among OC symptoms and two EFs (i.e., updating and set-shifting), meaningful conclusions are hard to draw with a mostly subclinical sample.

Notably, the present study investigated overall OC symptom severity and did not distinguish symptoms by symptom domain, as this was not feasible given limited endorsement of clinical symptom levels. However, the heterogeneity of OC symptom dimensions (i.e., contamination/checking, symmetry/ordering/arranging/counting, sexual/religious, aggression, somatic, hoarding/collecting, miscellaneous) has been identified as contributing to the mixed and inconsistent findings in the EF-OCD literature. OC symptom dimensions have been associated with distinct neural mechanisms, and research demonstrates EFs are differentially impacted depending on symptom presentation (Bragdon et al., 2018; Lawrence et al., 2006; Mataix-Cols et al., 2005; Rosario-Campos et al., 2006), making this a worthy area of continued study.

Although depressive symptoms were associated with both sleep impairment and OC symptoms, basic correlations of this study did not support a relationship between depressive symptoms and EF. Also similar to sleep and OC symptoms, research supports EF impairment in those with severe levels of depression with some studies showing small to moderate effects in those with major depressive disorder (Snyder et al., 2015). Participant scores on the DASS-21 depression scale ranged from 0 – 32 with approximately 62% scoring within the normal range of depressive symptoms. In fact, only 25% endorsed more than moderate levels of symptom occurrence, suggesting depressive symptoms were likely not severe enough to demonstrate an association with EF performance.

Thus far, much has been discussed about the severity of symptoms in relation to EF. Interestingly, though clinical severity may be required to demonstrate a relationship with EF

performance, general sleep impairment and depressive symptoms emerged as significant predictors of OC symptoms even at the subclinical level. Perhaps this further supports the idea that EF deficits emerge only with high cognitive load associated with managing severe OC symptoms over time.

Given EFs were consistently the only variables in this study not related to any other variables of interest, aspects specific to the EF tasks must be considered. Notably, research has demonstrated the role of emotion in relation to EF in various contexts. That is, distinctions have been made between "cold" (i.e., logical, mechanistic) and "hot" (i.e., emotional, based on beliefs and desires) components of EF. Specifically, "cold" components are activated in unemotional tasks of planning, problem-solving, sustained attention, etc., while "hot" components are activated in relation to reward and punishment, modulating one's social behavior, making important personal decisions (Raymond et al., 2008). The OCD literature provides support for the relevance of emotional content on EF performance. For example, a study examining set-shifting in OCD demonstrated EF deficits when participants were presented with OC-related stimuli, but not with neutral stimuli (Morein-Zamir et al., 2010). This makes theoretical sense given the ACC region of the brain is implicated in the neural circuitry associated with all three basic EFs, as well as OCD (Suveg & Zeman, 2004). Stimuli selection is certainly an important consideration in the present study, in that none of the EF tasks employed OC-related (emotional) stimuli, which may have otherwise yielded an association between the two. Future research could benefit from integrating OC-related stimuli into these EF tasks to determine whether emotionally valanced stimuli impacts EF performance.

A number of other limitations existed within this study. Due to social distancing guidelines set forth in response to the COVID-19 pandemic, several adaptations to a typical in-

person experimental design were made to enable remote data collection. Adaptations included meeting with participants via videoconferencing (i.e., Zoom Video Communications, Inc.; Zoom) and use of computerized variations of well-known cognitive assessments to assess EF performance. While there were certainly advantages to remote data collection (e.g., health safety, greater flexibility in scheduling, broader recruitment reach), several novel limitations were also presented. Recruitment was limited to individuals with access to the internet, video-conferencing technology, and certain types of computers that supported the EF software. Additionally, because participants could participate in the study from anywhere, controlling for potential naturally occurring confounds (e.g., environmental distractions) was limited. Although participants were instructed to select a study timeslot when they could be located in a quiet area sans interruptions, many participants completed the study in their homes or dorm rooms where family members and roommates were also present. Other potential confounding factors include technological variations across participants, including differences in computer features (e.g., desktop vs. laptop, external mouse vs. trackpad) and internet connectivity. To some extent, this could have enhanced the ecological validity of the study environment compared to a laboratory setting, as some have argued that experimental EF tasks are vastly discrepant from real-world conditions requiring EF (Burgess et al., 1997; Van der Sluis, 2007).

This study examined the role of sleep in the relationship between EF and OCD as it has been previously demonstrated to contribute to the experience of both OCD and EF impairment separately. Although main findings were null, results nonetheless contribute to the literature in terms of offering points for future consideration in a relatively unstudied area of research. Areas for future study of these three variables have been highlighted, including individual factors shown to influence symptom expression (e.g., demographics, genetic factors), length of illness,

acute versus chronic impairment, and effects of psychotropic medication. As has been repeatedly mentioned, the future use of clinical populations would be important for determining sleep as a moderator of the EF-OCD association prior to investigating more subtle effects likely to appear in subclinical populations. Furthermore, although beneficial in many regards, the remote methodology introduced several novel limitations. Future studies should consider an in-person, laboratory paradigm to allow for adequate experimental control when determining the existence of the hypothesized relations. Overall, much is still unclear regarding EF deficits and OCD. The EF-OCD literature points to the interplay of enumerable factors influencing their relation, making two already very complex constructs all the more complicated. Future studies are needed to investigate specific emotional and behavioral components that impact the EF-OCD relationship, as well as factors related to sleep impairment co-occurring with each.

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APPENDIX

Table 1. *Overall sample demographics.*

		n	*%*
Age (*M* ± *SD*)	25.87 ± 12.50		
Gender	Male	28	30.8
	Female	62	68.1
	Other	1	1.1
Race/Ethnicity	White	79	86.8
	Black/African American	2	2.2
	Asian	5	5.5
	Native Hawaiian/Other Pacific Islander	1	1.1
	Multiracial	4	4.4
	Hispanic	2	2.2
Marital Status	Never married	64	70.3
	Married	20	22.0
	Not married, living with a partner	3	3.3
	Divorced/Annulled	3	3.3
	Widowed	1	1.1
Education	High school diploma or equivalent	19	20.9
	Some college, no degree	37	40.7
	Two-year degree	6	6.6
	Bachelor's degree	14	15.4
	Master's degree	14	15.4
	Doctoral or professional degree	1	1.1
Employment	Unemployed	40	44.0
	Homemaker	3	3.3
	Part-time	20	22.0
	Full-time	27	29.7
	Armed Forces	1	1.1
Income	Less than $25,000	20	22.0
	$25,000 - $50,000	9	9.9
	$50,000 - $100,000	19	20.9
	$100,000 - $200,000	32	35.2
	$200,000 - $300,000	6	6.6
	Greater than $300,000	5	5.5
Religion	Protestant	38	41.8
	Catholic	14	15.4
	Other Christian	15	16.5
	Muslim	1	1.1
	Not Religious	23	25.3
Medications	Yes	47	51.6
	No	44	48.4

Note. (*N* = 91).

Table 2. *Descriptive statistics and Spearman correlations.*

Variable	*M* (*SD*)	Observed Range	Possible Range	*1*	*2*	*3*	*4*	*5*	*6*	*7*
1. OC Symptoms	14.74 (11.27)	0 – 49	0 – 80	--						
2. Updating	.88 (.09)	.67 – 1.22	--	-.01	--					
3. Set-Shifting	.9996 (.0017)	.9956 – 1.0047	--	-.002	-.07	--				
4. Response $Inhibition_{eff}$	1.02 (.27)	.47 – 1.91	--	.02	-.03	.06	--			
5. Response $Inhibition_{err}$	2.32 (2.49)	0 – 11	--	.06	-.09	-.10	.40**	--		
6. General Sleep Impairment	7.42 (3.43)	2 – 18	0 – 21	.41**	.06	-.03	.04	.004	--	
7. Depressive Symptoms	4.26 (4.26)	0 – 32	0 – 42	.61***	.11	-.13	-.11	-.01	.52**	--

Note. ($N = 91$); DOCS = Dimensional Obsessive-Compulsive Scale; PSQI = Pittsburgh Sleep Quality Index; $DASS\text{-}21_{dep}$ = Depression subscale of the Depression, Anxiety, Stress Scales-21; EF = Executive Function. $p < .001$***; $p < .01$**; $p < .05$*

Table 3. *Summary of hierarchical multiple regression analyses.*

			OC Symptoms	
Hypotheses 2a – 2c	***ΔR²***	***ΔF***		
Step 1:	.47	39.20***	***β***	***t***
General Sleep			.27	2.99**
Depressive Symptoms			.52	5.79***
Hypothesis 2a				
Step 2:	.00	.12		
Updating			-.03	-.34
Hypothesis 2b				
Step 2:	.001	.13		
Set-Shifting			.03	.36
Hypothesis 2c				
Step 2:	.01	1.18		
Response Inhibition$_{eff}$			.09	1.08
Response Inhibition$_{err}$			.11	1.47

Note. (N = 91); OC = Obsessive-Compulsive; Response Inhibition$_{eff}$ = Response Inhibition Efficiency; Response Inhibition$_{err}$ = Response Inhibition Error; β = Standardized beta weight provided for hierarchical multiple regression. $p < .001$***; $p < .001$**; $p < .05$*

Table 4. Sleep as a moderator of the relationship between EF and OC symptoms.

	OC Symptoms		
Hypothesis 3a	*b*	*SE*	*p*
Updating	-15.11	24.97	.55
General Sleep	.08	2.76	.98
Updating x General Sleep	2.07	3.18	.52
Hypothesis 3b			
Set-Shifting	410.78	1492.06	.78
General Sleep	74.62	195.02	.70
Set-Shifting x General Sleep	-72.93	195.07	.71
Hypothesis 3c			
Response Inhibition$_{eff}$	-3.47	8.36	.68
General Sleep	1.01	1.00	.32
Response Inhibition$_{eff}$ x General Sleep	-.65	.89	.47
Response Inhibition$_{err}$	-.25	.84	.77
General Sleep	1.41	.41	.001
Response Inhibition$_{err}$ x General Sleep	.09	.09	.33

Note. (N = 91); OC = Obsessive-Compulsive; EF = Executive Function; Response Inhibition$_{eff}$ = Response Inhibition Efficiency; Response Inhibition$_{err}$ = Response Inhibition Error.

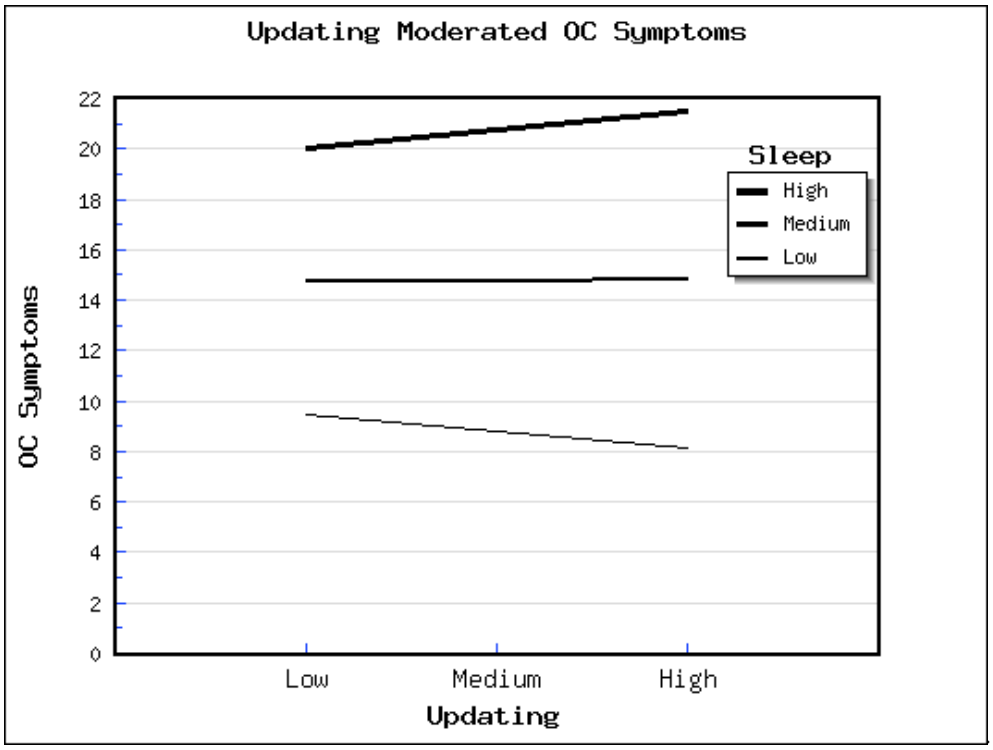

Figure 2. *Sleep as a moderator of the relationship between set-shifting and OC symptoms.*

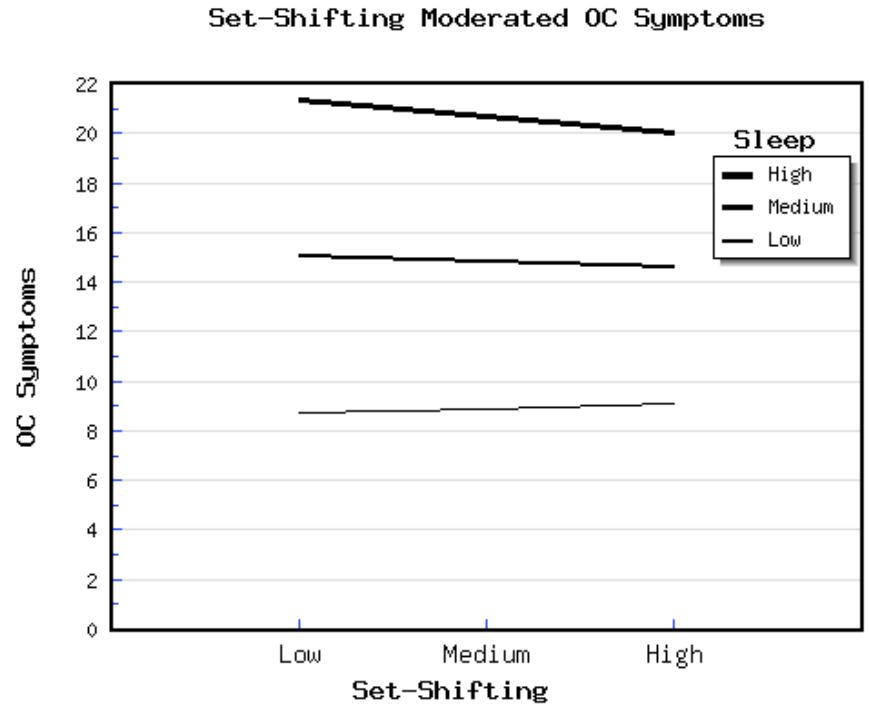

Figure 3. *Sleep as a moderator of the relationship between response inhibition efficiency and OC symptoms.*

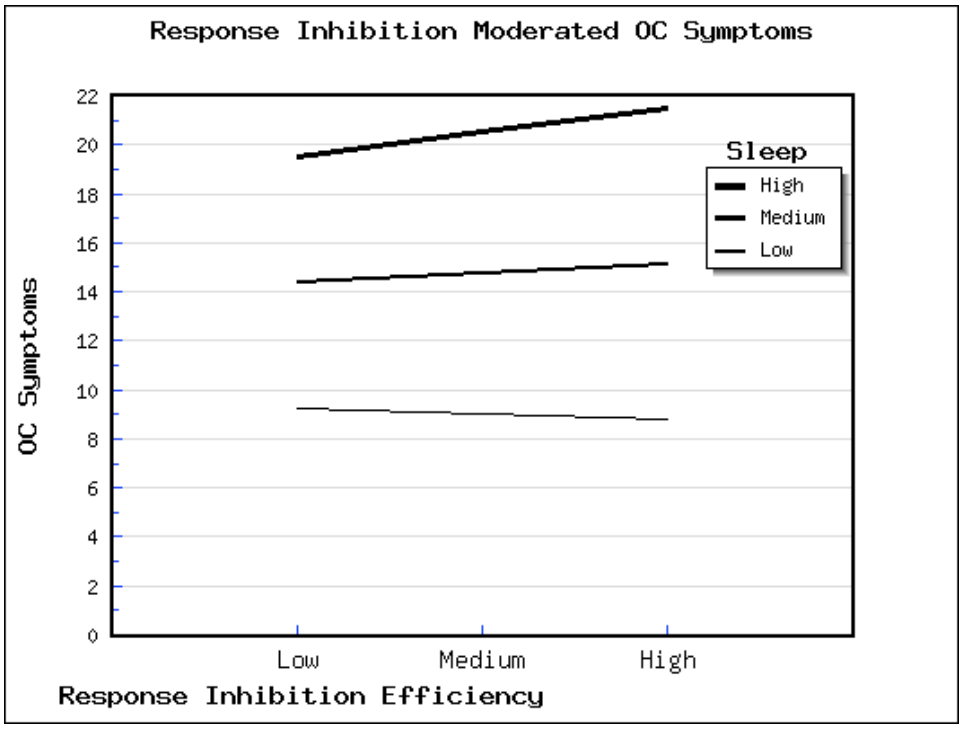

Figure 4. *Sleep as a moderator of the relationship between response inhibition errors and OC symptoms.*

VITA

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EDUCATION

2022 (Anticipated)	**Doctor of Philosophy, Clinical Psychology** **University of Mississippi, University, MS (APA-Accredited)**
Proposed: 09/14/2020	Dissertation: *Repetitively counting sheep: Sleep as a moderator of executive function performance on obsessive-compulsive symptoms* Major Advisor: Danielle J. Maack, Ph.D.
2021 -	**Predoctoral Psychology Internship (APA-Accredited)** **Mississippi State Hospital, Whitfield, MS 39193** Training Director: Mary Ashley Angelo, Ph.D.
2019	**Master of Arts, Clinical Psychology** **University of Mississippi, University, MS (APA-Accredited)** Thesis: *Emotion regulation as a predictor of pediatric obsessive-compulsive symptoms* Major Advisor: Danielle J. Maack, Ph.D.
2013	**Master of Science, Mental Health Counseling** **Mississippi College, Clinton, MS**
2011	**Bachelor of Arts, Psychology** **University of Mississippi, University, MS**
2009	**Associate of Arts** **Pearl River Community College, Poplarville, MS**

LICENSES AND CERTIFICATIONS

2020	**Provider Training for Cognitive Behavioral Therapy of Insomnia** *Medical University of South Carolina*

Completed 6.5 hours of Continuing Education training

2020 **Unified Protocol for Transdiagnostic Treatment of Emotional Disorders in Children and Adolescents Training**
Trainer: Jill Ehrenreich-May, Ph.D.
Completed 8 hours of Continuing Education training

2020 **American Psychological Association Telepsychology Best Practice 101**
American Psychological Association
Completed 8 hours of Continuing Education training

2019 **Examination for Professional Practice in Psychology (EPPP)**
Passed at the Doctoral level

2016 – Present **Licensed Professional Counselor**
Mississippi State Board of Examiners for
Licensed Professional Counselors, License # 2061

CLINICAL EXPERIENCE

August 2021 – Present **Predoctoral Intern**
Mississippi State Hospital, Whitfield, MS
Training Director: Mary Ashley Angelo, Ph.D.

Rotation: Male Receiving Unit (April 2022 – Present)
Supervisors: Mary Ashley Angelo, Ph.D., Joseph Griebler, Ph.D., Jasmine Ezagui, Psy.D.

Rotation: Administrative Psychology (December 2021-April 2022)
Supervisor: Joseph Griebler, Ph.D.

Rotation: Substance Use Services (August – December 2021)
Supervisors: Mary Ashley Angelo, Ph.D. and Vicki Prosser, Ph.D.

July 2020 – July 2021 **Therapist**
Communicare, Lafayette County Office
Region 2 Community Mental Health Center, Oxford, MS
Supervisors: Dixie Church, LMFT and Scott Gustafson, Ph.D., ABPP

- Conduct structured clinical intake assessments
- Develop individualized treatment plans
- Provide evidence-based CBT interventions

- Coordinate case management
- Deliver crisis intervention
- Complete documentation and paperwork for underserved clients with various psychiatric disorders, serious mental illness, and comorbid chronic health conditions and substance used disorders
- Manage a caseload of 20 – 30 clients at a time

July 2019 – June 2020 **Therapist**
Behavior, Attention, and Developmental Disabilities Consultants, LLC, Southaven, MS
Supervisor: Emily Thomas Johnson, Ph.D., BCBA-D

- Conducted biopsychosocial intake assessments
- Performed psychological diagnostic and developmental assessments, primarily consisting of attention-deficit/hyperactivity disorder and autism spectrum disorders evaluations, ages ranged from 3 – 18
- Scored and interpreted assessment results
- Completed approximately 2 – 4 hours of assessment report writing each week
- Delivered 8 hours of Applied Behavior Analysis per week to children ages 6 – 10
- Collected and entered ongoing behavioral data each week

July 2018 – June 2019 **Therapist**
Stonewater Adolescent Recovery Center, Oxford, MS
Supervisor: Scott Gustafson, Ph.D.

- Conducted psychological assessments and biopsychosocial intakes on incoming patients
- Facilitated group DBT skills psychotherapy a population of adolescent males struggling with substance use
- Provided individual psychotherapy using evidence-based CBT
- Participated on an interdisciplinary treatment team consisting of a clinical psychologist, psychiatrist, nurse, licensed social worker, licensed counselor, family counselor, academic advisor, in addition to social work interns
- Managed a caseload of 5 – 8 adolescents at a time

July 2017 – June 2018 **Therapist**
The Baddour Center, Education and Behavioral Supports Division; Senatobia, MS
Supervisor: Shannon Hill, Ph.D., BCBA-D

- Completed intake assessments with adults with intellectual disabilities

- Administered neurocognitive screeners and functional behavior assessments
- Led a DBT skills group adapted to individuals with intellectual disabilities
- Delivered individual behavioral therapy and developed treatment and behavior plans
- Aided in the development and facilitation of staff trainings on various topics as needed
- Assisted in the development of a Direct Service Professional (DSP) Support Group for those responsible for the direct care of residents
- Managed a caseload of 15 – 20 residents at a time

June 2017 – July 2021 **Graduate Student Therapist**
Psychological Services Center, University, MS
Supervisors: Danielle J. Maack, Ph.D.
Stefan Schulenburg, Ph.D.
John Young, Ph.D.
Laura J. Dixon, Ph.D.
Alan Gross, Ph.D.

- Conducted structured clinical interviews
- Administered self-report measures to assist in monitoring treatment progress
- Delivered individual therapy to children and adults, as well as parent training, using evidence-based techniques
- Maintained treatment documentation
- Observed CBT techniques for children and adults
- Managed a caseload of 2 – 4 clients at a time

RESEARCH EXPERIENCE

April 2020 – Present **Dissertation Research**
Anxiety, Depression, Emotion, Personality, and Temperament (ADEPT) Lab
University of Mississippi, University, MS
Supervisor: Danielle J. Maack

2016 – 2019 **Thesis Research**
ADEPT Lab
University of Mississippi, University, MS
Supervisor: Danielle J. Maack, Ph.D.

2016 – 2021 **Graduate Research Assistant**
ADEPT Lab
University of Mississippi, University, MS
Supervisor: Danielle J. Maack, Ph.D.

- **Pregnancy Initiative**
  - Recruited pregnant and postpartum patients at two OBGYN clinics to take part in a study investigating levels of anxiety, depression, nausea and vomiting, disgust, and quality of sleep
  - Provided mental health feedback to nurses and physicians
- **Emetophobia Study**
  - Assisted in the development of Behavioral Avoidance Tasks (BATs) used in a study examining behavioral avoidance associated with the fear of vomiting

2015 – 2016 **Research Assistant**
Department of Psychiatry and Human Behavior
University of Mississippi Medical Center, Jackson, MS
Supervisor: Laura J. Dixon, Ph.D.

- Recruited and assessed pregnant women in a study examining anxiety sensitivity as a vulnerability factor of anxiety and mood disorders in pregnant women

August 2011 – December 2011 **Undergraduate Research Assistant**
Department of Psychology
University of Mississippi, University, MS
Supervisor: Marilyn Mendolia, Ph.D.

- Assisted in the examination of the recognition of facial expression of emotions

PROFESSIONAL PRESENTATIONS

ORAL PRESENTATIONS

Sapp, B. S. & Maack, D. J. (2021, March). *Sleep-deprived mamas: Prenatal depression and contributing sleep quality components.* Paper accepted for presentation at the 67th annual meeting of the Southeastern Psychological Association in Orlando, FL.

Sapp, B. S. & Maack, D. J. (2019, September). *Rock-a-bye, Mommy, in the treetop: Sleep quality components impact on prenatal depression.* Data blitz presented at the 70th annual convention of the Mississippi Psychological Association in Biloxi, MS.

Sapp, B. S., Maack, D. J., & Young, J. N. (2019, April). *Emotion regulation and obsessive-compulsive symptoms: The moderating effects of gender.* Data blitz presented at the 6th annual UM Conference of Psychological Science in University, MS.

Sapp, B. S. & Maack, D. J. (2018, October). *Pregnancy glow, easier said than done: Feasibility of an in-house support group.* Oral presentation presented at the Three Minute Thesis (3MT) competition in University, MS.

Sapp, B. S. & Maack, D. J. (2018, September). *Sleeping for two: Does sleep quality predict suicidality in pregnancy?* Data blitz presented at the 69th annual convention of the Mississippi Psychological Association in Biloxi, MS.

Sapp, B. S. & Maack, D. J., (2018, April). *Does quality of sleep predict suicidality in pregnancy?* Data blitz presented at the 5th annual UM Conference of Psychological Science in University, MS.

Sapp, B. S. (2017, October). *Pediatric OCD and emotion dysregulation.* Oral presentation presented at the Three Minute Thesis (3MT) competition in University, MS.

Maack, D. J. & **Sapp, B. S.** (2017, September). *Pregnancy glow (or gloom?): Preliminary findings of psychiatric concerns in pregnancy and how to promote integrated care.* Oral presentation presented at the 68th annual convention of the Mississippi Psychological Association in Biloxi, MS.

Sapp, B. S., Maack, D. J., & Young, J. N. (2017, April). *Does emotion regulation predict pediatric obsessive-compulsive symptoms?: Preliminary findings.* Data blitz presented at the 4th annual UM Conference of Psychological Science in University, MS.

Prendergast, K.L., **Sapp, B. S.**, and Maack, D.J. (2017, April). *Pregnancy practice makes perfect?: The effect of gravidity on anxiety, disgust, and emetophobia symptoms*. Data blitz presented at the 4thAnnual UM Conference for Psychological Sciences in University, MS.

POSTER PRESENTATIONS

Frantz, D., **Sapp, B. S.**, & Maack, D. J. (2019, April). *Does education matter? Assessing potential relations between education and depressive symptoms in pregnancy.* Poster presented at the 6th annual UM Conference of Psychological Science in University, MS.

Garner, B., **Sapp, B. S.**, & Maack, D. J., (2019, April). *Maternal age as it relates to depression and nausea in pregnancy.* Poster presented at the 6th annual UM Conference of Psychological Science in University, MS.

Sapp, B. S., Maack, D. J., & Young, J. N. (2017, September). *Did I regulate my emotions? Check and re-check: The relation between emotion dysregulation and pediatric obsessive-compulsive symptoms in a clinical sample.* Poster presented at the 68th annual convention of the Mississippi Psychological Association in Biloxi, Mississippi.

Scott, S. M., Tynes, B. L., Zhao, M. S., Pineau, D., Wickenhauser, M. E., **Sapp, B. S.,** & Maack, D. J., (2016, October). *Bible burning, dog fighting, and partner violence: Is moral disgust a real construct? Piloting a multi-method assessment of moral disgust.* Poster presented at the 50th annual convention of the Association for Behavioral and Cognitive Therapies in New York, NY.

Zhao, M., Maack, D. J., Tynes, B., Scott, S. M., Pineau, D., **Sapp, B. S.**, & Wickenhauser, M. E. (2016, October). *Disgust domains and fear of contamination predict safety behavior usage in an analogue sample.* Poster presented at the 50th annual convention of the Association for Behavioral and Cognitive Therapies.

Sapp, B. S., Dixon, L. J., Viana, A. G., Tull, M. T., & Collier, C. C. (2016, September). *An examination of the associations among socio-demographics, and stress, depression, and anxiety in pregnant women.* Poster presented at the 67th annual convention of the Mississippi Psychological Association in Biloxi, Mississippi.

RELEVANT EMPLOYMENT EXPERIENCE

October 2016 – Present

Mental Health Therapist
Delta Autumn Consulting, LLC, Oxford, MS
Supervisors: John Young, Ph.D.
Danielle J. Maack, Ph.D.

- Conduct structured clinical intake assessments and collect ongoing assessment data to measure treatment progress
- Deliver evidence-based interventions to children and adults with emotional and behavioral disorders, as well as parent training and couples therapy interventions
- Engage in deliberate practice exercises, including conducting and reviewing clinical role plays, to enhance delivery of evidence-based interventions
- Practice self-reflection and skill refinement through completion of self-rating forms including identification of problem areas, as well as review of personal role plays and peer feedback

November 2015 – August 2016

Day Treatment Specialist
Mississippi Children's Home Services, Flowood, MS
Supervisor: Heather Statham, LPC

- Managed and developed individualized treatment plans for a caseload of approximately 10 children, ages 7 – 10
- Implemented behavioral modification techniques in the classroom setting
- Introduced, monitored, and evaluated use of appropriate social skills
- Advocated for patient needs within academic and home settings

February 2015 – October 2015

Outpatient Clinic Therapist
Mississippi Children's Home Services, Flowood, MS
Supervisor: Pete Bishop, LPC

- Managed a caseload of approximately 50 children and adolescents, ages 2 – 18

- Administered clinical assessments and diagnostic interviews and developed individualized treatment plans
- Implemented PracticeWise treatment protocols consisting of CBT techniques within therapeutic practice as part of collaborative project
- Facilitated individual and family therapy on weekly and biweekly bases and educated caregivers in appropriate behavior modification/parenting skills

May 2014 – February 2015

Behavior Specialist/Therapist
Region 8 Mental Health Services, Hazlehurst, MS
Supervisor: Julie Thibodeaux, Clinical Coordinator

- Maintained a caseload of approximately 20, including 8 adolescent day treatment patients
- Implemented behavioral modification techniques in the classroom setting
- Introduced, monitored, and evaluated the use of effective social skills
- Coordinated with other mental health service providers, guardians, and school authorities to discuss treatment needs and progress

January 2014 – May 2014

Therapist
Region 8 Mental Health Services, Hazlehurst, MS
Supervisor: Julie Thibodeaux, Clinical Coordinator

- Developed patient treatment plans, goals, and interventions for a caseload of approximately 60, ages 3 – 60+
- Conducted home visitations for individual/family therapy sessions
- Provided therapeutic support for children and adolescents via the school setting

May 2013 – January 2014

Case Manager
Brentwood Behavioral Healthcare, Jackson, MS
Supervisor: Wanda Steinwinder, LCSW

- Provided case management to entire facility of 105 beds as needed, including child, adolescent, and adult units, as well as intensive outpatient and partial hospitalization programs
- Implemented inpatient treatment goals and developed hospital discharge planning
- Coordinated discharge outpatient and residential treatment

January 2013 – December 2013

Clinical Intern
Brentwood Behavioral Healthcare, Jackson, MS
Supervisor: Danny Daniel, LPC, LMFT

- Facilitated daily group therapy sessions of approximately 10 – 12 individuals
- Conducted patient interviews/assessments, as well as patient-family conferences

MENTORING EXPERIENCE

April 2020 – June 2020 **Graduate Student Mentor: Undergraduate Honors Thesis Project**
ADEPT Lab, University of Mississippi, University, MS
The nightmare before delivery: A study of nightmares, depressive symptoms, and suicidality in pregnant women
Denise Franz, Defended: July 3, 2020

October 2016 – May 2019 **Undergraduate Research Assistant Supervisor**
ADEPT Lab, University of Mississippi, University, MS

TEACHING EXPERIENCE

August 2020 – May 2021 **Teaching Assistant**
University of Mississippi, University, MS
Course: Developmental Psychology
Instructor: Kurt D. Streeter, Ph.D.

January 2020 – May 2020 **Teaching Assistant**
University of Mississippi, University, MS
Course: Developmental Psychology
Instructor: Kurt D. Streeter, Ph.D.

May 2019 – June 2019 **Teaching Assistant**
University of Mississippi, University, MS
Course: General Psychology
Instructor: Jennifer Caldwell, Ph.D.

HONORS AND ASSOCIATIONS

2017 **Finalist at the 3MT Competition**
University of Mississippi

PROFESSIONAL MEMBERSHIP

2019 – Present Southeastern Psychological Association
2017 – 2020 Mississippi Psychological Association

2010 – 2011	**Gamma Beta Phi Honor Society** University of Mississippi
	Phi Theta Kappa Honor Society
2009 – 2011	University of Mississippi
2008 – 2009	Pearl River Community College