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Anxiety, Sleep, and Functional Impairment among Clinically Anxious Youth and Healthy Controls

A DISSERTATION

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Anxiety, Sleep, and Functional Impairment among Clinically Anxious Youth and Healthy Controls

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Childhood anxiety disorders are characterized by non-transient excessive fear or worry that often affects various domains of life, including academic performance and social relationships (Albano & Detweiler, 2001; Langley, Bergman, McCracken, & Piacentini, 2004). A particular concern for children with anxiety disorders are sleep disturbances (Alfano, Ginsburg, & Kingery, 2007; Gregory, Eley, O’Connor, Rijsdijk, & Plomin, 2005). Without sufficient sleep, children experience functional impairments such as increased anxiety severity, academic problems, and behavior problems at school or home. Interestingly, the impairments of sleep deprivation are similar to those of anxiety in children (Crabtree & Witcher, 2008; Sadeh, Gruber, & Raviv, 2002). Although sleep problems are common in children with an anxiety disorder, the relationship between sleep difficulties and anxiety disorders in youth has been minimally studied (Alfano & Lewin, 2008). Moreover, whether children with a comorbid anxiety disorder and sleep difficulties experience greater functional impairment than do children with an anxiety disorder alone has not been empirically examined. The current study examined 60 non-depressed clinically anxious youth (ages 7-18) and 30 healthy controls. All participants underwent a diagnostic assessment and completed the parent/child Screen for Child Anxiety Related Emotional Disorders (SCARED), Life Events Scale (LES), Social Responsiveness Scale (SRS), and Family Risk Factor Checklist – Parent (FRFC-P) at baseline. Findings indicate that
sleep functioning did not significantly differ between groups when controlling for age. Rather, sleep functioning was significantly related to life event stressors. In contrast, group differences were revealed in social functioning. Notably, greater social functional impairment was best predicted by anxiety symptom severity rather than sleep functioning. Together, these findings lend support to the necessary examination of compounded anxiety symptom severity and sleep functioning in an effort to better understand impairments associated with youth anxiety disorders, particularly social functioning. The results of this investigation can be used to inform the assessment and treatment of youth with anxiety disorders in an effort to minimize the development, maintenance, and impairments of anxiety associated with sleep problems (Alfano & Lewin, 2008).
This dissertation by Delia Raquel Cumba fulfills the dissertation requirement for the doctoral degree in Clinical Psychology approved by Sandra Barrueco, Ph.D., as Director, and by Brendan Rich, Ph.D. and Barry Wagner, Ph.D., as Readers.

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CHAPTER 1

Introduction

Childhood anxiety disorders are characterized by non-transient excessive fear or worry that often affect various domains of life, such as academic performance or social relationships (Albano & Detweiler, 2001; Langley, Bergman, McCracken, & Piacentini, 2004). A particular concern for children with anxiety disorders are sleep disturbances (Alfano, Ginsburg, & Kingery, 2007; Gregory, Eley, O’Connor, Rijsdijk, & Plomin, 2005). Without sufficient sleep, children experience functional impairments such as increased anxiety severity, academic problems, and school or home behavior problems. Interestingly, the impairments of sleep deprivations are similar to those of anxiety in children (Crabtree & Witcher, 2008; Sadeh, Gruber, & Raviv, 2002). Although sleep problems are common in children with an anxiety disorder, the relationship between sleep difficulties and anxiety disorders in youth has been minimally studied (Alfano & Lewin, 2008). Moreover, whether children with a comorbid anxiety disorder and sleep difficulties experience greater functional impairment than do children with an anxiety disorder alone has not been empirically examined.

Anxiety Disorders in Youth

Anxiety disorders are one of the most common mental health disorders in childhood (Gurley, Cohen, Pine, & Brook, 1996), with 8 to 15% of youth reporting one or more such disorders (Anderson, 1994; Fergusson, Horwood, & Lyskey, 1993; Merikangas et al., 2010; Silverman & Ginsburg, 1998). Anxiety disorders have a high rate of comorbidity with other psychological disorders, such as depression and attention-deficit/hyperactivity disorder (ADHD) (Essau, Conradt, & Petermann, 2000; Fergusson et al., 1993; Russo & Beidel, 1994).
Furthermore, youth with anxiety disorders can experience life-long significant psychosocial impairment across a variety of domains (e.g., social, family, academic). These impairments highlight the importance of investigating the variables bidirectionally associated with the maintenance and development of anxiety disorders (Langley, Bergman, McCracken, & Piacentini, 2004).

For children to receive an anxiety disorder diagnosis, fear and/or worry must be excessive, non-transient, and beyond what is developmentally appropriate (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition: DSM-V; American Psychiatric Association, 2013). While fear and worry may be displayed differently as a function of the particular anxiety disorder, a common feature of anxiety disorders is avoidance of the feared or anxiety-provoking situation, person, or object (DSM-V; American Psychiatric Association, 2013). In addition, for a child or adolescent to meet criteria for an anxiety disorder, he/she must present with significant impairment in functioning (e.g., academic difficulties, home or school behavior problems, social relationship difficulties, or disruptive family functioning).

Empirical research indicates that anxiety disorders emerge early in childhood and adolescence (Achenbach, Howell, McConaughy, & Stanger, 1995; Pine, Cohen, Gurley, Brook, & Ma., 1998), and often have a chronic and fluctuating course (Woodward & Fergusson, 2001). The age of onset for anxiety disorders varies across specific disorders. For example, social phobia is more likely to begin during childhood and pre-teen years (Beidel, Turner, & Morris 1999; Burke, Burke, Rae, & Regier, 1991; Liebowitz, Gorman, Fyer, & Klein, 1985; Rosellini, Rutter, Bourgeois, Emmert-Aronson, & Brown, 2013; Strauss & Last, 1993). Separation anxiety
disorder (SAD) is more commonly diagnosed among young children, whereas generalized anxiety disorder (GAD), obsessive – compulsive disorder (OCD), and panic disorder are more likely to be diagnosed in late childhood or adolescence (DSM-V; American Psychiatric Association, 2013; Westenberg, Siebelink, Warmenhoven, & Treffers, et al., 1999).

Over time, anxiety disorders diagnosed in childhood and adolescence are often associated with difficulties during adulthood, including psychiatric hospitalizations along with symptoms of anxiety and depression (Biederman et al., 2005; Goodwin, Fergusson, & Horwood, 2004; Ferdinand & Verhuist, 1995; Kendall, Kortlander, Chansky, & Brady, 1992; Pine, Cohen, Gurley, Brook, & Ma, 1998; Woodward & Fergusson, 2001). In a 21-year longitudinal study, researchers found that adolescents with a primary anxiety disorder diagnosis were at later risk for illicit drug use, failure to attend a university, and other anxiety and major depressive disorders (Woodward & Fergusson, 2001). Furthermore, the long-term sequelae of anxiety disorders result in a national economic load due to medical costs and loss of productivity (Martin & Leslie, 2003).

**Anxiety Disorders and Functional Impairment**

Anxiety disorders diagnosed in children and adolescents are associated with functional impairments in multiple domains (Langley, Bergman, McCracken, & Piacentini, 2004). According to the DSM-V (American Psychiatric Association, 2013), functional impairments are defined as significant distress experienced in social, occupational, familial, and/or any other important domains in the individual’s life. For example, research indicates that up to 60% of children diagnosed with OCD experience severe global impairments (Markarian et al., 2010;
Whitaker et al., 1990). Further, children diagnosed with an anxiety disorder are rated as less liked and are socially neglected by peers (Barrow, Baker, & Hudson, 2011), and experience increased rates of behavioral problems (Manassis, Avery, Butalia, & Mendlowitz, 2004; Manassis & Hood, 1998). Functional impairments have the potential to negatively influence critical developmental periods in a child’s later life (Beidel, Turner, & Morris, 1999; Kearney, 2001; Silverman & Ginsburg, 1998), such as dating and employment. Finally, the impairments associated with anxiety disorders may be further compounded when a child or adolescent has a comorbid diagnosis (Kendall, Kortlander, Chansky, & Brady, 1992).

**Academic Functioning.** Academic performance is a functional domain that has consistently been linked to anxiety disorder symptoms (e.g., fear, worry, and avoidance) (Ialongo et al., 1995; Kashani & Orvaschel, 1990). For example, refusal to attend school may occur in children diagnosed with SAD because of anxiety experienced when separating from a caregiver (Bell-Dolan & Brazeal, 1993). Youth with social phobia also present with school refusal in an effort to avoid potentially embarrassing social situations (Kearney, 2001). This is also evident among youth with specific phobia who will avoid school due to school-related fear stimuli, such as fire drills (Albano, Chorpita, & Barlow, 2006). Furthermore, youth with GAD may experience excessive fear of negative peer and teacher evaluation based on school performance; this can lead them to avoid age appropriate school events or inhibit their ability to complete school tasks (Kendall, Pimentel, Rynn, Angelosante, & Webb, 2004). More generally, anxiety disorders are also associated with school dropout in adolescents (Kessler et al., 1995; Vander, Stoep, Weiss, McKnight, Beresford, & Cohen, 2002). Therefore, the nature of anxiety disorders
can result in poor school performance as a result of school avoidance as well as worry related to academic performance.

**Social Functioning.** Additionally, social development is negatively impacted by anxiety disorders (Benjamin, Costello, & Warren, 1990; Kendall & Chansky, 1997; Essau et al., 2000). Clinically anxious youth are reported to have fewer friends and less positive social interactions, and can be targets of peer victimization (Crawford & Manassis, 2011; Pedersen, Vitaro, Barker, & Borge, 2007; Spence, Donovan, & Brechman-Toussaint, 1999). According to a meta-analysis, peer victimization is significantly related to both generalized and social anxiety (Hawker & Boulton, 2000). Further, peer victims are three times more likely than non-victims to have an anxiety disorder (Kumpulainen, Rasanen, & Puura, 2001). Notably, feelings of loneliness, friendship quality, and social efficacy have been identified as treatment outcome predictors for children with anxiety disorders, with children who report higher quality peer relationships showing better treatment response (Alfano, Pina, Villalta, Beidel, Ammerman, & Crosby, 2009; Barrow, Baker & Hudson, 2011).

Restricted social interactions can be a direct or indirect function of the anxiety disorder. For instance, as a direct result of the fear of separating from parents or significant caregivers, children with SAD are less likely to remain in clubs or sports, particularly if their parents are not actively involved (Last, 1989). In other anxiety disorders, limited social interactions are an indirect consequence of the anxiety symptoms. For example, while the obsessions and compulsions related to a child’s OCD may not be specifically related to social interactions, children with OCD may be less inclined to invite peers for sleepovers or attend sleepovers due to
negative emotions linked to complex rituals associated with bedtime (Albano, Chorpita, & Barlow, 2006). As a result of limiting their participation in social activities, the child is restricting his/her opportunities for social engagement, development of appropriate social skills, and association of positive feelings with social situations (Kearney, 2001). Ultimately, youth with an anxiety disorder can become delayed in meeting age-specific developmental milestones, including dating and employment. Therefore, early interventions may be of considerable importance given the significant academic and social functional impairments associated with anxiety disorders, particularly because long-term consequences can project into adulthood in the absence of appropriate intervention (Klein & Pine, 2002).

Cognitive and Gene-Environmental Factors Contributing to Anxiety Disorders and Related Impairments. To better understand the complex bidirectional relationship between social development and anxiety disorders, cognitive and gene-environmental factors have been investigated (La Greca & Harrison, 2005; Oh, Rubin, Bowker, Booth-LaForce, Rose-Krasnor, & Laursen, 2008). A cognitive model of social anxiety has been proposed to better explain the association between anxiety disorders and social avoidance (Chorpita, Albano, & Barlow, 2006). The cognitive model indicates that clinically anxious children experience an increased preoccupation with threat (e.g., anticipation of a negative social outcome or interaction) (Chorpita, Albano, & Barlow, 2006; Pine, Klein, Roberson-Nay, Mannuzza, Moulton, Woldehawariat, & Guardino, 2005). The maladaptive preoccupation with threat appraisal limits the child's ability to attend and respond to social cues appropriately, which may contribute to an unsuccessful peer interaction and lead to the inaccurate conclusion that social interactions or
activities need to be avoided or limited. Avoidance behavior is maintained through negative reinforcement, as it serves to reduce aversive feelings of anxiety (Bouton, Mineka, & Barlow, 2001). Clinically anxious children tend to avoid social interactions to varying degrees regardless of disorder type, and this can result in limited opportunity for social skills development and fewer meaningful interpersonal relationships (Rubin & Burgess, 2001). For example, children with social phobia attend fewer social activities (Beidel, Turner, & Morris, 1999), and report having fewer friends and greater feelings of loneliness than their non-social-phobic peers (Beidel, Turner, & Morris, 1999; La Greca, 2001).

Research has focused on understanding the heritability of anxiety from two perspectives (Gregory & Eley, 2007): 1) twin and adoption studies (Biederman, Rosenbaum, Bolduc, Faraone, & Hirschfield, 1991; Hettema, Prescott, Myers, Neale, & Kendler, 2005), and 2) longitudinal investigations on the persistence of anxiety disorders over the life course (Pine, Cohen, Gurley, Brooke, & Ma, 1998). In recent years, research has focused on identifying specific genes that contribute to the development of anxiety disorders. Findings for the specificity of a one-to-one gene-anxiety relationship are mixed (Schmidt, Fox, Rubin, Hu, & Hamer, 2002). Nevertheless, variations of the 5-HTT (serotonin transporter protein) allele have been found to be associated with shyness (Arbelle, Benjamin, Golin, Kremer, Belmaker, & Ebstein, 2003; Battaglia et al., 2005). Furthermore, gene studies have begun to include the examination of environmental factors in an effort to better understand the gene-environment interaction (Eley, Bolton, O’Connor, Perrin, Smith, & Plomin, 2003; Moffitt, Caspi, & Rutter, 2005). For example, increased risk for behavioral inhibition and shyness was evident in children
with the short 5-HTT allele if their families endorsed low levels of support (Fox et al., 2005). However, further research is needed in comprehending how social functioning develops in the context of gene-environment interaction predicting an anxiety disorder (Kendler, Kuhn, Vittum, Prescott, & Riley, 2005).

**Sleep Problems and Functional Impairments in Non-Psychiatric Youth**

Sleep disturbances have been linked to similar types of functional impairments as those seen in anxiety disorders alone. These include problems in academic performance, along with behavioral, social and family functioning (Sadeh, Gruber, & Raviv, 2002; Sadeh, Raviv, & Gruber, 2000; Smaldone, Honig, & Byrne, 2007; Wolfson & Carskadon, 2003). Sleep is a vital and active process that helps regulate various physiological and behavioral functions through cues from the circadian clock (Dunlap, 1999). Specifically, circadian rhythms regulate sleep patterns through changes in sleep-wake cycles and the release of hormones like melatonin. Typically, individuals are most alert in the mid-morning and evening, while least alert from 3pm to 5pm and from 3am to 5am (Mindell & Owens, 2009). However, individual differences are present. For example, some individuals who are referred to as “night owls” are more likely to report better functioning in the evening, whereas “larks” are more likely to report better functioning in the early morning and to report tiring early in the evening (Jenni & Carskadon, 2005; Mindell & Owens, 2009). The sleep-wake cycle is also affected by developmental factors (Beebe, 2008), such as delayed sleep onset during adolescence (Wolfson & Carskadon, 2003).

According to the International Classification of Sleep Disorders (ICSD – 2), childhood sleep disorders are classified into two categories. Dyssomnias include difficulties initiating and
maintaining sleep (e.g., insomnia), and parasomnias include difficulties with arousal and sleep stage transitions (e.g., sleep walking). One of the most commonly diagnosed sleep problems experienced by children and adolescents during their school-age years is pediatric insomnia, with prevalence rates ranging from 25% to 40% (Meltzer & Mindell, 2006; Owens, Spirito, McGuinn, & Nobile, 2000). Pediatric insomnia is defined as repeated difficulties in sleep initiation, duration, and quality of sleep, resulting in daytime and family functional impairment (ICSD – 2, 2005). Up to 50% of youth diagnosed with pediatric insomnia have a comorbid psychiatric disorder (Ivanenko et al., 2004). The ICSD – 2 identifies another relevant diagnostic category, Behavioral Insomnia of Childhood (BIC). BIC includes insomnias that are specific to childhood and due to extrinsic factors, such as parental presence and bedtime resistance commonly associated with SAD.

Research indicates that children and adolescents are increasingly sleeping fewer than the recommended 10 to 11 hours per night (National Sleep Foundation, 2004). Without sufficient sleep, children experience impairments across multiple domains. Specifically, sleep-deprived children have difficulty sustaining attention during school hours, produce less verbal creative output, and have poorer abstract reasoning skills (Crabtree & Witcher, 2008; Sadeh, Raviv, & Gruber, 2000; Fallone, Acebo, & Seifer, 2005; Randazzo, Muelbach, & Schweitzer, 1998). The negative consequences of insufficient sleep on children and adolescents’ development have been documented worldwide (Bharti, Malhi, & Kashyap, 2006; Ottaviano, Giannotti, Cortesi, Bruni, & Ottaviano, 1996; Smedje, Broman, & Hetta, 1998). Particularly, an extensive amount of research has focused on the impact of sleep deprivation on the cognitive functioning of healthy
youth. Adequate sleep is essential to learning, specifically in the promotion of memory encoding and consolidation (Stickgold, 2005). Youth who report inadequate sleep are more likely to experience reduced attention, poor academic achievement, and school failure (Sadeh, Raviv, & Gruber, 2000; Wolfson & Carskadon, 2003). A landmark community investigation (School Start Time Study) examined implications of a district-wide delay in high school start times (Wahlstrom, 2002a, 2002b), designed to allow youth to sleep later. In a sample of over 18,000 students, the study found increased student attendance, reduced tardiness, increased graduation rates, and increased overall academic performance.

Sleep has also been associated with emotion regulation (Fallone, Owens, & Deane, 2002; Beebe et al., 2008; Sadeh, et al, 2002). For example, increased oppositionality and irritability have been reported by adolescents whose sleep was restricted to 6.5 hours for five days (Beebe et al., 2008). Conversely, another study found that improved sleep quality was associated with increased positive self-image and better control of aggression in a sample of children and adolescents ages 9 to 14 (Meijer, Habekothe, & van den Wittenboer, 2000).

The comorbidity between psychiatric disorders and pediatric insomnia often makes it difficult for clinicians to differentiate between depressive symptoms and the normative effects associated with delayed circadian rhythms during adolescence (Dahl & Lewin, 2002). Puberty is associated with changes in the sleep patterns of adolescents (Carskadon, 1999). Specifically, adolescents experience a sleep phase delay, which refers to a shift in the sleep cycle towards sleeping and waking at a later time (Wolfson & Carskadon, 1998). The sleep phase delay conflicts with early school start times leading to a sleep debit that is often recuperated over the
weekend. As such, adolescents sleep on average an hour and fifty minutes more over the weekend in comparison to the weekday (Wolfson & Carskadon, 1998). A persistent irregular sleep schedule can result in poor sleep quality, which has been found to be associated with weaker school performance (Wolfson & Carskadon, 1998), increased use of stimulants (Carskadon, 1990), and decreased mood (Wolfson & Carskadon, 1998). Dahl (1999) proposed an intricate cycle between emotional difficulties and sleep difficulties, where emotional difficulties lead to fragmented and diminished sleep, which furthers the dysregulation of emotions. Although the precise sleep mechanisms involved in emotion dysregulation continue to be explored, the effects of sleep deprivation in relation to emotion dysregulation appear to be consistent (Dahl, 1999; Wolfson & Carskadon, 1998).

In summary, sleep is an essential process; when disrupted, the effects can severely influence the quality of a child’s life. Many of the studies that have examined the complex relationship between sleep restriction and cognitive, behavioral, and emotional problems among children and adolescents have been naturalistic studies (Dahl, 1999; Crabtree & Witcher, 2008). However, more empirical research studies have emerged in recent years supporting a causal association between sleep restriction and cognition, behavioral, and emotional regulation (Crabtree & Witcher, 2008).

**Sleep Problems and Functional Impairments in Clinically Anxious Youth**

Youth diagnosed with a primary anxiety disorder report functional impairments in academic performance, behavioral, and social functioning. Similarly, children who report sleep disturbances without a psychiatric diagnosis experience functional impairments in those same
domains. The compounding effect of sleep disturbances on psychiatric disorders has been closely studied in adults (Mittler, Dinges, & Dement, 1994). A study found that individuals experiencing both insomnia and anxiety disorders reported poorer sleep quality and experienced lower levels of mental health quality of life in comparison to adults with anxiety disorders alone (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009). Among the various anxiety disorders examined, the comorbidity of an anxiety disorder and insomnia had an additive effect on role limitations in participants diagnosed with OCD. To date, the additive effects of sleep disturbances in youth with an anxiety disorder have been minimally studied.

Researchers have examined the relationship between sleep disturbances and childhood psychiatric disorders, particularly depression and ADHD (Alfano & Gamble, 2009). For example, shortened rapid eye movement (REM) sleep has been found in depressed persons in comparison to healthy controls (Emslie, Rush, Weinberg, Rintelmann, & Roffwarg, 1990; Dahl, Ryan, Birmaher et al., 1991). Self-report of hypersomnia and insomnia in depressed youth has also been associated with more severe depressive symptoms, longer psychiatric illness, and more comorbid anxiety disorders in comparison to depressed youth who only endorsed insomnia or hypersomnia alone (Liu et al., 2007). Similarly, per parent report, children with ADHD experience delayed sleep onset, increased movement during sleep, and bedtime resistance (Accardo, Marcus, Leonard, Shults, Meltzer, & Elia, 2012). The relationship between sleep problems and childhood psychiatric disorders continues to be explored, as discrepant findings are evident between subjective and objective measures (Accardo et al., 2012).
Specifically related to anxiety, Leahy and Gradisar (2012) reviewed studies examining the bidirectional relationship between anxiety and sleep, and found that sleep problems among youth tend to predict later development of anxiety. However, they acknowledge that there is limited research examining whether anxiety predicts sleep problems (Leahy & Gradisar, 2012). For example, several longitudinal investigations indicate that chronic sleep problems are significant predictors of the development and maintenance of anxiety disorders (Gregory & O’Connor, 2002; Gregory, Eley, O’Connor, & Plomin, 2004; Gregory, et al., 2005). Leahy and Gradisar (2012) recommend that future research include experimental and treatment designs to build on existing longitudinal studies (as minimal studies have examined the opposite relationship).

Factors Related to the Understanding of Sleep and Anxiety Disorders. The exact mechanism behind the relationship between anxiety disorders and sleep problems in youth remains unclear. However, a few factors have been identified that may be associated with this relationship. In recent years, many researchers have shifted focus to the role of genes in psychiatric disorders. Interestingly, the serotonin transporter gene (5HTTLPR), which is implicated in anxiety disorders, is also involved in sleep disturbances (Barclay, Eley, Mill, Wong, Zavos, Archer, & Gregory, 2011; Brummett et al., 2007). While the precise genes involved in these mechanisms are still being investigated, twin studies have found that an overlap in genes accounts for 74% of the variance in the relationship between sleep difficulties and anxiety symptoms (Gregory et al., 2011).
Similar to genetic components, neurobiological systems are involved in the sleep and anxiety relationship. A number of brain regions are associated with both processes, including the prefrontal cortex (PFC), thalamus, limbic midbrain, pons, and medulla oblongata (Dahl, 1996). For example, the PFC, an area associated with executive functioning, has been implicated in numerous emotional disorders, including anxiety (e.g., Bishop, Duncan, Brett, & Lawrence, 2004). Studies examining sleep deprivation have found deactivation in the same region (e.g., Harrison & Horne, 2000; Thomas et al., 2000). The pons is another region that is involved in both sleep and emotional regulation (Aston-Jones, Rajkowski, Kubiak, & Alexinsky, 1994; Curtis & Valenti, 1994; Kitayama, Kayahara, Nakano, Murase, Otani, & Nomura, 1997; Osaka & Matsumura, 1994). Specifically, the physiological arousal experienced in anxiety disorders and REM sleep are both attributed to the pons (Kitayama, Kayahara, Nakano, Murase, Otani, & Nomura, 1997). Investigations examining the impact of neurobiological systems on the complex relationship of sleep and anxiety disorders are continuing to emerge, and more research needs to be conducted to better understand this relationship.

Another potential mechanism relates to hyperarousal and hypervigilance, key characteristics of anxiety disorders that may prevent the relaxed state necessary for onset and maintenance of sleep (Dahl & Lewin, 2002). Considerable research has examined the function of the hypothalamic-pituitary-adrenal (HPA) axis, a neurophysiological system involved in the control and response to stress, in psychiatric disorders and sleep in adults (e.g., Lattova et al., 2011; O’Keane, Frodl, & Dinan, 2012). Cortisol is a stress hormone that is secreted by the adrenal gland and serves as an index of the HPA function, where novel, stressful, or anxiety-
arousing tasks are associated with increased cortisol levels (McBurnett et al., 1991). Further, high levels of basal cortisol have been consistently found in depressed adults (Young, Carlson, & Brown, 2001). However, only two studies have systematically examined the relationship between sleep and the HPA axis in youth with anxiety disorders. Forbes and colleagues (2006) found that children with an anxiety disorder had increased cortisol levels prior to sleep onset in comparison to depressed adolescents and non-psychiatric control adolescents. They hypothesized that the difference in cortisol levels between anxious children and adolescents prior to sleep onset may be attributable to adaptation to chronic stress over time. Whereas another study that examined cortisol levels in children diagnosed with GAD, found no significant differences in comparison to healthy controls (Alfano, Reynolds, Scott, Dahl, & Mellman, 2013). However, further study is needed in this area of research.

Cognitive factors pertaining to sleep and anxiety disorders have also been identified. Adults with anxiety disorders commonly report greater worry prior to sleep onset, thus delaying sleep onset (Belanger, Morin, Gendron, & Blais, 2005). A few community-based studies have additionally found a relationship between sleep problems and pre-sleep worry or fear among children experiencing anxiety symptoms (Alfano, Zakem, Costa, Taylor, & Weems, 2009; Gregory & Eley, 2005; Gregory, Eley, O’Connor, & Plomin, 2004). To date, only one known study has examined the relationship between sleep and cognitive factors in a sample of clinically anxious children (Alfano, Pina, Zerr, & Villalta, 2010). This study found that pre-sleep arousal examining worries were related to decreased total sleep and greater sleep problems, based on both parent and child sleep reports (Alfano, Pina, Zerr, & Villalta, 2010).
In turn, environmental and familial factors have been found to account for up to 30% of the variance in the relationship between anxiety disorders and sleep problems (Gregory, Eley, O’Connor, & Plomin, 2004). Some researchers suggest a role of parental psychopathology and/or permissive parenting style (Meijer, Habekotie, Van den Wittenboer, 2001; Owens-Stively et al., 1997). Specifically, a permissive parenting style may impede the use of appropriate structure during bedtime routines, which may further exacerbate anxiety symptoms in children (Graziano & Mooney, 1980; King, Cranstoun & Josephs, 1989). For example, anxious children often prefer not to sleep alone; parents who have difficulty setting limits may allow the child to fall asleep in their bed upon becoming frustrated with managing bedtime resistance. In some instances, they will return the children to their own bed once asleep (Hudson, Gradisar, Gamble, Schniering, & Rebelo, 2009; Muris, Merckelbach, Ollendick, King, & Bogie, 2001). Over time, children learn to associate safety and tranquility with their parents’ bed, and are given limited opportunities to habituate to the initial arousal state experienced prior to falling asleep in their own bed. Additionally, familial stress is associated with youth sleep problems (Liu, Uchiyama, Okawa, & Kurita, 2000). For example, maternal report of stressful life events has been found to be associated with poorer bedtime routines (Bates, Viken, Alexander, Beyers, & Stockton, 2002). Furthermore, restless sleep and sleepiness are related to marital conflict on both subjective and objective measures of sleep (El-Sheikh, Buckhalt, Mize, & Acebo, 2006).

**Subjective Measures of Sleep Problems and Anxiety Disorders.** The use of parent- or child-report measures in the examination of sleep functioning in relation to psychiatric disorders are defined as subjective measures. Based on parent-report measures, over 85% of children and
adolescents with an anxiety disorder have clinically significant sleep problems (Alfano, Ginsburg, & Kingery, 2007; Alfano, Pina, Zerr, & Villalta, 2010). The sleep problems identified in children include difficulties with sleep onset delay and maintenance, behavioral symptoms related to bedtime resistance and limit-setting, and various parasomnias such as nightmares, sleep walking and talking, bruxism, and enuresis (Alfano, Beidel, Turner, & Lewin, 2006; Alfano, Ginsburg, & Kingery, 2007; Gregory & Eley, 2005; Ivanenko, Crabtree, & Gozal, 2006).

Hudson and colleagues (2009) found that clinically anxious children reported significantly lower total sleep time on school nights in comparison to non-anxious children. However, the clinically anxious group did not differ from the non-anxious group on sleep-onset latency (i.e., time spent awake in bed prior to falling asleep) and wake after sleep onset (i.e., number of times waking up after falling asleep). Hudson and colleagues (2009), found sleep disturbances were evident for the anxious youth only on school nights, and not during the weekend. Additionally, studies have determined per parent and child report that GAD and SAD are more strongly associated with sleep difficulties than other anxiety subtypes (Alfano, Beidel, Turner, & Lewin, 2006; Alfano, Pina, Zerr, & Villalta, 2010), with moderate correlations found for children with panic disorder, Social Phobia, OCD, and PTSD (Alfano et al., 2007).

Furthermore, one study examined sleep-related problems in clinically anxious youth following treatment with fluvoxamine (Alfano et al., 2007). The most commonly reported sleep problems were insomnia, nightmares, and refusal to sleep alone. They also found that clinically anxious children who were randomized to fluvoxamine demonstrated significantly greater improvements in sleep problems than the control group (Alfano et al., 2007).
Objective Measures of Sleep Problems and Anxiety Disorders. Researchers have also used objective measures, which are defined as physiological measures (e.g., wrist actigraphy and polysomnography [PSG]) to examine sleep-wake cycles or bodily movements in relations to psychiatric disorders in youth. Although some findings are mixed, the sleep problems recorded with physiological measures generally appear similar to those indicated in behavioral self-report measures. For instance, Rapoport and colleagues (1981) used PSG to examine sleep in adolescents with OCD, and found that the OCD group presented with poor sleep efficiency and increased sleep latency in comparison to matched healthy controls. Studies using wrist actigraphy (noninvasive devices worn on the wrist that estimate sleep patterns based on the analysis of motion data) have yielded similar findings (Gold, Teicher, Hartman, & Harakal, 1997; Sadeh, Hauri, Kripke, & Lavie, 1995).

Studies that are more recent have begun to combine both subjective and objective measures in the examination of the relationship between sleep problem and anxiety. Forbes and colleagues (2008) examined this relationship among three groups: a clinically anxious group, a clinically depressed group without comorbid anxiety, and a healthy control group. PSG data showed that anxious youth presented with a greater number of arousals compared to depressed youth, less slow wave sleep, and greater sleep onset latency compared to depressed and control groups (Forbes et al., 2008). When considering subjective data, the anxious group reported greater sleep latency only on the second and final night of the study. Thus, anxious youth may have limited awareness of and/or tend to underreport their experience of sleep problems (Forbes et al., 2008).
Alfano and Kim (2011) collected child and parent reports of sleep disturbances and actigraphy data from six healthy controls and six children (ages 7 to 11) diagnosed with OCD (non-depressed). The findings indicate that the children with OCD experienced significantly fragmented sleep patterns, decreased total sleep time, and longer wake periods after falling asleep. Another study examined sleep disturbances in fifteen children (ages 7 to 11) diagnosed with GAD (non-depressed) compared to fifteen matched healthy controls using PSG, salivary cortisol levels, and behavioral rating scales (Alfano, Reynolds, Scott, Dahl, & Mellman, 2013). Children with GAD demonstrated significantly increased sleep onset latency and reduced latency to REM, but they did not differ from controls on cortisol levels.

**Methodological Limitations of Sleep Problems and Anxiety Disorders.** Compared to the progress established investigating sleep disturbances in other childhood psychiatric disorders, the examination of sleep disturbances in clinically anxious youth is in its early stages. Further, there are methodological limitations in existing studies examining this relationship (Forbes et al., 2008; Hansen, Skirbekk, Oerbeck, Richter, Kristensen, 2011). When considering the generalizability of the current research findings to clinically anxious youth, it is important to take note of factors such as the type and quality of the sleep measure administered (e.g., subjective, objective, or a combination), whether community or clinically anxious samples were used, whether comorbid diagnoses were considered, and the appropriate use of control groups. These factors will be discussed in further detail below.

First, there is a lack of agreement on how to define and measure sleep problems when assessing their relationship with anxiety disorders (Gregory & Sadeh, 2012). Sleep diaries are
thought to be the most reliable of the various self-report measures since they include daily recordings from the subject and relevant caregivers over the course of multiple days (Gregory et al., 2011). Despite this recommendation, studies vary in the type of self-reported sleep measures used; with some studies using standardized sleep measures and others using unstandardized sleep measures. As such, discrepancies are evident when examining the self-report of sleep problems in anxious youth. Most studies identify sleep onset latency as a problem when examined solely by self-report measures among anxious youth (e.g., Alfano, Beidel, Turner, & Lewin, 2006; Gregory & Eley, 2005). However, a study that used a comparison group found no significant differences between clinically anxious youth and healthy controls in their self-report of sleep latency (Hudson et al., 2009). On the other hand, objective sleep measures appear to report a consistent difficulty with sleep onset latency as measured by PSG and wrist actigraphy (Alfano & Kim, 2011; Forbes et al., 2008). These findings highlight the discrepancy that exists between subjective and objective report of sleep problems in anxious youth. These discrepancies indicate youth may have limited awareness of and/or tend to underreport their experience of sleep problems (Forbes et al., 2008). Therefore, it is best to include objective measures in addition to a comprehensive self-report.

Second, the existing research base is founded on community samples and anxiety symptom severity scales, as opposed to clinical populations assessed and those diagnosed with the use of a structured diagnostic interview (Forbes et al., 2008; Leahy & Gradisar, 2012). Community-based studies suggest an association between sleep problems and anxiety symptom severity in youth (e.g., Nielson et al., 2000; Zavos, Rijsdijk, Gregory, & Eley, 2010). However,
community studies are limited in their lack of generalizability to clinical samples and lack of measurement precision (e.g., Gregory, Eley, O’Connor, Rijsdijk, & Plomin, 2005). Structured diagnostic interviews increase reliability and validity, thereby ensuring accuracy of diagnoses based on strict diagnostic criteria established by the DSM-V (American Psychiatric Association, 2013; Frick, Barry, Kamphaus, 2010). The use of rating scales alone in non-clinical samples suggests an association between anxiety symptom severity and sleep problems; however, this line of research does not necessarily translate to a relationship between clinical anxiety disorders and sleep problems. While non-clinical research can inform our understanding of clinical phenomena, these methods are limited in their ability to parse the neurobiological and environmental factors that are unique to the development, maintenance, and treatment of anxiety disorders.

Third, few studies have controlled for comorbid disorders, particularly depression. Yet, sleep difficulties have been identified as one of the biological markers of Major Depressive Disorder (MDD) and is also one of the diagnostic symptoms in the DSM-V for MDD (American Psychiatric Association, 2013). The correlation between sleep problems and depression is strong in both adults and children (Ivanenko & Johnson, 2008). The sleep disturbances experienced in MDD have been found to be significantly impairing and linked with an increased likelihood to attempt suicide (Ivanenko & Johnson, 2008). A study of 553 children diagnosed with MDD between the ages of 7 and 14 found that 73 percent of the sample reported sleep problems (Liu et al., 2007). Additionally, those who reported the most sleep problems tended to have severe depressive symptoms and increased rates of comorbid anxiety (Liu et al., 2007). Studies also
indicate that 13 to 27 percent of parents of depressed children between 4 and 12 years of age report sleep difficulties in their children, such as bedtime resistance, delayed sleep onset, co-sleeping, snoring, enuresis, nighttime awakenings, nightmares, night terrors, and sleep walking (e.g., Blader, Koplewicz, Abikoff, & Foley, 1997; Smedje, Broman, & Hetta, 1998; Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001). Given comorbidity rates between anxiety and depression as high as 70 percent among youth (Angold, Costello, & Erkanli, 1999; Kovacs & Devlin, 1998), controlling for comorbid depression in the investigation of anxiety disorders and sleep difficulties is extremely important. Not doing so may potentially obscure research findings when trying to elucidate the relationship between sleep problems and anxiety disorders (Forbes et al., 2008).

Fourth, the use of comparison groups can help account for potential confounding variables and strengthen our confidence in the findings, particularly when all subjects are assessed in the same environment under the same conditions. Hudson and colleagues (2009) highlight the importance of using comparison groups when examining the relationship between sleep disturbances and anxiety disorders. As previously discussed, they found that clinically anxious youth did not differ on time spent awake prior to falling asleep or the number of times waking up after falling asleep in comparison to healthy controls. Further, they also noted the importance of distinguishing sleep patterns during the weekday versus the weekend, as anxious youth only reported sleep problems during the weekday.

In summary, the number of studies that examine the relationship between sleep and anxiety in clinically anxious youth incorporating healthy controls, subject and objective
measures, and control for comorbid depressive disorder are few (Alfano & Kim, 2011; Alfano et al., 2006; Alfano et al., 2007; Alfano et al., 2010; Forbes et al., 2006; Forbes et al., 2008; Hudson et al., 2009; Johnson et al., 2006). However, these studies have found a consistent relationship between anxiety disorders, difficulties with sleep onset delay, and disturbances in the sleep-wake cycle when compared to healthy controls. As is evident, the relationship between sleep and anxiety disorders in youth is multifaceted and complex. As studies continue to improve their methodologies, we will be better able to comprehend the intricacies of this relationship and how it presents not only in clinically anxious youth in general, but also how it may vary by anxiety disorder (i.e., GAD vs. Social Phobia). Additionally, we will be able to examine further the bidirectional relationship between anxiety and sleep, and determine whether the combination of symptoms is associated with additive functional impairments in comparison to youth in the general community. Ultimately, these future findings have the potential to inform targeted treatment interventions in clinically anxious youth.

The Current Study

In his proposal of a cyclical relationship between sleep and emotion dysregulation, Dahl (1999) highlights the importance of evaluating the contribution of sleep deprivation to psychiatric disorders among adolescents. He points out that the complex relationship between sleep and psychiatric disorders has been poorly understood and under examined. Since Dahl’s (1999) proposal, a few studies have lent support to the bidirectional relationship between anxiety and sleep problems, but have not examined the potential compounding effects of sleep and anxiety difficulties (Alfano & Lewin, 2008; Keller, Buckhalt, & El-Sheikh, 2008). The present
study aims to address this limitation and contribute to this emerging literature investigating the association of sleep deprivation and a psychiatric disorder, primarily anxiety disorders among youth.

As indicated, impairments associated with sleep problems in youth without a psychiatric disorder appear similar to those experienced by youth with an anxiety disorder and no sleep problems (Crabtree & Witcher, 2008; Sadeh, Gruber, & Raviv, 2002). However, the additional impairment that may be attributed to sleep problems over and above that attributed to anxiety has been minimally examined in comparison to healthy controls. While there is some evidence indicating that sleep problems are more common in anxious children than in children without a psychiatric disorder, there is insufficient research to address whether the additional impairment associated with sleep problems in anxious children differs in degree from the impairment associated with sleep problems in non-psychiatric controls. Further clarification of this relationship can be used to minimize the development and maintenance of anxiety disorders, and impairments associated with comorbid anxiety and sleep problems, and ultimately inform the assessment and treatment of childhood and adolescent anxiety disorders (Alfano & Lewin, 2008).

Without appropriate psychological early intervention, social functioning impairments among clinically anxious youth increase youth’s risk for potential long-term functional impairments (Klein & Pine, 2002). For example, youth with an anxiety disorder alone experience developmental delays in areas such as in dating and employment. For this reason, examining whether the experience of sleep problems in addition to an anxiety disorder contribute
to greater impairments in social functioning is of particular importance and is a specific focus of this study.

Therefore, this study aims to evaluate the association between sleep functioning and social functioning in a clinical sample of youth with an anxiety disorder compared to children with no anxiety disorder. The specific research questions are:

1. Do sleep functioning and social functioning differ by group?
2. How well do sleep functioning and anxiety symptom severity predict social functioning when controlling for age?
3. Does inadequate sleep predict youth social functional impairment over and above anxiety symptom severity alone?

To address the limitations in previous investigations, this study examined sleep problems in clinically anxious children (established through a structured clinical interview) in comparison to healthy controls in the community. Additionally, in an effort to control for the high comorbidity between sleep disturbances and depression, youth who met criteria for a comorbid depressive disorder were excluded.

**Hypotheses**

The following hypotheses were evaluated to examine the relationship between sleep functioning and social functioning in children with and without an anxiety disorder:

1. Children with an anxiety disorder will report significantly greater sleep inadequacies and social functional impairment compared to the healthy control group.
2. Child social functional impairment will be better predicted by both sleep difficulty and anxiety symptom severity than by either sleep difficulty or anxiety symptom severity alone.

3. A significant interaction effect will be found between sleep adequacy and anxiety when predicting children's social functioning.
CHAPTER 2

Methods

Participants

Data were collected from an ongoing study at the National Institute of Mental Health (NIMH). Specifically, this study was conducted at the Experimental Therapeutics Branch within the Mood and Anxiety Disorders Program of the Intramural Research Program. Children with an anxiety disorder were recruited when seeking treatment at NIMH. The larger study aims to examine neurocognitive functioning of pediatric and adult mood and anxiety disorders through the use of functional magnetic resonance imaging (fMRI).

A total of approximately 1,200 children, adolescents, and adults were initially recruited into the larger study. The investigation aimed to identify individuals with an anxiety disorder and no comorbid diagnosis (60 adults, 150 youth), major depression and no comorbid diagnosis (60 youths, 60 adults), and without a psychiatric disorder (150 youths and 150 adults). Those with major depression were not included in this study. Inclusion criteria for the child anxious group required meeting full criteria for a current anxiety disorder (social phobia, separation anxiety disorder, generalized anxiety disorder, or panic disorder) and scoring greater than nine on the Pediatric Anxiety Rating Scale (PARS; RUPP, 2001). Exclusion criteria were current use of any psychoactive substances, OCD, Tourette’s syndrome, PTSD, conduct disorder, suicidal ideation, ADHD, a past or current history of mania or psychosis, a pervasive developmental disorder, or an IQ < 70. Healthy controls were recruited through advertisements and postings, and psychiatric patients were recruited into the study when seeking treatment through the Mood and Anxiety Disorders Program of the Intramural Research Program through advertisements and
referrals. Inclusion criteria for non-anxious children were an absence of medical and psychiatric illness.

The broader investigation included four phases: 1) subject recruitment, assessment, and training, 2) pre-treatment MRI scanning, 3) treatment, 4) post-treatment MRI scanning. During the first phase (visit one), the participants were screened via self-report measures, structured interviews with trained clinicians, and physical examination. For the second phase (second and third visits), participants underwent neuropsychological testing and fMRI procedures and completed the same self-report measures that were administered during visit one. Eligible participants were then randomized into either fluoxetine or placebo treatment in the third phase. In the fourth and final phase, participants were subjected to post-treatment MRI scanning and neurological testing.

Data for variables relevant to the current study were collected at visit one or at visit two. Unfortunately, subsequent visits did not systematically collect follow-up data on the key variables of interest. Measures examining demographics, anxiety symptom severity (parent and youth report), social functioning, life events stressors, family risk factors, and sleep functioning were pertinent to the present study. Sleep data was collected from 118 youth in the anxiety group and 116 healthy controls. However, the total sample size varied across measures because the life event stressors and family risk factors measures were introduced into the general investigation after recruitment had already begun and thus were not available for all participants. Furthermore, in order to examine the relationship of sleep with the other variables of interest, participants were included in the present study if sleep data were collected no more than one
month apart from other variables of interest. While the interpretability of results would have been most accurate if all measures were administered within one week of each other, this would have significantly limited the sample size. This resulted in a final sample consisting of 96 (depending on the analysis) participants, with 60 participants in the anxiety group and 36 in the healthy control group. Additional demographic information for each group is provided in the Results section.

Procedures

The Institutional Review Board at the NIMH approved the larger study. Informed consent and assent were obtained from parents and youth prior to participation. To protect confidentiality, participants were assigned numerical codes with identifying information stored separately. Data for the present study was received in a de-identified format, and only baseline assessments were examined.

Measures

*Family background/demographics:* Familial demographic information was captured through the Child Demographics Form, including gender, age, race, and household income. The Family Risk Factor Checklist - Parent (FRFC-P; Dwyer, Nicholson, & Battistutta, 2003) was also administered, which is a 48-item measure used to assess children’s potential mental health risk based on exposure to stressors and chronic adversity. In addition to the total FRFC-P total risk score, the FRFC-P has five indexes that examine adverse life events and instability (ALI), family structure and social economic status (SES), parenting practices (PAR), parental verbal conflict and mood problems (VCM), and parental antisocial and psychotic behavior (APB). The total
risk scores are classified as low risk (score = 0 to 6), medium risk (score = 7 to 12), and high risk (score = 13 or more). The score classifications for the ALI, PAR, VCM, and APB are low risk (score = 0), medium risk (score = 1 to 2), and high risk (score = 3 or more), and the score classification for the SES index is low risk (score = 0 to 2), medium risk (score = 3 to 4), and high risk (score = 5 or more). The FRFC-P demonstrates good internal consistency (Cronbach’s alpha in the .70 range), and good test re-test reliability (r = .80) (Dwyer, Nicholson, & Battistutta, 2003). The FRFC-P total score confirmed adequate construct validity when correlated with other established measures, with correlations in the .47 to .59 ranges (Dwyer, Nicholson, & Battistutta, 2003).

**Diagnostic assessment:** All children were screened for lifetime history and current psychiatric disorders using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997). The K – SADS – PL is a semistructured interview based on DSM-IV criteria (APA, 1994). It is reported to have strong test-retest reliability with correlations ranging from .77 to 1.00 and adequate validity with other well-established self-report measures (K-SADS-PL; Kaufman et al., 1997). Participants who met criteria for a current anxiety disorder(s) were assigned to the clinically anxious group.

**Anxiety severity:** Child anxiety severity was measured by parent report and child self-report with the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997, 1999). The SCARED includes 41 items each rated on a 3-point scale with an ability to differentiate clinical from sub-clinical levels of anxiety. The SCARED has five empirically
derived subscales (panic disorder/significant somatic symptoms, generalized anxiety disorder, separation anxiety disorder, social anxiety disorder, and significant school avoidance). A total score of 25 is indicative of a possible anxiety disorder and scores higher than 30 can be further examined using one or more of the empirically derived subscales, which are scored by totaling specific items that correspond with each subscale. It has demonstrated good internal consistency with coefficient alphas reported in the .74 to .89 ranges. The SCARED has also demonstrated moderate parent-child agreement, good test-retest reliability, and solid inter-class correlations in the .70 to .90 ranges (Birmaher et al., 1997, 1999). In an effort to minimize multicollinearity, a composite total score was created in which the parent and child SCARED total scores were averaged together.

Sleep: Sleep functioning was assessed by a 3-item parent report examining variables related to the sleep-wake behaviors. First, participants were asked what time of day they thought they functioned best (e.g., early morning, late morning, afternoon, late afternoon, evening, or late evening). The six categories were then collapsed into three variables for analysis (e.g., morning, afternoon, or evening). This question was used to capture patterns of alertness or sleepiness in participants, which is determined by the amount and quality of sleep and time awake since the individual last slept (Mindell & Owens, 2009). Second, the participants retrospectively reported the time they awoke, went to bed, and fell asleep in the previous three days. Distinctions between weekend and weeknights were not recorded. The average number of hours slept over the course of three days was then calculated. Finally, daytime sleepiness was assessed by asking how tired the participant felt on the day of the study on a 1 – 10 rating scale (1 = Not tired at all;
10 = Extremely tired). According to Owens (2005), daytime sleepiness has been found to be indicative of disrupted sleep. Unfortunately, objective measures of sleep were not obtained, as it was not a variable being studied as part of the larger investigation.

**Functioning related to life stressors:** The Life Events Checklist (LEC; Johnson & McCutcheon, 1980) was administered to the youth and their parents to assess the perceived impact of life changes in the last year. It is a 46-item questionnaire where an event is marked as “good” or “bad” in the last 12 months and the impact is measured on a 0 to 3-point scale (0 = no effect, 1 = some effect, 2 = moderate effect, 3 = great effect). The total score is obtained by summing both the negative and positive impact scores. The measure includes questions that are related to the individual as well as the impact of familial stressors, such as “Changing to new school” and “Increased number of arguments between parents.” The internal consistency for the LEC is .97 and test re-test reliability is .74 (Greene, Walker, Hickson, & Thompson, 1985).

**Global functioning:** The Children’s Global Assessment Scale (CGAS; Shaffer, Gould, Brasic, Ambrosini, Fisher, Bird, & Aluwahlia, 1983) and the Clinical Global Impressions – Severity (CGI-S; Guy, 1976) Scale are clinician-administered scales used to assess the child’s overall functioning in the clinically anxious group only. The CGAS assessed functional impairment at baseline on a 0 (severe impairment) to 100 (superior functioning) rating scale, where 70 is considered normal functioning. The CGAS test-retest inter class correlations were in the .69 to .95 range and in previous research it significantly correlated with other clinician rated measures of impairment with correlations ranging from .76 to .92 in normative and clinical samples (Canino, Costello, & Angold, 1999; Winters, Collett, & Myers, 2005). The CGI – S
assessed the severity of the illness on scale from 1 (normal) to 7 (extremely ill) at time of baseline. Despite being a widely used assessment tool, the limited studies that have examined the psychometric properties of the CGI – S are varied and inconclusive (Forkmann et al., 2011; Khan, Khan, Shankles, & Polissar, 2002). Forkmann et al. (2011) recommend the CGI – S be used along with other clinician-rated severity functioning measures.

**Social functioning:** The Social Responsiveness Scale (SRS; Constantino et al., 2003) was administered to assess severity and type of social functional impairment. The SRS is a 65-item parent report scale primarily used to assess autistic traits on a 4-point Likert scale. The SRS has strong reliability with Cronbach’s alpha in the .93 to .97 ranges. It was validated with parent diagnostic interviews with correlations in the .52 to .79 ranges (Constantino et al., 2003). The SRS examines symptom domains such as social awareness, social cognition, social communication, social motivation, and stereotypic behaviors. It includes questions such as: “Seems much more fidgety in social situations than when alone” and “Doesn’t recognize when others are trying to take advantage of him or her” (Constantino et al., 2003). While the SRS primarily serves as a screen and diagnostic instrument for assessing autism spectrum disorders, recent studies demonstrate an association between SRS and both internalizing and externalizing behaviors, indicating the SRS to be a useful measure of social functioning in a non-autistic sample (Aldridge, Gibbs, Schmidhofer, & Williams, 2012; Hus, Bishop, Gotham, Huerta, & Lord, 2012; Pine, Guyer, Goldwin, Towbin, & Leibenluft, 2008). The SRS raw scores are often converted to T-scores to account for gender differences observed in normative samples in clinical settings. Constantino and Gruber (2005) recommend using SRS raw scores in research
studies for comparability to early studies of the SRS. The clinical raw score cutoff is identified as 8
CHAPTER 3

Results

Preliminary Analysis

Data were entered into SPSS 15.0 for Windows. The data were screened for outliers, normality, and missing data prior to analyses. Data normality was examined for skewness, symmetry of distribution, and kurtosis. All variables fell within normal statistical limits.

To examine potential confounding variables, groups were compared on gender, ethnicity, household income, and age. The anxiety group did not differ from the healthy comparison group on gender, \( \chi^2 (1) = 0, p > .05 \), ethnicity, \( \chi^2 (2) = 1.38, p > .05 \), or household income, \( \chi^2 (6) = 10.61, p > .05 \). However, the groups differed on age, whereby participants in the healthy control group were older than the participants in the anxiety group, \( t(94) = 2.83, p < .05 \) (anxiety group: \( M = 12.02, SD = 2.49 \); healthy controls: \( M = 13.59, SD = 2.85 \)). Thus, subsequent analyses included age as a covariate.

Additionally, the primary assumptions associated with a MANCOVA were examined (hypothesis 1). A moderate correlation between dependent variables is recommended to continue with a MANCOVA (Leech, Barrett, & Morgan, 2005). Correlations are provided in Table 1. A moderate relationship was found between average number of hours slept over three days and social functioning (\( r = .24, p < .05 \)), and the number of hours slept and the sleep tiredness variable (“How tired are you right now?”, \( r = -.34, p < .05 \)). A low correlation was present between the sleep tiredness variable and social functioning, (\( r = -.03, p > .05 \)). The sleep variable examining alertness patterns (“In general, what time of day do you function best?”) was not used in the MANCOVA analysis, as it is not a continuous dependent variable. Lastly,
assumption of equality of covariance was examined by Box’s Test, \( p = .13 \), indicating that the covariance matrices are equal and the assumptions are met.

To examine predictability of social functioning from sleep functioning and anxiety symptoms severity (hypothesis 2) and interaction effects of sleep functioning and anxiety severity (hypothesis 3), continuous predictor variables were centered and the SCARED parent and youth report total scores were averaged to minimize multicollinearity and increase interpretability of results.

**Descriptive Analysis**

The total sample size consisted of 96 participants, with 60 participants in the anxiety group and 36 in the healthy control group. The total sample size varied depending on the analysis from 32 to 96 because certain measures (e.g., LES and FRFC-P) were introduced after recruitment had already begun. Thus, the LES and FRFC-P were not available for all participants. Detailed demographics information for the anxiety group and healthy volunteer group is provided in Table 2. Overall, there were more female participants (58.3%) across both groups. As previously presented, the participants’ mean ages were 12-years-old in the anxiety group and 13.6-years-old in the healthy control group. The majority of the sample was Caucasian (non-Latino) across both groups (anxiety group = 86.7%; healthy control group = 83.3%). In the anxiety group, the majority of households had an income between $90,000-$179,999. In the healthy control group, the majority of households had an income between $60,000-$89,999. As noted earlier, no significant differences were found for mean household income.

The average score on the SCARED was 27.82 for the parent report and 31.11 for the youth report in the anxiety group. The average scores on the SCARED for the healthy control group
were 7.03 for the parent report and 11.97 for the youth report. A score above 25 is indicative of a possible anxiety disorder (SCARED; Birmaher et al., 1997, 1999). When examining global functional impairment in the anxiety group, the mean CGI score was 4.2, indicating that the majority of the sample was “moderately ill.” The mean CGAS score was 50.4, which denotes moderate to serious functional impairment. See Table 3 for means of all relevant variables with the exception of the sleep and social functioning variables that are presented in the following section.

Overall, the mean impact score for the total life event stressors reported by the anxiety group was 8.7, and the healthy control group mean was 5.7. When examining positive life events, the anxiety group mean score was 3.8 and the healthy control mean score was 3.5. Regarding negative life events, the anxiety group reported a mean score of 4.9 in comparison to the healthy control mean of 2.2.

When examining risk factors or stressors associated with the family environment, the FRFC-P mean score for the anxiety group was 7.6. This mean score identifies the anxiety group to be at medium risk for mental health difficulties related to familial stressors. The mean for the healthy control group was 2.6, which indicates the healthy controls are at low to medium risk.

**Hypothesis 1: Relationship of Anxiety to Sleep Functioning and Social Functioning:**
Children with anxiety disorders will report significantly greater sleep inadequacies and social functional impairment in comparison to the healthy control group.

To examine whether sleep functioning (average hours slept over three days, and tiredness) and social functioning (as measured by the SRS total score) significantly differed by group (anxiety vs. healthy control), multivariate analysis of covariance (MANCOVA) was conducted
with the two sleep functioning variables and social functioning as dependent variables. When controlling for age, there was a significant effect of group (anxiety group vs. healthy controls) on sleep functioning (hours slept and tiredness) and social functioning, Wilk’s Λ = .56, F (3, 90) = 24.08, p < .001 (see Table 4). The multivariate η² Wilk’s Λ was solid, .45. To further examine this relationship, the MANCOVA analysis was followed by univariate analysis of covariance (ANCOVA), shown in Table 5. When controlling for age, the average number of hours slept did not significantly differ between groups, F (1, 93) = .56, p > .05. The sleep tiredness variable also did not significantly differ between groups, F (1, 93) = 1.77, p > .05.

A chi-square analysis was conducted to determine whether the sleep functionality variable asking what time of day the individual functions best differed between groups. The anxiety group did not differ from the healthy comparison group on the sleep functionality variable, χ² (2) = 2.75, p > .05. Anxious youth slept an average of 9 hours over the course of three days while healthy controls slept 8.40 hours (see Table 5). In terms of age spans, anxious children (ages 7-11) slept on average 9.33 hours and healthy control children (ages 7-11) reported sleeping 9 hours. In turn, anxious adolescents (ages 12-18) slept 8.40 hours and healthy control adolescents (12-18) slept 8.09 hours. When asked how tired the participant was at the present moment, both participants in the anxiety group and healthy control group per parent reported a mean of 5.00 (on a 1-10 scale; 1 = Not tired at all; 10 = Extremely tired). Forty-three percent of youth in the anxiety group functioned best in the afternoon, while 22% functioned best in the morning and 32% in the evening. In comparison, 56% of youth in the healthy control group functioned best in the afternoon as opposed to the morning (25%) or evening (17%).
However, when controlling for age, social functioning did significantly differ between groups, $F (1, 93) = 67.27, p < .001$. The strength of the relationship between group and social functioning was strong, as assessed by a partial $\eta^2$, which suggested that group accounted for 42% of the variance of social functioning, holding constant the participants’ age. The means of social functioning scores adjusted for initial differences were ordered as expected across groups. Youth with an anxiety disorder had a larger adjusted mean ($M = 76.75$ vs. $M = 35.70$) than the healthy comparison group indicating that youth in the anxiety group reported significant moderate social functional impairment than healthy controls. Overall, the anxiety group did not differ from the healthy comparison group on number of hours slept, tiredness, and sleep functionality. However, the anxiety group did differ from the healthy comparison group on social functional impairment.

**Hypothesis 2: Prediction of Social Functioning from both Sleep Functioning and Anxiety Symptom Severity:**

Greater child social functional impairment will be better predicted by both sleep difficulty and anxiety symptom severity than by sleep difficulty or anxiety symptom severity alone.

It was predicted that greater child social functional impairment in the sample as a whole would be best accounted for by both sleep difficulty and anxiety symptom severity rather than by sleep difficulty or anxiety symptom severity alone. As previously mentioned, the continuous predictor variables were centered to increase interpretability of results. Since the average number of hours slept was the only sleep related variable that was significantly related to social functioning, the remaining sleep variable examining tiredness was not used in the remaining
analyses. The hours slept variable was centered on the mean. Additionally, to further minimize multicollinearity, the parent and child SCARED total scores were averaged and the averaged variable created was then centered on the mean. Pearson correlations were first calculated to determine the relationship between predictor and outcome variables (see Table 6). A low non-significant correlation was found between social functioning and age, $r(77) = -0.09$, $p > .05$. Social functioning approached significance with hours slept $r(77) = .21$, $p = .06$.

However, social functioning and anxiety symptom severity were significantly correlated, $r(77) = .52$, $p < .01$. As social functional impairment increased, youth experienced increased anxiety symptom severity and slept more. A significant correlation was found between age and hours slept, $r(77) = -0.45$, $p < .01$, and anxiety symptom severity, $r(77) = -0.20$, $p < .05$. Thus, older youth reported lower anxiety symptom severity and fewer hours slept. A non-significant correlation was found between hours slept and anxiety symptom severity, $r(77) = .19$, $p > .05$.

Multiple regression analyses were conducted to examine the relationships of anxiety symptom severity (as measured by the SCARED) and hours slept on social functioning (see Table 7). Holding age constant, hours slept alone significantly predicted social functioning, $R^2$ change $= .05$, $F(1, 92) = 4.72$, $p < .05$. Additionally, anxiety symptom severity alone significantly predicted social functioning when controlling for age, $R^2$ change $= .26$, $F(1, 75) = 26.24$, $p < .001$. Hours slept and report of anxiety symptom severity, as a second set of predictors, accounted for a significant proportion of the social functioning variance after controlling for age effects, $R^2$ change $= .28$, $F(2, 73) = 14.46$, $p < .001$. 
When controlling for age effects, number of hours slept did not account for a significant portion of the variance above and beyond anxiety symptom severity alone, $R^2$ change = .02, $F(1, 73) = 1.96, p > .05$ (see Table 8). However, anxiety symptom severity alone was found to account for a significant amount of the variance (24%) in social functional impairment above and beyond number of hours slept after controlling for age, $R^2$ change = .24, $F(1, 73) = 24.94, p < .001$ (see Table 9). Anxiety symptom severity and hours slept, as a set of predictors, did significantly predict social functioning. Therefore, greater child social functional impairment is best accounted for by anxiety symptom severity alone, rather than the combination of anxiety severity and hours slept or hours slept alone, when controlling for age.

**Hypothesis 3: Interaction Effect between Sleep Functioning and Anxiety Severity:**

A significant interaction effect will be found between sleep functioning and anxiety on children's social functioning.

More specifically, youth will report increased social functional impairment as anxiety symptom severity increases and the average hours slept decreases. A linear regression analysis was conducted to determine whether sleep functioning and anxiety symptom severity interact to predict children’s social functional impairment. A significant interaction effect was not found, $\beta = -.07, t(73) = -.63, p > .05$.

**Post-hoc Analyses**

Based on prior research that highlights sleep variations by specific anxiety disorders, post-hoc analyses were conducted to determine whether hours slept differed across specific anxiety symptom subtypes. The anxiety subtypes (e.g., panic, generalized, separation, social, and
school) were measured by the SCARED parent and child report measures. The empirically derived SCARED anxiety subscales can be examined when total measure scores are 30 and higher (SCARED; Birmaher et al., 1997, 1999). There were a total of 49 participants across both the child and parent SCARED measure with a total score of 30 or higher on either measure.

Within the subsample of 30 participants with a total score of 30 or higher on the child SCARED, anxiety subscales were not significantly correlated with sleep functioning: panic \( r(30) = .33, p > .05 \), generalized \( r(30) = -.24, p > .05 \), separation \( r(30) = .34, p > .05 \), social \( r(30) = -.30, p > .05 \), school \( r(30) = -.20, p > .05 \). Similarly, in the subsample of 19 participants with a total score of 30 or higher on the parent SCARED, the anxiety subscales were not significantly correlated with sleep functioning: panic \( r(19) = .28, p > .05 \), generalized \( r(19) = -.37, p > .05 \), separation \( r(19) = .20, p > .05 \), social \( r(19) = .07, p > .05 \), school \( r(19) = .03, p > .05 \).

While significant correlations were not found between anxiety subscales and hours slept, the direction of the relationship between several of the anxiety subscales were notable. The social and school anxiety subscales were found to be inversely related to hours slept per youth report and the generalized anxiety subscale was found to be inversely correlated with hours slept on both youth and parent report. The inverse correlations indicate that greater anxiety symptom severity was correlated with decreased hours slept within these particular anxiety subtypes.

Given previous research studies that have found specific anxiety disorders (e.g., GAD and SAD) have stronger associations with sleep functioning over others (Alfano, Reynolds, Scott, Dahl, & Mellman, 2012; Alfano et al., 2010), the study’s main hypothesis was analyzed once more utilizing the subsample of participants who reported a total score of 30 or higher on the
SCARED (youth and parent report) to determine whether group differences would be evident between the elevated anxiety group in comparison to healthy controls.

As previously stated, moderate correlations are recommended between dependent variables to continue with a MANCOVA (Leech, Barrett, & Morgan, 2005). A significant low moderate correlation was found between number of hours slept and the sleep tiredness variable \((r = -.26, p < .05)\). Low correlations were found between social functioning and hours slept \((r = .19, p > .05)\) and the tiredness variable \((r = .01, p > .05)\). The assumption of equality of covariance was examined by Box’s Test, \(p = .10\), indicating that the covariance matrices are equal and the assumptions are met. However, the results should be interpreted with caution given the weak correlations between social functioning and the two sleep variables. Additionally, to determine whether age continued to be a confounding variable when examining the elevated anxiety group, groups were compared. The elevated anxiety group significantly differed from the comparison group on age, \(t(68) = 2.38, p < .05\) (anxiety group: \(M = 12.14, SD = 2.37\); healthy controls: \(M = 13.59, SD = 2.85\)). Thus, subsequent analyses included age as a covariate. When controlling for age, there was a significant effect of group on sleep functioning (hours slept and tiredness) and on social functioning, Wilk’s \(\Lambda = .49, F(3, 70) = 24.42, p < .001\). This relationship was examined further with ANCOVAs. When holding age constant, the average hours slept did not significantly differ between groups, \(F(1, 72) = .17, p > .05\). The sleep tiredness variable also did not significantly differ between groups, \(F(1, 72) = 1.84, p > .05\). A significant group difference was found between groups on social functioning, when controlling for age, \(F(1, 72) = 70.61, p < .001\), with 50% of the variance described. These findings are similar to those obtained in Hypothesis 1. The subsample of those who reported increased anxiety symptom severity on the
SCARED (both parent and child) did not provide any additional clarification. Thus, analyses with the elevated anxiety group was only conducted for the analysis of Hypothesis 1 and not for Hypothesis 2 or 3 since the results of Hypothesis 1 were identical to the results conducted with the original anxiety group in comparison to healthy controls.

Next, post-hoc analyses were conducted to assess whether a relationship exists between hours slept and environmental factors, as environmental and familial factors have been found to account for significant portions of the variance in the relationship between anxiety disorders and sleep problems (Gregory, Eley, O’Connor, & Plomin, 2004). These analyses were conducted on the entire anxiety group in comparison to healthy controls. First, groups were compared on life event stressors. The anxiety group did not differ from the healthy comparison group on positive life events, $\chi^2 (30) = .24, p > .05$, and total life events, $\chi^2 (30) = .60, p > .05$. However, the groups differed on negative life events, $\chi^2 (30) = 8.74, p < .05$, with the clinically anxious youth reporting increased instances of negative life events. Second, the associations between life event stressors and hours slept were examined. Multiple linear regression analysis demonstrated a trend toward a relationship between increased hours slept and positive life events when controlling for age on the entire sample, $R^2$ change = .10, $F(1, 29) = 3.87, p = .06$. Among healthy controls, increased positive life events were found to significantly predict increased sleep functioning, $R^2$ change = .16, $F(1, 17) = 4.27, p < .05$, when controlling for age effects. These results indicate the more positive life events experienced by youth; the more hours they reported sleeping on average across three days. Among the anxiety group, positive life events did not significantly predict hours slept, $R^2$ change = .05, $F(1, 29) = .55, p > .05$, when controlling for age effects.
In terms of negative life events, such relationships with sleep functioning were not found, $R^2$ change = .00, $F(1, 29) = .00, p > .05$ (whole sample), $R^2$ change = .25, $F(1, 17) = .59, p > .05$ (healthy controls), and $R^2$ change = .00, $F(1, 9) = .02, p > .05$ (anxiety group). Additionally, the total life events score did not significantly predict sleep functioning, $R^2$ change = .04, $F(1, 29) = 1.46, p > .05$ (whole sample), $R^2$ change = .05, $F(1, 17) = 1.19, p > .05$ (healthy controls), and $R^2$ change = .01, $F(1, 9) = .10, p > .05$ (anxiety group). The non-significant findings among the anxiety group may be due to a small size ($N = 12$).

Finally, a post-hoc analysis was conducted to assess the relationship between sleep functioning and the family environment (as measured by the FRFC-P) when controlling for age. First, groups were compared on familial stressors. The anxiety group significantly differed from the healthy comparison group on familial stressors, $\chi^2 (30) = 8.73, p < .05$, meaning the clinically anxious youth presented with increased familial stressors. Second, multiple linear regression indicated family environment was not related to sleep functioning when examined in the sample as a whole, $R^2$ change = .03, $F(1, 39) = 1.28, p > .05$. A non-significant finding was also found when examined by group type, although a trend toward significance was identified in both the anxiety group, $R^2$ change = .14, $F(1, 19) = 3.34, p = .08$ and the healthy control group, $R^2$ change = .16, $F(1, 17) = 3.73, p = .07$. Among the anxiety group, increased hours of sleep were also associated with increased familial stressors. However, this relationship was the reverse among the healthy controls, where more hours slept was related to decreased familial stressors.
CHAPTER 4

Discussion

Prior research has identified a strong association between anxiety and sleep difficulties in children and adolescents. However, there has been insufficient research examining whether the additional impairment associated with sleep problems in anxious children differs in degree from the impairment associated with sleep problems in non-psychiatric controls. Given the impact of anxiety disorders on social functioning, examining whether a combination of sleep and anxiety difficulties are related to greater functional impairments in the social domain was the main focus of this study.

Anxiety and Sleep Functioning

First, the present study aimed to establish whether sleep and social functioning significantly differed between clinically anxious youth and healthy controls. The hypothesis was partially confirmed. In the present study, sleep functioning did not significantly differ between groups when controlling for age. Both anxious and healthy control youths experienced similar sleep functioning per parent report, including: average number of hours slept averaged over the course of three consecutive days, time of day they functioned best, and how tired they felt at the time of the evaluation. On average, the clinically anxious youth slept 8.9 hours per night over the course of three days and the healthy controls reported sleeping 8.4 hours per night. When examined by age group, clinically anxious children (ages 7-11) slept 9.33 hours and healthy controls slept 9 hours. Clinically anxious adolescents (ages 12-18) on average reported sleeping 8.40 hours per night and healthy controls reported sleeping 8.09 hours per night. Additionally, both the clinically anxious group and healthy control group reported feeling “somewhat tired” at
the time of the study and both groups reported they functioned best in the afternoon. The mean number of hours slept decreased as age increased. This is consistent with developmental research in which adolescents sleep less as result of the sleep phase delay that is experienced due to puberty. During the sleep phase delay, adolescents’ biological need for sleep shifts to as late as 11pm and they are unable to sleep the full recommended 10 to 11 hours because most schools have a very early start time (National Sleep Foundation, 2004; Wolfson & Carskadon, 2003).

Nevertheless, when the relationship between sleep functioning and anxiety was examined further, a significant relationship was not found between anxiety subtypes (e.g., panic, school refusal, separation, generalized, and social anxiety) as reported by parent or child and sleep functioning. These results are in contrast to studies that have found strong relationships between sleep difficulties and specific anxiety disorders such as GAD and SAD (Alfano et al., 2006, 2010). Despite the non-significant relationship, the severity of generalized anxiety, social anxiety, and school refusal subtype increased as the number of hours slept decreased based on parent sleep report. The direction of these correlations is consistent with current research and indicates the importance of continuing to examine the relationship between sleep functioning and specific anxiety disorders to better differentiate sleep patterns between anxiety disorders.

There may be various reasons for the lack of significant group differences on sleep functioning. For example, healthy controls reported considerably lower than expected hours of sleep in comparison to previous population-based studies in which the mean has been reported to range from 10 to 10.66 hours (Owens, Spirito, McGuinn, & Nobile, 2000; Liu, Liu, Owens, & Kaplan, 2005; van Litsenburg, Waumans, van den Berg, & Gemke, 2010). Indeed, the National Sleep Foundation (2004) cautions that children and adolescents are increasingly sleeping less
than the recommended 10 to 11 hours per night. Furthermore, Hudson and colleagues (2009) found that clinically anxious children slept significantly 30 minutes less over the course of a week in comparison to healthy controls. Interestingly, sleep disturbances were only evident for the anxious youth on school nights, but not during the weekend. The distinction between weekend and weeknights was not captured as part of the current study and may have obscured findings. However, consistent with the findings of this study, Hudson et al (2009) also found that the clinically anxious participants did not significantly differ from controls in their report of daytime sleepiness or their experience of feeling tired from lack of sufficient sleep. The authors hypothesized that the increased levels of anxiety leading to hyperarousal mask the experience of feeling tired or sleepy, and that this may account for the lack of sleepiness among anxious youth despite insufficient sleep. Thus, they recommend that not only nocturnal patterns be investigated in youth, but also their daytime impairments that extend beyond asking about fatigue or daytime sleepiness. Therefore, measures that examine reaction time, attention, or concentration are suggested to better ascertain daytime impairments associated with sleep disturbances in anxious youth (Hudson et al., 2009).

Although the current study did not find a significant difference in sleep functioning between clinically anxious youth and healthy controls, it is important to note that even minimal loss of sleep over a period of time can have significant consequences on youths’ overall functioning (Sadeh, Raviv, & Gruber, 2000; Fallone, Acebo, & Seifer, 2005). For example, in a community study of school-aged children, even a modest sleep restriction of 35 minutes over the course of three days resulted in poor performance on neurobehavioral tasks (such as reaction time, memory, and concentration) that can have a significant impact on school performance.
Additionally, when examining the impact of sleep deprivation in children diagnosed with ADHD compared to healthy controls on a measure of vigilance and sustained attention, both groups performed poorly (Gruber et al., 2011). Remarkably, the ADHD group not only performed poorly, but also their performance declined from the subclinical ranges to the clinical range. These findings are important to keep in mind, as the clinically anxious youth and healthy controls are sleeping about an hour less than the recommended 10-11 hours per night (National Sleep Foundation, 2004).

**Anxiety, Sleep Functioning, and Social Functioning**

In contrast to sleep functioning, social functioning was found to significantly differ between the anxiety and healthy control groups. Clinically anxious youth reported greater social functioning difficulties than healthy controls. On average, the social functioning impairment reported by the anxiety group was categorized as moderately socially impaired. This finding is consistent with extensive literature indicating that youth with an anxiety disorder tend to have social functioning impairments in comparison to same age peers without an anxiety disorder (Beidel et al., 1999; Rubin & Burgess, 2001; Silverman & Ginsburg, 1998).

Another main target of the study was to determine whether, in the full sample, child social functional impairment would be better predicted by both poor sleep functioning (average hours slept over three days) and anxiety symptom severity combined, rather than by poor sleep functioning or anxiety symptom severity alone. Increased anxiety symptom severity was found to be significantly correlated with increased social functional impairment, which is consistent with existing literature indicating that children with anxiety symptoms experience increased difficulties related to social functioning (e.g., Hawker & Boulton, 2000; La Greca & Harrison,
Increased anxiety symptom severity is associated with poor social functioning. To explain this relationship, research has found that children who report severe anxiety symptoms are more inclined to attend to threatening stimuli (Waters, Henry, Mogg, Bradley, & Pine, 2010). The increased attention to threatening stimuli may lead to social withdrawal or social avoidance that can result in limited social engagement. Limited social engagement is related to poor social skills, and ultimately this cycle works to maintain anxiety symptoms (Rubin & Burgess, 2001). Unexpectedly, increased social functional impairment and anxiety symptom severity were associated with increased number of hours slept. Additionally, increased number of hours slept significantly predicted increased social functional impairment when controlling for age effects. As previously discussed, Hudson and colleagues (2009) found that clinically anxious youth reported no significant differences in sleep patterns over the weekend in comparison to healthy controls. Both the clinically anxious group and the healthy control group fell asleep later, woke up around the same time, and slept about the same amount of hours. However, the clinically anxious group fell asleep faster on the weekend in comparison to healthy controls. The present study did not differentiate between weekday and weekend sleep patterns. The total hours slept averaged over the course of three days included solely weekdays, weekend, or a combination of both. Thus, the direction of the relationships between number of hours slept and anxiety symptom severity and social functioning impairment may or may not have been different if weekend versus weeknight variability had been accounted for in this study.

Ultimately, the number of hours slept was found to significantly predict social functional impairment. Additionally, the number of hours slept was not found to predict social functioning
above and beyond anxiety symptom severity alone, when controlling for age. The combination of anxiety symptom severity and sleep functioning was found to predict social functioning than sleep functioning alone. However, anxiety symptom severity was found to best predict social functioning above and beyond the combination of sleep functioning and anxiety symptom severity. Thus, the hypothesis that social functional impairment would be best predicted by both poor sleep functioning and anxiety symptom severity was partially supported. While additive effects of anxiety symptom severity and sleep functioning were not found in relation to social functional impairment, these findings further corroborate prior research indicating a strong correlation between anxiety disorders and poor social outcomes (Verduin & Kendall, 2008; Settipani & Kendall, 2013). In addition, these findings extend the scientific knowledge base as increased anxiety symptom severity was found to be associated with poorer social functioning; social functioning also may be predictive of anxiety symptoms. Indeed, the current findings related to social functioning lend support to the notion that these specific skills should be assessed and targeted in treatment with anxious youth. This notion is also supported by a recent study that found that social competence was the only predictor of treatment response among other social functioning variables (e.g., peer conflict) in a clinically anxious group of children (Settipani & Kendall, 2013).

**Anxiety, Sleep Functioning, and Environmental Stressors**

Regarding the role of youth life stressors, within the anxiety group, a greater frequency of negative life events was reported in comparison to the healthy controls. This finding is consistent with studies that have found a relationship between increased report of negative life events and anxiety disorders in children (Boer, Markus, Maingay, Lindhout, Borst, &
Hoogendijk, 2002). The loss of a sense of control during the experience of negative life events is thought to disrupt the relaxed stated that is needed for sleep through worry and rumination (Harvey, 2002). However, despite the greater instances of negative life events among the anxiety group, negative life events were not significantly related to sleep functioning. Additionally, negative life events did not significantly predict number of hours slept among healthy controls when controlling for age. These results contradict prior research that finds negative life event stressors are related to sleep problems (Barclay, Eley, Rijsdijk, & Gregory, 2011; Vahtera et al., 2007).

Report of positive life events in children and adolescents are associated with increased well-being (Doyle, Wolchik, & Dawson-McClure, 2002; Jackson & Warren, 2000). On average, both the anxiety group and healthy controls reported similar instances of positive events. Among healthy controls, positive life events were found to significantly predict number of hours slept when controlling for age. Instances of positive life events were significantly related to increased number of hours slept among the healthy control group. However, positive life events did not significantly predict number of hours slept when holding age constant in the anxiety group. The experience of positive life events in healthy controls may facilitate the relaxed physiological state necessary for sleep that can result in increased hours slept.

Additionally and accordant with current research (e.g., Tiet et al., 2001), clinically anxious youth reported greater instances of familial stressors in comparison to healthy controls. Interestingly, a non-significant positive relationship was found between mean hours of sleep and familial factors in the clinically anxious sample. The direction of this relationship is inconsistent with current research findings investigating factors such as parental psychopathology and family
disorganization, in which increased parental psychopathology or family organization is associated with decreased number of hours slept (Gregory, Eley, O’Connor, Rijsdijk, & Plomin, 2005). Among healthy controls, familial stressors were not found to predict sleep functioning either.

In summary, while clinically anxious youth reported increased instances of negative life events and familial stressors in comparison to healthy controls, these environmental factors were unrelated to sleep functioning. Whereas positive life events were found to significantly predict sleep functioning in healthy controls, this relationship was not found in the clinically anxious group. A prior study found that positive life events were found to be protective for children as long as their level of stress was low to moderate and not chronic, but positive life events were unrelated to improved well-being in children when they experienced chronic or moderate to severe stressors (Sandberg, McCann, Ahola, Oja, Paton, & McGuinness, 2002). Thus, it is hypothesized that the impact of increased negative life events and familial stressors in the clinically anxious group counterbalanced any potential benefits of positive life events in relation to sleep functioning. Additionally, of note was the positive relationship between negative life events and familial stressors in relation to sleep functioning, in which greater number of hours slept was associated with increased negative life events and familial stressors. It is unclear what is driving the direction of these relationships. However, studies have found that children diagnosed with GAD or SAD are more likely to report negative life events in comparison to children diagnosed with other types of anxiety disorders (Tiet et al., 2001). Interestingly, children diagnosed with GAD or SAD report a stronger association with sleep problems in comparison to other types of anxiety disorders (Alfano et al., 2006, 2010). Thus more
differentiated findings may have been obtained in the present study, if we had analyzed separately by anxiety disorder rather than using a heterogeneous sample of anxiety disorders. Further, the lack of significance should be interpreted with caution as it may have been due to the small sample size and the low frequency of cases reporting extremes of negative life events, which limited statistical power to detect associations.

**Strengths and Weaknesses of Present Study**

The present study attempted to address certain methodological limitations noted in previous investigations. First, whereas most other studies have mainly focused on the relationship between sleep difficulties and anxiety alone, this study aimed to expand on existing research by examining the relationship of anxiety symptom severity and sleep functioning on functional impairment, particularly social functional impairment. Second, sleep and social functioning were examined in a clinically anxious sample that was assessed with a structured clinical interview so that conclusions could be generalized based on a clinical anxiety disorder diagnosis as opposed to anxiety symptom severity alone. Third, to control for the high comorbidity between sleep disturbances, anxiety disorders, and depression, youth who met criteria for a comorbid depressive disorder were excluded from the clinically anxious group. Fourth, comparison groups aided in controlling for extraneous variables and ensuring that all subjects were assessed under the same environmental conditions.

Despite the strengths of this study, it is not without limitations. First, the lack of a standardized comprehensive sleep measure may have significantly restricted the ability to identify differences between groups. The measure used to assess sleep functioning was a limited assessment of sleep behaviors including the average number of hours slept over the course of
three days, how tired the participant felt at the time of the study, and what time of day they functioned best. Given the multifaceted behaviors associated with sleep disturbance, it is preferable to use a standardized measure that expands on the sleep-related behaviors assessed (e.g., parasomnias, sleep onset, night-time awakenings) (Hudson, Gradisar, Gamble, Schniering, & Rebelo, 2009). In addition, the present study did not differentiate between weekday and weekend sleep patterns. Therefore, the direction of the relationships between number of hours slept and anxiety symptom severity and social functioning impairment may have been different if a distinction between weekends versus weeknights had been accounted for in this study (Wolfson & Carskadon, 1998). Seco, parents of children with an anxiety disorder are said to be better reporters of sleep problems than their children, since their children’s sleep problems are likely to cause a disruption in their own sleep schedule (Alfano et al., 2010; Storch et al., 2008; Alfano, Pina, Zerr, & Villalta, 2011; Paavonen, Solantaus, Almqvist, & Aronen, 2003). The sleep measure for this study was based on parent report, but collaboration between adolescents and parents was likely in completion of the measure, as parents may not monitor adolescents’ bedtime routine as closely as younger children. Further, in instances in which a scheduled bedtime is endorsed by parents it may not be an accurate account of what time the adolescent actually fell asleep. Additionally, biased parent report has also been noted due to parents’ own disrupted sleep (Alfano et al., 2010). Therefore, to address the potential bias in parent report of sleep problems, both parent and youth report should be used in addition to objective measures to accurately capture physiological sleep-related problems in youth with an anxiety disorder in comparison to healthy controls.
Finally, the small sample size within each group for certain statistical analyses may have limited power to detect effects. Thus, non-significant effects should be interpreted with caution as group differences particularly regarding sleep difficulties may have been missed. Also, the generalizability of the results are limited due to the sample demographics. As noted, majority of both the anxiety and healthy control group are predominantly of high social economic status (SES) and Caucasian. Thus, it is unclear how these results may have differed based on other ethnicities and SES. Furthermore, given the cross-sectional design, causation and direction of influence between anxiety disorders, sleep difficulties, and social functioning cannot be inferred.

Implications

As discussed, prevailing literature suggests that a complex bidirectional relationship exists between sleep functioning and anxiety disorders. The current study, on the contrary, did not find a significant relationship between anxiety disorders and sleep functioning. Additionally, sleep functioning was not found to predict social functioning above and beyond anxiety symptom severity alone. While the current study did not find a significant difference between the anxiety and healthy control groups on sleep functioning, the lack of significance should be interpreted with caution, as it is difficult to draw definitive conclusions given the investigation’s limitations. The National Sleep Foundation (2004) indicates that children and adolescents in general are increasingly sleeping less than the recommended 10 to 11 hours per night. The low average hours of sleep reported by both groups is concerning given the significant consequences from even moderate sleep deficits on overall functioning (Sadeh, Keinan, & Daon, 2004). These findings also lend further support to the importance of examining “daytime functioning and
feelings” to better understand sleep functioning in the context of anxiety disorders (Hudson et al., 2009, pg. 343).

The current study investigated social functioning in addition to daytime functioning (e.g., sleepiness and what time of day one functions best) to better understand the additive impairment associated with inadequate sleep functioning and anxiety disorders. In the current study, an interaction effect between sleep functioning and social functioning was not present and sleep functioning did not significantly predict social functioning above and beyond anxiety symptom severity alone. Despite these non-significant findings, it is important to note when comparing clinically anxious youth to healthy controls where both groups report similar rates of inadequate sleep, clinically anxious youth still present with increased rated of negative life events, familial stressors, and social functional impairments. These findings highlight the importance of assessing for functional impairments in addition to anxiety symptom severity among anxious youth. In particular, social functioning is a domain that has significant long-term consequences if not appropriately assessed and treated. Social functioning should be examined across all anxiety disorders and not only limited to those diagnosed with Social Phobia. As noted, the degree of social functional impairments experienced by clinical anxious youth are distinctive from the general population, and a thorough assessment can inform treatment goals and has the potential for providing a more effective treatment. This is particularly notable given that research has found that social competence is a significant predictor of differential response to CBT treatment for anxiety disorders in children (Settipani & Kendall, 2013). Principally, it is noted that lack of social competence may impede children from building rapport with their therapist. Further, the psychoeducational treatment component focused on emotions may not be
readily comprehended and generalized if the child has unaddressed social communication and reciprocity deficits, which understandably limits the success of the overall treatment.

Overall, the findings and limitations of this study stress the importance of continued research investigating anxiety disorders and sleep disturbances and their relation to functional impairments. Future research should include objective measures of sleep functioning in addition to a more structured and comprehensive measure of sleep reported by both parent and child. Additionally, the field would benefit from being examining the relationships between social functioning, sleep disturbances, and anxiety longitudinally, especially when considering the bidirectional and shared developmental course of these variables. In turn, results from longitudinal studies would help to inform targeted interventions, which would assist in preventing chronic psychopathology.
Table 1
*Correlations of MANCOVA Dependent Variables.*

<table>
<thead>
<tr>
<th>Hours Slept</th>
<th>Tiredness</th>
<th>SRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours Slept</td>
<td>-.34**</td>
<td>.24*</td>
</tr>
<tr>
<td>Tiredness</td>
<td></td>
<td>-.03</td>
</tr>
<tr>
<td>SRS</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. Ave. Hrs. = Average Hours; SRS = Social Responsiveness Scale.*
*significant at the 0.05 level (2-tailed).*
**significant at the 0.01 level (2-tailed).*
Table 2
Anxiety Group and Healthy Control Group Demographics.

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Anxiety (N = 60)</th>
<th>HC (N = 36)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>35 (58.3%)</td>
<td>21 (58.3%)</td>
</tr>
<tr>
<td>Male</td>
<td>25 (41.7%)</td>
<td>15 (41.7%)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Children (7-11)</td>
<td>31 (51.7%)</td>
<td>13 (36.1%)</td>
</tr>
<tr>
<td>Adolescents (12-18)</td>
<td>29 (48.3%)</td>
<td>23 (63.9%)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian (Non-Latino)</td>
<td>52 (86.7%)</td>
<td>30 (83.3%)</td>
</tr>
<tr>
<td>African-American</td>
<td>5 (8.3%)</td>
<td>2 (5.6%)</td>
</tr>
<tr>
<td>Asian</td>
<td>1 (1.7%)</td>
<td>1 (2.8%)</td>
</tr>
<tr>
<td>Unreported</td>
<td>2 (3.3%)</td>
<td>3 (8.3%)</td>
</tr>
<tr>
<td><strong>Household Income</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$15,000 – 24,999</td>
<td>0 (0%)</td>
<td>1 (2.8%)</td>
</tr>
<tr>
<td>$25,000 – 39,999</td>
<td>1 (1.7%)</td>
<td>1 (2.8%)</td>
</tr>
<tr>
<td>$40,000 – 59,999</td>
<td>6 (10%)</td>
<td>3 (8.3%)</td>
</tr>
<tr>
<td>$60,000 – 89,999</td>
<td>10 (16.7%)</td>
<td>13 (36.1%)</td>
</tr>
<tr>
<td>$90,000 – 179,999</td>
<td>25 (41.7%)</td>
<td>10 (27.8%)</td>
</tr>
<tr>
<td>Over $180,000</td>
<td>15 (25%)</td>
<td>3 (8.3%)</td>
</tr>
<tr>
<td>Unreported</td>
<td>3 (5%)</td>
<td>5 (13.9%)</td>
</tr>
</tbody>
</table>

*Note: Due to rounding, not all numbers do not add up to 100%. HC = Healthy Controls.*
Table 3
Number of Participants, Means, and Standard Deviations of Clinician, Parent, and Child Completed Measures.

<table>
<thead>
<tr>
<th>Group</th>
<th>Anxiety</th>
<th></th>
<th>Healthy Controls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean (SD)</td>
<td>N</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>SCARED</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent Subtypes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Panic</td>
<td>55</td>
<td>4.5 (4.0)</td>
<td>33</td>
<td>1.1 (2.2)</td>
</tr>
<tr>
<td>Generalized</td>
<td>56</td>
<td>8.8 (4.6)</td>
<td>35</td>
<td>1.8 (3.9)</td>
</tr>
<tr>
<td>Separation</td>
<td>56</td>
<td>5.1 (4.6)</td>
<td>35</td>
<td>0.8 (2.5)</td>
</tr>
<tr>
<td>Social</td>
<td>55</td>
<td>6.3 (3.9)</td>
<td>35</td>
<td>2.6 (3.2)</td>
</tr>
<tr>
<td>School</td>
<td>58</td>
<td>2.5 (2.3)</td>
<td>35</td>
<td>0.51 (1.1)</td>
</tr>
<tr>
<td>Total</td>
<td>49</td>
<td>26.6 (13.4)</td>
<td>33</td>
<td>7.0 (11.4)</td>
</tr>
<tr>
<td>Child Subtypes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Panic</td>
<td>57</td>
<td>6.8 (5.0)</td>
<td>36</td>
<td>2.0 (1.9)</td>
</tr>
<tr>
<td>Generalized</td>
<td>56</td>
<td>8.89 (4.3)</td>
<td>36</td>
<td>3.2 (2.8)</td>
</tr>
<tr>
<td>Separated</td>
<td>56</td>
<td>5.8 (4.3)</td>
<td>36</td>
<td>1.8 (2.1)</td>
</tr>
<tr>
<td>Social</td>
<td>57</td>
<td>6.8 (3.7)</td>
<td>36</td>
<td>3.9 (3.1)</td>
</tr>
<tr>
<td>School</td>
<td>57</td>
<td>2.8 (2.2)</td>
<td>36</td>
<td>1.0 (1.2)</td>
</tr>
<tr>
<td>Total</td>
<td>55</td>
<td>31.1 (14.6)</td>
<td>36</td>
<td>12.0 (7.9)</td>
</tr>
<tr>
<td>CGI</td>
<td>46</td>
<td>4.2 (0.8)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>CGAS</td>
<td>48</td>
<td>50.4 (6.2)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>LE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>12</td>
<td>3.8 (2.7)</td>
<td>20</td>
<td>3.5 (2.9)</td>
</tr>
<tr>
<td>Negative</td>
<td>12</td>
<td>4.9 (3.5)</td>
<td>20</td>
<td>2.2 (1.8)</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>8.7 (4.5)</td>
<td>20</td>
<td>5.7 (3.8)</td>
</tr>
<tr>
<td>FRFC-P</td>
<td>22</td>
<td>7.6 (3.9)</td>
<td>20</td>
<td>2.6 (1.9)</td>
</tr>
</tbody>
</table>

Note. SD = Standard Deviation; SCARED = Screen for Child Anxiety Related Emotional Disorders; Ave. Hrs. = Average Hours; LE = Life Events; SRS = Social Responsiveness Scale; FRFC-P = Family Risk Factor Checklist; CGI = Clinical Global Impressions; CGAS = Children’s Global Assessment Scale. Dashes indicate the CGI and CGAS were not administered to Healthy Controls.
Table 4  
*Multivariate Analysis of Covariance of Sleep Functioning and Social Functioning on Group.*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wilk’s Λ</th>
<th>F</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>.70</td>
<td>12.95***</td>
<td>.30</td>
</tr>
<tr>
<td>Group</td>
<td>.56</td>
<td>24.08***</td>
<td>.45</td>
</tr>
</tbody>
</table>

***significant at the 0.001 level (2-tailed).
Table 5
Sleep and Social Functioning: Means, Standard Deviations, and Percentages.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Anxiety</th>
<th>Healthy Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours Slept</td>
<td>59</td>
<td>8.9 (1.1)</td>
</tr>
<tr>
<td>Tiredness</td>
<td>59</td>
<td>5.0 (2.1)</td>
</tr>
<tr>
<td>Functionality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morning</td>
<td>13 (21.7%)</td>
<td>9 (25%)</td>
</tr>
<tr>
<td>Afternoon</td>
<td>26 (43.3%)</td>
<td>20 (55.6%)</td>
</tr>
<tr>
<td>Evening</td>
<td>19 (31.7%)</td>
<td>6 (16.7%)</td>
</tr>
<tr>
<td>Unreported</td>
<td>2 (3.3%)</td>
<td>1 (2.8%)</td>
</tr>
<tr>
<td>SRS</td>
<td>59</td>
<td>76.8 (27.0)</td>
</tr>
</tbody>
</table>
Table 6
Summary of Follow-up Analysis of Covariance of Social Functioning and Sleep Functioning on Group

<table>
<thead>
<tr>
<th>Source</th>
<th>Mean Square</th>
<th>df</th>
<th>F</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours Slept</td>
<td>.66</td>
<td>1</td>
<td>.56</td>
<td>.01</td>
</tr>
<tr>
<td>Tiredness</td>
<td>6.57</td>
<td>1</td>
<td>1.77</td>
<td>.02</td>
</tr>
<tr>
<td>SRS</td>
<td>38122.07</td>
<td>1</td>
<td>67.27***</td>
<td>.42</td>
</tr>
</tbody>
</table>

*** significant at 0.001 level (2-tailed).
Table 7
*Correlations between Predictors (centered) and Outcome Variables*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hours Slept</th>
<th>SCARED</th>
<th>Age</th>
<th>SRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours slept</td>
<td>.19</td>
<td>-.45**</td>
<td>.21</td>
<td></td>
</tr>
<tr>
<td>SCARED</td>
<td>-.20</td>
<td>.52**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.09</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. SCARED = Screen for Child Anxiety Related Emotional Disorders; SRS = Social Responsiveness Scale. **significant at the 0.01 level (2-tailed).
Table 8

Summary of Multiple Regression Analyses for Anxiety Symptom Severity Predicting Social Functioning

<table>
<thead>
<tr>
<th>Model</th>
<th>$\beta$</th>
<th>Beta</th>
<th>$R^2$ Change</th>
<th>$F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>26.24***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>.46</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SCARED</td>
<td>.79</td>
<td>.52</td>
<td>.26</td>
</tr>
<tr>
<td>Model 2</td>
<td>4.72*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>.26</td>
<td>.02</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hours Slept</td>
<td>6.39</td>
<td>.25</td>
<td>.05</td>
</tr>
<tr>
<td>Model 3</td>
<td>14.46***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>.93</td>
<td>.08</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SCARED</td>
<td>.76</td>
<td>.51</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hours Slept</td>
<td>4.07</td>
<td>.16</td>
<td>.28</td>
</tr>
</tbody>
</table>

Note. SCARED = Screen for Child Anxiety Related Emotional Disorders.
*significant at the 0.05 level (2-tailed).
***significant at the 0.001 level (2-tailed).
Table 9

Hierarchical Regression Analyses for Sleep Functioning Predicting Social Functioning

<table>
<thead>
<tr>
<th>Model</th>
<th>β</th>
<th>Beta</th>
<th>$R^2$ Change</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
<td>.54</td>
</tr>
<tr>
<td>Age</td>
<td>-.94</td>
<td>-.09</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
<td>26.61***</td>
</tr>
<tr>
<td>Age</td>
<td>.20</td>
<td>.02</td>
<td>.26</td>
<td></td>
</tr>
<tr>
<td>SCARED</td>
<td>.79</td>
<td>.52</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 3</td>
<td></td>
<td></td>
<td></td>
<td>1.96</td>
</tr>
<tr>
<td>Age</td>
<td>.93</td>
<td>.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCARED</td>
<td>.76</td>
<td>.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours Slept</td>
<td>4.07</td>
<td>.16</td>
<td>.02</td>
<td></td>
</tr>
</tbody>
</table>

Note. SCARED = Screen for Child Anxiety Related Emotional Disorders.

***significant at the 0.001 level (2-tailed).
### Table 10

*Summary of Hierarchical Regression Analyses for Anxiety Symptom Severity Predicting Social Functioning*

<table>
<thead>
<tr>
<th></th>
<th>β</th>
<th>Beta</th>
<th>$R^2$ Change</th>
<th>$F$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.94</td>
<td>-.09</td>
<td>.01</td>
<td>.54</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.15</td>
<td>.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours Slept</td>
<td>5.76</td>
<td>.22</td>
<td>.04</td>
<td>3.00</td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.93</td>
<td>.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours Slept</td>
<td>4.07</td>
<td>.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCARED</td>
<td>.76</td>
<td>.51</td>
<td>.24</td>
<td>24.94***</td>
</tr>
</tbody>
</table>

*Note.* SCARED = Screen for Child Anxiety Related Emotional Disorders.

***significant at the 0.001 level (2-tailed).
References


with 5HTTLPR, PER3, and CLOCK 3111. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 156*(6), 681-690.


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