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LONGITUDINAL RELATIONSHIPS BETWEEN SLEEP PROBLEMS AND EXTERNALIZING BEHAVIOR IN CHILDREN: INVESTIGATING GENETIC AND TEMPERAMENTAL MODERATORS

Gabriel Casher
Southern Illinois University Carbondale, gabriel.casher@gmail.com

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by

Gabriel A. Casher

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Approved by:

Lisabeth DiLalla, Ph.D., Chair

Philip Anton, Ph.D.

Mary Louise Cashel, Ph.D.

Holly Cormier, Ph.D.

Michelle Kibby, Ph.D.

Graduate School

Southern Illinois University Carbondale

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The current study aimed to evaluate multiple longitudinal determinants of externalizing behavior problems in twins/triplets aged 7 to 12 years. Specifically, a prospective longitudinal design was utilized to assess relationships between age 5 sleep problems, age 5 temperament traits, and later externalizing problems. Additionally, heritability of sleep problems was assessed by utilizing the twin method, and genetic contributions of two specific genes – DRD4 and 5-HTTLPR – were evaluated. A total of 93 twins/triplets (40 boys and 53 girls) and their parents participated in the current study, and data were collected through self-report, parent-report, and molecular and behavioral genetic methods. Results suggest that sleep disturbances are significantly heritable, and that neither early sleep problems, temperament traits, nor specific genes significantly predicted follow-up externalizing problems. Post-hoc analyses assessing gene X environment interactions showed that externalizing problems were significantly predicted by the interaction between stressful life events and DRD4 risk, which is consistent with differential susceptibility models. This study has implications for future research as well as clinical practice, including for early screening, prevention, and intervention efforts aimed at decreasing childhood externalizing and sleep problems.

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CHAPTER 1

INTRODUCTION

By the age of 18, children have spent, on average, 40% of their lives sleeping (Mindell & Owens, 2010). However, childhood sleep problems are common, with approximately 25% of children experiencing some type of sleep problem (Owens, 2008). These issues range from clinical sleep-wake disorders (e.g., narcolepsy) to other non-clinical sleep-related problems (e.g., difficulty falling asleep, nightmares). Additionally, sleep problems commonly co-occur with other emotional/behavioral problems, including autism, ADHD, bipolar disorder, depressive disorders, anxiety disorders, obsessive-compulsive disorder, stress-related disorders, and externalizing disorders (Gregory & Sadeh, 2016). Whereas the importance of sleep for several aspects of childhood health and well-being has gained recognition in recent years, most of the existing literature focuses on concurrent relationships between sleep and these other problems, which is problematic because it does not address potential directional effects. Specifically, the current study will focus on relationships between sleep problems and externalizing behavior, which includes "acting-out" behaviors such as physical aggression and rule-breaking. Given the multiple negative consequences of externalizing behavior spanning from childhood (Frick & McMahon, 2013) into adulthood (Deater-Deckard, Dodge, Bates, & Pettit, 1998), the identification of patterns of risk factors, including sleep problems, is important and provides researchers and clinicians with knowledge to inform screening and intervention efforts.

A critical question concerning relationships between sleep problems and externalizing behavior is whether there is a directional effect between the two, or if sleep is simply a co-occurring symptom of externalizing problems. Whereas some suggest that sleep problems precede externalizing behavior (Goodnight, Bates, Staples, Pettit, & Dodge, 2007), it is also

possible that sleep problems are a manifestation of externalizing behavior in children with compromised regulatory abilities. Indeed, research clarifying this question is lacking. For current study, I evaluated the longitudinal relationships between sleep problems and externalizing behavior by utilizing a prospective design with measurements of both sleep problems and externalizing at two time points.

Importantly, the development of externalizing behavior in childhood is certainly related to multiple factors other than sleep problems. Thus, in addition to sleep problems, two other sources of risk for externalizing were evaluated in the current study. First, multiple temperament traits, which are genetically-driven traits that are evident in infancy, are related to externalizing behavior in childhood. There is also limited evidence that these traits might interact with sleep problems in the development of externalizing behavior (Goodnight et al., 2007). For the current study, I evaluated if children with sleep problems in addition to particular "risk" temperament traits are more likely to develop externalizing problems, compared to children with sleep problems but no "risk" temperament traits. Second, specific genetic variations related to neurotransmitter (i.e., serotonin and dopamine) functioning are suggested to place children at greater risk for emotional and behavioral problems (e.g, DiLalla, Bersted, & John, 2015). The mechanisms of these genetic effects remain unclear, but researchers have suggested several models by which specific genes confer risk for maladjustment. One model, the diathesis-stress model (Gottesman, 1991), states that "risk" genes place individuals at a higher risk for adverse reactions to stressful life events (i.e., trauma, childhood neglect). As it pertains to the current study, it may be that genes confer sensitivity to the effects of sleep problems, such that children with sleep problems and a risk genotype are more likely to develop externalizing problems, compared to children with sleep problems but without a risk genotype. Another model, the differential susceptibility model

(Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007), states that genetic variations confer malleability rather than simply risk. In this sense, it may be that children with a "risk" genotype may have poor outcomes with a stressful environment, whereas those same children would have favorable outcomes in an enriching and supportive environment. For the current study, I evaluated genetic effects in line with these genetic vulnerability models.

Existing literature on these relationships suffers from two major limitations. First, when assessing for the presence of early risk factors, researchers frequently rely on retrospective reports. This method is frequently used to assess temperament traits by asking parents to recall their children's behaviors from several years earlier (e.g., Bates, Pettit, Dodge, & Ridge, 1998; Goodnight et al., 2007), which can lead to biased reports of temperament. Retrospective ratings may be affected by changed memories as a reflection of subsequent events, as well as due to the passing of time. Second, existing literature has largely neglected the impact of genetic factors, including heritability and specific genetic factors, on the relationship between sleep and externalizing. Genetic vulnerability theories (e.g., Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Gottesman, 1991) suggest that genetic differences must be considered to best predict outcomes. For the current study, I addressed these limitations and extended existing literature on risk factors for externalizing problems.

Thus, for the current study, I utilized a prospective longitudinal design to examine the relationships between multiple sources of risk for externalizing problems in 4- to 12-year-old children. Additionally, I utilized a twin sample, which allowed for further examination of genetic effects. I addressed several research questions in this study: 1) what are the longitudinal relationships between sleep problems and externalizing problems in childhood? 2) to what degree are sleep problems related to shared genes? and 3) how do temperament traits and genes affect the

relationship between sleep and externalizing? Given existing literature regarding links between sleep problems and externalizing behavior (e.g., Goodnight et al., 2007), I expected that temperament traits would moderate the longitudinal relationship between sleep and externalizing, such that children with a "risk" temperament trait are more vulnerable to the long-term effects of early sleep problems, compared to children without a "risk" temperament. Similarly, I expected that children with "risk" genotypes (related to dopamine and serotonin) would be more vulnerable to the effects of sleep problems, compared to children without "risk" genotypes.

Results from this study have implications for theories regarding risk factors for behavioral problems, as well as for clinical research and practice. Research on mechanisms of genetic vulnerability adds to our understanding of how genes exert an influence on behavioral outcomes. This study evaluated whether a genetic vulnerability can help explain why some children with sleep problems develop externalizing behavior, whereas others do not. Understanding these interactions, in turn, guides clinical research on developmental trajectories of behavior problems and the risk factors associated with those trajectories. In practice, the identification of combinations of risk factors that are likely to lead to behavioral problems guides screening and intervention techniques.

CHAPTER 2

LITERATURE REVIEW

Given the high prevalence of sleep problems in childhood (Owens, 2008), research demonstrating the impacts of sleep problems on developmental outcomes has gained attention in recent years. This body of research suggests that early sleep problems are associated with several other problems, including behavioral, mood, and academic issues (Fallone, Owens, & Deane, 2002; Morrison, McGee, & Stanton, 1992) as well as clinical psychopathology (Gregory & Sadeh, 2016). There is also limited evidence that sleep problems interact with other factors, including temperament, in the development of problematic behavior (Goodnight et al., 2007). The current study aims to extend findings of relationships between sleep problems, temperament traits, and externalizing behavior in 5- to 12-year-old children. Specifically, this study addressed several limitations of the existing literature, including the reliance on retrospective reporting and the lack of longitudinal designs. Also, this study included a genetically-informed sample, which allowed for examination of specific genetic effects on the relationship between sleep and externalizing. Overall, this study contributes to the existing body of research on risk factors for externalizing problems in childhood, which guides clinical assessment, prevention, and intervention efforts.

Sleep Problems in Childhood

Over the past few decades, research on child and adolescent sleep has grown significantly. Researchers have studied the normal development of sleep habits over the pediatric age span, but have also identified abnormal sleep behaviors that occur in childhood and adolescence. Approximately 25% of children experience sleep problems (Owens, 2008), and there is a general decrease in sleep problems from preschool through adolescence (Gregory & O'Connor, 2002;

Owens, 2008). Of note is the difference between "sleep problems" and sleep-wake disorders. Sleep-wake disorders such as insomnia, breathing-related sleep disorder (e.g., sleep apnea), and narcolepsy are diagnosable disorders according to the Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition (DSM-5; American Psychiatric Association, 2013). Sleep problems, however, are a more broadly-defined set of behaviors including sleep disorders, but also including non-clinical problematic behaviors related to sleep (e.g., difficulty initiating/maintaining sleep).

Developmental studies have added considerably to our understanding of sleep in typically developing youth. Whereas infants sleep for approximately 14 hours/day at age 6 months, the average time spent asleep for 16-year-old adolescents is about 8 hours/day (Iglowstein, Jenni, Molinari, & Largo, 2003). Additionally, the duration of nighttime sleep increases early in infancy during nocturnal sleep consolidation, which is when children transition to sleeping mostly during the night. From that point, nighttime sleep decreases throughout development. There is also evidence that sleep duration is decreasing over time for equivalent age groups, such that our parents likely slept slightly more than we do when they were our age. This is attributed to later bedtimes in more recent cohorts (Iglowstein et al., 2003), whereas morning awakening times have remained fairly stable.

In addition to identifying patterns of normal sleep development, researchers have studied patterns of sleep disturbance that are common in childhood. Using multiple methods to identify problematic sleep behavior, including polysomnography (PSG, which includes measurements of the brain, eyes, muscles, heart, and respiration during sleep), actigraphy (measurement of rest/activity cycles), and subjective reports (e.g., sleep diaries, questionnaires), we have discovered that the impact of sleep problems on mood, performance, behavior, and health is

profound in childhood (Owens, 2008). Childhood sleep problems correlate with daytime sleepiness, moodiness, behavioral problems, and school and learning problems (Fallone et al., 2002). Similarly, adolescents with sleep problems earn poorer grades and are more likely to have internalizing and externalizing problems (Morrison et al., 1992). Researchers have also demonstrated links between child/adolescent sleep problems and psychological disorders, including autism, ADHD, bipolar disorder, depressive disorders, anxiety disorders, obsessive-compulsive disorder, stress-related disorders, and externalizing disorders (see Gregory and Sadeh, 2016, for a review of sleep and psychopathology). However, the direction of causality of these links is unclear, as discussed below.

There are a number of explanations for the relationships between sleep and other problems in childhood. First, Gregory and Sadeh (2016) note that nosological systems like the DSM-5 (APA, 2013) include sleep-related symptoms under many different disorders. Thus, researchers correlating sleep problems with psychiatric diagnoses would be likely to find relationships between sleep problems and those disorders under which sleep problems are listed as criteria. Likewise, when evaluating relationships using scales such as the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), sleep-related items are often included in syndrome scales for other problems, although sleep items are not part of the Externalizing Problems scale. Thus, some of the relationships between sleep problems and other problems may be artifactual due to overlapping symptoms.

However, causal explanations have also been proposed, and these have some support. Theories underlying these explanations center around the premise that the effects of sleep loss on behavior are related to compromised self-regulation, including attentional, emotional, and behavioral control (Goodnight et al., 2007). In support of this effect, a neuroimaging study

showed that sleep restriction in adults (18-30 years old) causes differences in brain functioning that may be related to emotional dysregulation. Specifically, restricting sleep for 35 hours increased amygdala reactivity in response to negative emotional stimuli and decreases communication between the amygdala and the prefrontal cortex (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). However, the evidence for effects of sleep restriction on children's behavioral functioning is more mixed. One questionnaire-based study found that early school start time (a form of restricted sleep) is correlated with more daytime sleepiness, impaired concentration, and inattention in 5th graders (Epstein, Chillag, & Lavie, 1998). Likewise, researchers have found acute sleep restriction to impair performance on cognitive tasks in children aged 10-14 years (Randazzo, Muehlbach, Schweitzer, & Walsh, 1998) and to increase inattention in 8- to 15-year-olds (Fallone et al., 2001). However, in a small sample of adolescents, Carskadon and Dement (1981) found that functioning was only impaired following full-night sleep deprivation, but not following 4-hour sleep restriction. Overall, the literature suggests that acute sleep deprivation is related to some level of impaired performance, although small amounts of sleep deprivation might be less of a problem in adolescence. Furthermore, it is likely that the effect of sleep deprivation on performance is due to brain-related changes.

Of course, it may also be the case that behavioral/psychological problems precede sleep problems in a causal framework. For instance, induced rumination in college students has been shown to have a detrimental effect on sleep quality, especially for individuals with a trait tendency to ruminate (Guastella & Moulds, 2005). Indeed, there is a strong body of literature supporting the notion that unwanted intrusive thoughts can bring about insomnia (Harvey, Tang, & Browning, 2005). Additionally, anecdotal reports further suggest that children with externalizing problems (e.g., oppositional defiance) may have later bedtimes simply due to their

refusal to "go to sleep." In these cases, it may be difficult to elucidate direction of effect because it is impossible to determine which set of problems began first. Instead, measuring both sleep and externalizing at two time points allows us to make inferences regarding cause and effect. Thus, for the present project, I used a longitudinal design with measurements of both sleep and externalizing at two time points to allow for a clearer explanation of potential effects.

Moderators of Sleep Effects

Whereas the relationship between sleep problems and multiple negative outcomes in youth has been demonstrated, potential moderators of these relationships have remained relatively unstudied. Although children with sleep problems are more likely to develop other problematic behaviors, many children with sleep problems do not experience other problems. Two commonly studied potential moderators of effects of sleep problems are socio-economic status (SES) and sex of child. For instance, sleep problems at age 2 years were found to be associated with externalizing problems at age 4, but only in boys (Belanger, Bernier, Simard, Desrosiers, & Carrier, 2015). However, in a sample of older children (age 8-10), sex did not emerge as a significant moderator between sleep and internalizing/externalizing problems (El-Sheikh, Kelly, Buckhalt, & Hinnant, 2010). Additionally, El Sheikh and colleagues (2010) found SES and race to moderate the relationship between sleep problems and externalizing. They suggested that minorities and individuals of lower SES likely face greater chronic stress, thus limiting their capacity to deal most effectively with disruptions in primary biological regulation systems such as sleep.

Another potential moderator of sleep effects that has not been thoroughly addressed is temperament. Beginning in infancy, temperamental traits such as reactivity are related to sleep quality (Carey, 1974; De Marcas, Soffer-Dudek, Dollberg, Bar-Haim, & Sadeh, 2015; Gartstein,

Potapova, & Hsu, 2014). Whereas most of this research relies on subjective reports from parents, 1-year-old infants' behavioral reactivity measured in a laboratory setting is also associated with objective sleep measures. De Marcas and colleagues (2015) found that the relationship between reactivity and sleep quality, measured via actigraphy, followed an inverted-U shape, indicating that both hyposensitive and hypersensitive infants are at risk for poor sleep quality. Although researchers have addressed relationships between sleep and temperament, the bulk of this research has been conducted in infants, and few studies have examined temperament as a moderator of other sleep effects, such as the relationship between sleep problems and externalizing behavior. Furthermore, evidence for longitudinal relationships between temperament and sleep is limited to studies of infants. Whereas sleep problems are related to temperament traits when sleep and temperament are measured concurrently, the degree to which early temperament traits predict later sleep problems is unknown. For the current study, I included measures of later sleep problems in order to evaluate the predictive ability of temperament traits for those problems.

The causal direction of the sleep-externalizing link throughout childhood development remains unclear, as does the influence of temperament as a moderator. In addition to the utilization of a longitudinal design, the present study included an examination of potential moderators measured in preschool-aged children. Multiple facets of reactivity, as well as negative emotionality and an overall difficult temperament, are related to sleep, and the relationship between sleep problems and later behavioral disturbances was found to be moderated by temperamental resistance to control (Goodnight et al., 2007). For the current study, I examined how temperament moderates longitudinal relationships between sleep and externalizing.

Typical developmental sleep trends have been studied extensively, but further study of outcomes related to poor sleep is warranted. Understanding sleep problems as a risk factor is crucial for clinical science, as some estimate that sleep problems may be as predictive of internalizing and externalizing behavior as other common environmental risk factors such as poor parenting and maternal depression (Reid et al., 2009). Also, research on potential moderators of these risk effects will help in identifying children who are at the greatest risk for later problems. As externalizing behavior is common across childhood and adolescence (Achenbach, 2017), understanding the contribution of sleep and other factors to the development of these problems is especially important.

Externalizing Problems

Researchers in child and adolescent psychology commonly distinguish between internalizing and externalizing problem behaviors (Achenbach, 2017). Broadly, internalizing behavior involves characteristics such as withdrawal, dysphoria, and anxiety, whereas externalizing behavior includes defiance, impulsivity, disruptiveness, aggression, antisocial features, and hyperactivity (Hinshaw, 1992). Although both externalizing and internalizing behavior are related to sleep problems, research suggests that the direction of relationships with sleep problems may differ between externalizing and internalizing (e.g., Bates et al., 2002; Gregory & O'Connor, 2002; Lavigne et al., 1999). For externalizing, sleep problems are hypothesized to be precursors to problem behavior due to factors such as compromised self-regulation (Goodnight et al., 2007). Conversely, internalizing problems more likely lead to sleep problems due to factors related to internalizing symptoms, such as nighttime fears and bedtime avoidance (Alfano, Zakem, Costa, Taylor, & Weems, 2009). Because the current study evaluated

early sleep problems as a risk factor for later externalizing problems, I will focus on behavioral problems within the externalizing domain.

Because externalizing encompasses a broad spectrum of problematic behavior, sub-categories have been suggested to differentiate between different types of externalizing behavior, including Hinshaw's (1992) differentiation between inattention-hyperactivity and aggression-conduct problems. Within the aggression-conduct problems domain, Loeber and Schmaling (1985) described different aspects of aggression including overt, covert, destructive, and nondestructive aggression. Overt behaviors include those that are directly confrontational (e.g., aggression, defiance), whereas covert behaviors are nonconfrontational in nature (e.g., stealing, lying). The overt-covert continuum is bisected by a destructive-nondestructive continuum (Frick et al., 1993), which describes the degree to which aggressive behaviors are outwardly damaging. These continua intersect to form a matrix of four types of aggression: 1) Covert-Destructive (e.g., property violations), 2) Covert-Nondestructive (e.g., status violations), 3) Overt-Destructive (e.g., physical aggression), and 4) Overt-Nondestructive (e.g., oppositional-defiance), which were found to represent valid classifications of aggression in Frick and colleagues' (1993) meta-analysis. Childhood-onset aggression is more likely to be severe and persistent, whereas adolescent-onset aggression is more likely to be temporary, normative, and peer-influenced (Moffitt, 1993).

For the present study, I primarily focused on externalizing as defined by Achenbach & Rescorla (2001), which is associated with sleep problems in children (Belanger et al., 2005; Gregory & O'Connor, 2002). The empirically-based CBCL's superfactor of Externalizing behaviors includes rule-breaking and aggression, which are two primarily overt forms of externalizing behavior. Notably, the CBCL also includes an empirically-based attention problems

scale, which measures symptoms related to Hinshaw's (1992) inattention-hyperactivity domain, but which is not included in the Externalizing superfactor. Furthermore, because the CBCL is completed by parents, it is likely that the resulting externalizing scores are indicative of children's observable, or overt, behaviors, rather than covert behaviors. Therefore, out of the four types of aggression defined by Frick and colleagues (1993), the current study primarily focuses on the two that are overt.

Risk Factors for Externalizing

The study of risk factors for externalizing problems is important given the long-term consequences of early externalizing behavior. In typically developing children, externalizing behavior decreases from childhood through adolescence (Bongers, Koot, van der Ende, & Verhulst, 2003; Brame, Nagin, & Tremblay, 2001; Leve, Kim, & Pears, 2005). Children with higher levels of externalizing problems, however, are more likely to perform poorly in academic settings, have conduct problems and attention-deficit/hyperactivity disorder (ADHD), experience social problems, and become involved in delinquent activity during childhood and adolescence (Frick & McMahon, 2013). They are also more likely to develop antisocial psychopathology and to be incarcerated as adults (Deater-Deckard et al., 1998; Frick & McMahon, 2013). Thus, researchers have made great efforts to identify potential risk factors for the development of externalizing problems. These include environmental factors such as poverty (Huston, McLoyd, & Coll, 1994), stressful life events (Abidin, Jenkins, & McGaughey, 1992), conflict at home (Abidin et al., 1992), and social rejection (Hymel, Rubing, Rowden, & LeMare, 1990), as well as genetic and temperamental factors (Burt, 2009; Deater-Deckard et al., 1998; DiLalla, 2002; Kendler, 2013). Additionally, parent factors including parenting behaviors (Lovejoy, Graczyk, O'Hare, & Neuman, 2000) and parental marital discord (Grych & Fincham, 2001) are associated

with externalizing problems. Although there are numerous ways to classify all of these types of risk factors, most fit into one of four categories: sociocultural factors, parenting/caregiving factors, peer influences, and child-related factors.

Using those four categories of risk factors, Deater-Deckard and colleagues (1998) examined the associations between several risk variables and externalizing problems in a large sample of 5- to 9-year-olds. They found that child-related factors uniquely explained up to 19% of the variance in externalizing problems, whereas peer influences accounted for 13%, and parenting/caregiving (6%) and sociocultural (4%) factors were not as strongly predictive of externalizing problems. Importantly, child-related factors in this study only included three variables: 1) sex of child; 2) temperamental resistance to external control; and 3) birth complications. The findings of this study are valuable because they add to the strong body of literature on childhood risk factors for externalizing problems, but they also highlight the importance of different classes of risk factors. However, weaknesses include the use of retrospective reporting of temperament and the correlational rather than longitudinal research design; these two weaknesses were addressed in the present study.

Longitudinal studies of risk factors for externalizing problems also suggest that multiple child-related factors are associated with externalizing in childhood/adolescence. Eisenberg and colleagues (2009) found that externalizing problems at age 10 years were predicted by low effortful control, high impulsivity, and high negative emotionality at age 6 years. Similarly, impulsivity at age 5 is associated with externalizing behavior at age 17 (Leve et al., 2005). Importantly, some results suggest that child-related risk factors interact with other factors, including parenting/caregiving and other child-related factors such as sex. In the study by Leve and colleagues, for instance, an interaction between impulsivity and maternal depression was

found only for boys, suggesting that maternal depression was only predictive of later externalizing for boys with high early impulsivity. For girls, age 5 impulsivity interacted with harsh parental discipline, such that harsh discipline predicted later externalizing for girls with high but not low early impulsivity. Harsh discipline similarly interacted with age 5 fear/shyness, such that harsh discipline predicted externalizing for girls with low but not high fear/shyness. Thus, it is possible that child-related factors such as low impulsivity or high fear/shyness could protect at-risk children from developing externalizing problems.

Clearly, the development of externalizing is multifactorial, as risk factors from various domains (e.g., child-related, parenting/caregiving) interact with each other to bring about adaptive or maladaptive behavior. As previously discussed, sleep problems represent a strong risk factor for externalizing problems, and both sleep problems (Atkinson et al., 1995; De Marcas et al., 2015; Gartstein et al., 2014; Owens-Stively et al., 1997) and externalizing problems (Eisenberg et al., 2009; Leve et al., 2005) are related to temperament traits. Additionally, at least one study has demonstrated that early sleep problems interact with temperamental resistance to control in the development of externalizing problems (Goodnight et al., 2007). However, some of the aforementioned challenges remain to be addressed. For the current study, I utilized a prospective longitudinal design in order to better elucidate relationships between temperament, sleep problems, and externalizing problems.

Temperament

Given the relationship between temperament and both sleep and externalizing, further discussion of temperament is warranted. Many theorists have defined temperament as stable traits that are observed early in life (e.g., H. Hill Goldsmith, Mary Rothbart, Alexander Thomas and Stella Chess, and Arnold Buss and Robert Plomin). Although there are important distinctions

between different temperament models, they also share some underlying assumptions. First, most theories assume that temperament traits are relatively consistent across development (Goldsmith et al., 1987). Although some temperament traits are more malleable than others early in life (Rothbart, 2011), most traits are fairly stable by the preschool years (Roberts & DelVecchio, 2000). Second, temperament traits constitute individual differences in the domains of activity, affectivity, attention, and self-regulation (Shiner, 2012). These domains are reflected across models. Third, the expression of temperament traits is influenced by both genetic and environmental factors (Shiner, 2012). Whereas historically temperament has been viewed as primarily biological in nature with environmental influences becoming more prominent in later ages, more recent conceptualizations emphasize the dynamic interactions between genetic and environmental influences beginning even before birth (Huizink, 2012).

Review of Major Temperament Models

Although major temperament theories share some basic assumptions, they emphasize different important aspects of child behavior. For instance, Goldsmith's conceptualization of temperament focuses heavily on emotional experiences and the expression of emotion (Goldsmith et al., 1987). This approach to temperament rests on the beliefs that basic emotions are present in very early infancy, and that temperament is a reflection of variability in the development of emotional systems. Thus, Goldsmith's temperament traits revolve around discrete emotions, including joy, interest, sadness, anger, and fear. These traits are reflected in the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith and Rothbart, 1996) and the Toddler Behavior Assessment Questionnaire (TBAQ; Goldsmith, 1996), which were both originally designed to measure temperament in terms of motor activity, anger, fearfulness, pleasure/joy, and interest/persistence.

Buss and Plomin's well-established temperament model emphasizes three dimensions (emotionality, activity, and sociability) of behavior that are inherited and that appear in early infancy (Goldsmith et al., 1987). Buss and Plomin also emphasized the importance of temperament traits as the foundation for personality traits, and thus disregarded infant traits (e.g., rhythmicity) that do not appear to be as salient in later stages of development. This model of temperament is similar to Goldsmith's in that they both emphasize the presence of temperament traits in early infancy, but differ in that Buss and Plomin more strongly emphasize the continuity of temperament traits. Furthermore, whereas each of Goldsmith's temperament traits represents a specific emotion, the approach of Buss and Plomin considers emotionality, as a whole, to be a single trait. Therefore, Goldsmith's model might be considered more sensitive to emotional differences in infants and children, whereas Buss and Plomin emphasize other individual differences, including activity and sociability, which are less related to the expression and regulation of emotion and more related to general behavioral regulation. Rothbart's and Thomas and Chess' models of temperament are primary to the present project, and will be discussed next in further detail.

Rothbart's model of temperament (Rothbart & Derryberry, 1981). Rothbart's model of temperament is hierarchical in nature, as she identified over 20 factors that combine to form three higher-order traits: surgency, negative affectivity, and effortful control. These temperament factors are consistent across age and reporter (i.e., self- vs. parent- or teacher-report), and they are also present in multiple cultures (Shiner, 2012). Rothbart's three primary temperament factors are made up of several sub-factors. The first primary factor, Surgency, sometimes called Surgency/Extraversion, is made up of four scales: Activity Level (gross motor activity), High Intensity Pleasure (positive affect in response to high-intensity stimuli often involving risk),

Positive Approach/Anticipation (excitement/positive affect to pleasurable activities), and reversed Shyness (low approach or discomfort with novel stimuli). Overall, children with high surgency have high activity levels and positive emotionality (Rothbart, Ahadi, & Evans, 2000). Importantly, some of these traits are associated with higher rates of externalizing but lower rates of internalizing in adolescents (Ormel et al., 2005). Out of the three temperament factors, there is the least support for a relationship between surgency and externalizing. Still, high surgency is associated with hyperactivity and aggression in kindergarten children (Berden, Keane, & Calkins, 2008) and externalizing behavior in early childhood (Merviele, De Clerq, De Fruyt, & Van Leeuwen, 2005). Additionally, similar traits such as novelty or sensation seeking are also risk factors for externalizing problems (Kuo, Chih, Soong, Yang, & Chen, 2004). Thus, children who are highly active, enjoy risk, and are not shy are more likely to show externalizing behaviors throughout development.

Rothbart's second primary factor, Negative Affectivity, also is made up of four scales: Anger/Frustration (negative responses to interruption of tasks/goals), Fear (negative affect in relation to pain, distress, or perceived threat), Sadness (negative affect in relation to loss or disappointment), and reversed Soothability (rate of recovery from distress, excitement, or general arousal). Children with high negative affectivity have more negative emotional experiences, and some of these traits have been linked with internalizing disorders, especially fear (Rothbart & Bates, 1998). Negative emotionality has also been shown to moderate the relationship between attentional control and externalizing behavior, such that attentional regulation more strongly predicts externalizing problems in elementary schoolers with high negative emotionality (Eisenberg et al., 2000). In 9- to 13-year-olds, the combination of high levels of negative affectivity and low levels of effortful control was found to represent a temperamental

vulnerability to externalizing, although negative affectivity alone was related to both internalizing and externalizing (Muris, Meesters, & Blijlevens, 2007).

The third primary temperament factor, Effortful Control, also is made up of four scales: Attentional Focusing (capacity to sustain attention on a task), Inhibitory Control (ability to plan and suppress impulses), Low Intensity Pleasure (pleasure derived from low-intensity stimuli), and Perceptual Sensitivity (detection of low-intensity environmental stimuli). Given the emphasis on attention and inhibition in this construct, it is not surprising that low effortful control consistently is related to ADHD (Bussing et al., 2003; Nigg et al., 2002; Rettew, Copeland, Stanger, & Hudziak, 2004). Likewise, there is a large body of literature linking effortful control more broadly to externalizing problems in childhood (e.g., Eisenberg et al., 2009). When assessing specific domains of effortful control, inhibitory control seems to be the domain most strongly related to externalizing behavior (Martel, Nikolas, & Nigg, 2007). Thus, it is likely that low inhibitory control plays a role in externalizing behaviors commonly seen in ADHD, although those behaviors alone are not sufficient to constitute the disorder.

Rothbart's strong emphasis on regulatory functions, which enable children to change their behavior to respond adequately to environmental changes, is important for the current project. As previously noted, low effortful control, high negative affectivity, and high surgency (although to a lesser degree) have been linked with externalizing (Eisenberg et al., 2009; Leve et al., 2005; Muris, Meesters, & Blijlevens, 2007). Similar regulatory traits (e.g., resistance to control) are related to sleep problems (Goodnight et al., 2007), although evidence for links between sleep problems and Rothbart's temperament traits is limited. Because these temperament traits are related to both externalizing and sleep problems, these aspects of temperament, measured at age 4

years, were considered in the present project as they relate to longitudinal relationships between sleep problems and externalizing behaviors.

Thomas and Chess' model of temperament (Thomas & Chess, 1970). Thomas and Chess derived their temperament dimensions from the well-known New York Longitudinal Study (NYLS), which was conducted from 1956 until 1988. Data from the NYLS yielded nine temperament characteristics: sensory threshold (i.e., level of stimulation evoking a reaction), activity level (i.e., physical activity), intensity (i.e. the energy level of a response), rhythmicity (i.e., predictability of behavior), adaptability (i.e., responses to environmental changes), mood (i.e., level of positive and negative emotion, approach/withdrawal (i.e., responses to novelty), persistence (i.e., length of time pursuing an activity), and distractibility (i.e., ability for external stimuli to change a child's behavior). Chess and Thomas (1984) asserted that, based on combinations of these traits, most children can be classified as either temperamentally easy, difficult, or slow to warm up. Easy children (~40% of children) are characterized by positive mood, predictable bodily functions, low to moderate response intensity, adaptability, and high approach to novel situations. Difficult children (~10% of children) have unpredictable/irregular bodily functions, have more intense reactions, withdraw rather than approach novel stimuli, are negative in mood, and are less adaptable to environmental changes. Slow to warm up children (~15% of children) typically are low in activity level, have a low intensity of reactions, have a somewhat negative mood, display withdrawal when first encountering novel stimuli, and adapt slowly.

More recently, Chess and Thomas' model has been questioned by researchers who have found that the original nine dimensions are not empirically distinct (de Pauw & Mervielde, 2010). However, most still agree that the dimensions represent clinically important temperament

dimensions, and some emphasize the potential for practical application with this model (Shiner, 2012). For instance, high activity level, negative emotionality, and difficult temperament appear to be associated with later problem behaviors, including disruptive behavior disorders and ADHD (Rettew & McKee, 2012). Conversely, temperamental inhibition (high levels of withdrawal) is indicative of internalizing problems (e.g., anxiety). These clinical markers have been utilized in the development of interventions based on temperament, which have been shown to decrease temperamental inhibition in preschool children (Kennedy, Rapee, & Edwards, 2009) and reduce disruptive behavior in school-aged children (McClowry, Snow, Tamis-LeMonda, & Rodriguez, 2010). The malleability of these temperament traits is of clinical interest because it suggests that intervention may be effective in altering risk factors for later problems. Furthermore, the identification of specific patterns of risk allows us to identify at-risk children early in development and thus provide earlier intervention. Chess and Thomas' model was used to evaluate temperament characteristics of children in the current study at age 5 years.

In sum, temperament traits related to sleep generally revolve around regulatory and reactivity domains; however, this research has primarily been conducted in infants. Temperamental correlates of externalizing problems, on the other hand, have been extensively studied in young children. Researchers have drawn clear associations between externalizing problems and effortful control, and specifically the domain of inhibitory control. Links have also been found between surgency and externalizing. Additionally, high negative affectivity in combination with low effortful control is characteristic of children with externalizing problems. Understanding these temperament factors as risks for externalizing behavior is clinically useful because it can guide the development of screening instruments to identify children at risk for externalizing problems. However, because not all children with difficult temperaments have

behavioral problems later in life, it is important to know what other factors might be important in predicting those behavioral problems.

Interactions between Temperament and Other Factors

Interactions between early temperament traits and other factors warrant further discussion. Although it is well-established that early temperament traits interact with environmental factors (e.g., parenting, daycare, social/peer experiences), in the development of externalizing behavior (Bates & Pettit, 2007), research on interactions between temperament traits and sleep problems is limited. Because sleep problems are associated with several temperamental traits (e.g., reactivity, negative emotionality, difficult temperament style), and externalizing behavior is also associated with overlapping temperamental traits, temperament traits might moderate the longitudinal relationship between sleep problems and externalizing. In regard to temperament-environment interactions, Bates and colleagues (1998) found that maternal retrospective reports of infant temperamental resistance to control interacted with restrictive parenting in the development of externalizing problems at ages 7-11 years. Likewise, Hagekull and Bohlin (1995) found that infants' temperamental manageability interacted with daycare quality in the development of aggressive behavior at age 4 years (this was not a longitudinal study). Studies like these provide evidence that temperament interacts with the environment (e.g., parenting, daycare) in the development of externalizing behavior. Understanding interaction effects with different variables (e.g., sleep) and at different ages (e.g., preschool through adolescence) is crucial in further understanding potential developmental risks.

Because temperament interacts with environmental factors, researchers have suggested reasons that children with certain temperamental traits might respond differently to those factors. Thomas and Chess (1977) explained these interactions in the context of "goodness of fit,"

suggesting that behavior problems arise when a child's temperament is less adaptive given the expectations in a particular environment. This notion would suggest that children who are resistant to control, like those in the Bates and colleagues (1998) study, might fare better in a household characterized by high levels of parental restriction and control. In fact, the significant interaction effect in that study supports this notion of "goodness of fit." However, it is also theoretically possible that children with higher resistance to control might fare worse in restrictive and controlling environments specifically because they resist control. Kochanska (1995) suggested that lower levels of parental control provide a greater opportunity for children to autonomously internalize social limits. Although the results from Bates and colleagues (1998) indicate that low-resistant children might be better able to internalize those limits, it is possible that high parental control is not universally related to fewer externalizing problems.

As previously discussed, temperament traits related to externalizing behavior include low effortful control, especially in combination with high negative affectivity. Moreover, associations between regulatory traits and both externalizing behavior (Eisenberg et al., 2009; Leve et al., 2005) and sleep problems (Goodnight et al., 2007) highlight the potential shared temperamental antecedents of externalizing and sleep problems. However, the mechanisms for relationships between temperament and behavioral problems are not fully understood. Whereas the stability of temperament traits may be a direct cause for these relationships, indirect effects (e.g., through a child's difficult interactions with parents) also contribute to problematic outcomes for certain temperament traits (Rothbart & Bates, 1998).

Only one published study to date has considered the impact of sleep problems on the relationship between temperament and externalizing behavior. Goodnight and colleagues (2007) utilized multi-site data from children who were evaluated at ages 5, 6, 7, 8, and 9 years to assess

interactions between sleep and temperament. Sleep and externalizing data were collected each year, and mothers rated their children's infant temperament retrospectively when children were 5 years old. Using a growth curve model analysis, Goodnight and colleagues found that, overall, temperamental resistance to control was positively associated with both sleep problems and externalizing behavior at age 5 years. However, they also found that resistance to control moderated the relationship between sleep problems and externalizing from ages 5-9 years, such that children who were high in resistance to control and also had sleep problems were more likely to exhibit externalizing behavior. In explaining this interaction, they noted that the direction of effect remains unclear. It could be that sleep problems produced externalizing problems in this group of highly resistant children, but externalizing problems may also have produced sleep problems more strongly in this group. The current study will address some limitations of this study, including the use of retrospective reporting of temperament. The validity of these reports may have been compromised if, for instance, mothers of difficult children tended to recall their children as being more temperamentally difficult during infancy.

Together, the theoretical and empirical literature point to temperament as an important determinant of behavior when studied concurrently and longitudinally. However, it is also clear that environmental and other factors (e.g., sleep) interact with temperament traits in complex ways. Research on interaction effects between temperament and sleep is limited, but the study by Goodnight and colleagues (2007) provides early evidence that certain temperament traits might exacerbate long-term negative effects of early sleep problems, including the development of externalizing behavior.

Genetics of Externalizing Behavior

Heritability

The current study adds to the existing literature by considering genetic influences on sleep problems, temperament, and externalizing problems. There is broad support for a genetic influence on externalizing behavior from both the behavioral and molecular genetics literatures. Behavior genetics research suggests that siblings with more genetic relatedness (e.g., monozygotic (MZ) twins) , compared to siblings with less genetic relatedness (e.g., dizygotic (DZ) twins, non-twin siblings), share more similar externalizing characteristics (Bartels et al., 2003; Burt et al., 2009; Chen et al., 2015; DiLalla, 2002; Kendler, 2013; Spatola et al., 2007). Heritability estimates are derived by comparing the correlation between MZ twins and the correlation between DZ twins. By subtracting the DZ correlation (r_{DZ}) from the MZ correlation (r_{MZ}), and doubling that difference, we obtain a broad estimate of heritability (h^2). This estimate represents the degree to which a particular trait is influenced by genetics. Research on twins suggests that externalizing behavior is heritable. Overall, estimates of the heritability of externalizing behavior in children and adolescents range from below 20% to over 75%, depending on several factors. First, heritability estimates frequently differ based upon the definition of externalizing behavior that is used (DiLalla, 2002). The most consistent finding is that overt aggression is typically found to be more heritable than rule-breaking, which has stronger shared environmental influences (Eley, Lichtenstein, & Moffitt, 2003; Hudziak et al., 2003; Tackett et al., 2005). Because the current study utilized CBCL-rated externalizing behavior, which includes both aggression and rule-breaking, the consideration of genetics for this project is important.

Heritability estimates for externalizing behavior also differ between informants. Because externalizing behavior is typically measured using rating forms (e.g., CBCL), biases from parents,

teachers, and children need to be considered. These biases lead to different estimates of heritability for each type of rater. In a sample of 908 adolescent twin pairs (age 10-18 years) from the Beijing Twin Study (Chen, Yu, Zhang, Li, & McGue, 2015), heritability of externalizing was found to be strongest for child-report (42%), followed by teacher-report (40%) and then parent-report (34%). This pattern of results might indicate that because parents of twins typically have frequent contact with both twins, they are more attuned to differences between MZ twins, which could potentially result in lower heritability estimates. However, in a meta-analysis of studies conducted mainly in the United States, Burt (2009) found that the heritability of externalizing behavior was highest for parent-report (62%), followed by child-report (50%) and then teacher-report (41%). This pattern of results would suggest that parents in the United States are either rating MZ twins more similarly, or rating DZ twins less similarly, than parents in the Beijing Twin Study. Regardless, the overall genetic influence on externalizing behavior in adolescents appears to be higher when using parent ratings in the United States, suggesting that cultural context influences the degree to which externalizing behavior is heritable. Thus, considering both rater bias and cultural context is important when measuring heritability.

The presence of a significant heritable component to externalizing behavior suggests that phenotypic variation is at least in part driven by genotypic variation. Although heritability estimates help in understanding the degree to which a phenotypic trait has underlying genotypic influences, heritability provides no information about which specific genes are involved. Molecular genetic research, which focuses on the structure and function of specific genes, has only begun to disentangle the specific genetic differences that underlie externalizing behavior.

Gene-Environment Interactions (GxE)

Because some specific genetic variations affect externalizing behavior in children and adolescents, discussion of mechanisms of these specific genetic effects is warranted. Since Caspi and colleagues (2003) found that variations in the serotonin transporter gene (5-HTTLPR; discussed in detail below) affect individuals' risk for depression in response to stressful life events, hundreds of studies have investigated the relationship between 5-HTTLPR and depression. This research has emphasized the potential for different responses to stressful life events depending on genotype, a phenomenon called gene-environment interaction (GxE). GxE studies are important because they shed light on potential etiologies of psychological disorders and help to identify individuals who are more susceptible to environmental risk factors (Manuck & McCaffery, 2014). Most GxE research conforms to a diathesis-stress model, which focuses on genetic vulnerability, or diathesis, and environmental stressors (e.g., Caspi et al., 2003). In describing diathesis-stress in the development of schizophrenia, McGue and Gottesman (1989) suggested that genetic makeup is a determinant of one's vulnerability level. Thus, in a diathesis-stress GxE model, genetic vulnerabilities interact with environmental stressors such that individuals with a genetic risk experience more adverse outcomes after exposure to life stressors (Gottesman, 1991). A complementary GxE model, the vantage sensitivity model, focuses on the moderation of positive environmental effects by genetic variation (Manuck & McCaffery, 2014). In vantage sensitivity GxE, genetic variations interact with environmental factors such that positive environments have more positive effects in individuals with a sensitivity genotype. Vantage sensitivity has found support from studies of the dopamine receptor D4 gene (DRD4; discussed below), which suggest that children with a "risk" variant are more responsive to effects

of parent interventions, compared to children with other variants (Bakermans-Kranenburg & van IJzendoorn, 2008).

Taking from both the diathesis-stress and vantage sensitivity models, differential susceptibility models of GxE emphasize that genetic variants are not always exclusively positive or negative, but sometimes confer malleability. Whereas the diathesis-stress model emphasizes how individuals with a genetic vulnerability are negatively impacted by environmental stressors, differential susceptibility posits that children are affected by the environment "for better *and* for worse" (Belsky et al., 2007). Researchers have described this phenomenon through a botanical analogy, suggesting that some genetic features result in "dandelion" characteristics, whereas others result in "orchid" features. "Dandelion children" are resistant to environmental effects, both positive and negative. Like the dandelion flower, these children are relatively resistant to adversity, and also receive little increased advantage in supportive environments. Conversely, "orchid children" are vulnerable to positive and negative environmental influences. Thus, in adverse environments, these children will be negatively impacted, much like an orchid plant with insufficient water or sunlight. However, in supportive environments, orchid children thrive and outperform their dandelion counterparts (Ellis, Boyce, Belsky, Bakenmans-Kranenburg, & van IJzendoorn, 2011). According to this model, the range of potential outcomes is wider for orchid children, whereas the resistance of dandelion children narrows their potential range of outcomes. In this sense, people with one specific genetic variation are susceptible to both positive and negative effects, whereas other alleles of the same gene may confer a resistance to environmental influence. Findings related to differential susceptibility to dopamine and serotonin functioning are discussed below.

Dopamine receptor D4 (DRD4). A considerable body of research suggests that genes related to dopaminergic neurotransmission may be important in the development of externalizing behavior. The dopamine receptor D4 (DRD4) is located in widespread prefrontal and subcortical brain regions, which are associated with cognitive processes including reward sensitivity, emotion processing, and complex thinking skills (Oak et al., 2010). Variations in DRD4, which is functionally related to central nervous system binding of dopamine (Plomin & Rutter, 1998), have been implicated in hyperactivity/impulsivity (Banaschewski et al. 2010), addictive behavior (McGeary et al., 2007), novelty-seeking (Ray et al., 2009), aggression (DiLalla Elam, & Smolen, 2009; Farbiash et al., 2014), oppositional defiant disorder (Kirley et al., 2004), depressive/mood disorders (Lopez Leon et al., 2005), and difficult temperament characteristics (DiLalla et al., 2009).

Research suggests that the number of "repeats" in the DRD4 gene is important, and the presence of 7 repeats (repeats range from 2 to 11) is related to less efficient binding of dopamine in Caucasians, compared to other DRD4 variations (Plomin & Rutter, 1998). The 7-repeat (7R) allele has most consistently been found to place individuals at risk for problem behavior (Jiang et al., 2013). Additionally, children with the DRD4-7R allele are found to be more susceptible to effects of early environmental stressors on later externalizing behavior (Bakermans-Kranenburg & van IJzendoorn, 2006; DiLalla et al., 2015), suggesting that DRD4-7R might function as a vulnerability factor that increases children's sensitivity to stressful life events. It is also important to consider differences in risk effects based on the population being studied, as the 2-repeat variation has been shown to transmit comparable risk in Asian populations (Jiang et al., 2013).

Support for links between DRD4 and externalizing behavior is in line with GxE hypotheses. Bakermans-Kranenburg and van IJzendoorn (2006) found that infants with exposure

to insensitive maternal care at age 10 months showed more externalizing behaviors at age 3 years if they carried the DRD4-7R allele. Maternal insensitivity did not impact externalizing behavior in children with other DRD4 allele repeats. These findings were replicated and extended by Windhorst and colleagues (2014), who used structural equation modeling to evaluate relationships between maternal insensitivity and externalizing measured at multiple time points between ages 18 months and 5 years. They found that the overall effect of maternal insensitivity on later externalizing problems was only statistically significant in DRD4-7R carriers.

Importantly, the effects of DRD4-7R do not appear to be universally negative, and researchers have demonstrated differential susceptibility effects with DRD4-7R. Positive effects of the DRD4-7R allele were demonstrated by Bakermans-Kranenburg and colleagues (2008), who found that DRD4-7R carriers were more responsive to an early intervention aimed at decreasing externalizing behaviors in 1- to 3-year-old children. Results from that study suggest that DRD4-7R carriers were more responsive than non-DRD4-7R carriers to changes in maternal disciplinary behaviors, specifically. Likewise, DiLalla and colleagues (2015) found that children with at least one DRD4-7R allele were differentially affected by peer victimization; these children, compared to children without the DRD4 risk allele, had fewer externalizing behaviors if they experienced little to no victimization, but had more externalizing behaviors if they experienced high levels of victimization. Together, these studies provide some evidence for differential susceptibility related to DRD4-7R.

Serotonin Transporter Promoter Region (5-HTTLPR). Another genetic variation that has been extensively studied in relation to behavioral problems is a functional polymorphism of the serotonin transporter gene (5-HTTLPR). Since the identification of the polymorphism in the 1990's (Heils et al., 1995), hundreds of studies have investigated 5-HTTLPR, its role in the

central nervous system, and human behavioral correlates. 5-HTTLPR appears to affect serotonin functioning in the human amygdala, a brain structure that is critical for fear and emotional processing (LeDoux, 2000). Researchers have been motivated to examine 5-HTTLPR because several psychopharmacological agents target serotonin functioning to treat psychiatric disorders, including depression and anxiety disorders (Li & Lee, 2014). However, the role of serotonin functioning and 5-HTTLPR in other behavioral problems, including externalizing behavior, has also been examined (discussed below).

5-HTTLPR is commonly identified by two variations: short (S) and long (L). These variations correspond to the number of "repeats" within the region. 5-HTTLPR consists of a repetitive sequence of base pairs on the human chromosome 17q11.1–q12 (Nakamura, Ueno, Sano, & Tanabe, 2000). The short (S) allele contains 14 repeats, whereas the long (L) allele contains 16 repeats (Lesch et al., 1996). The role of 5-HTTLPR variants in emotional processing is supported by evidence that carriers of the S allele have greater reactivity to emotional stimuli (Munafo, Brown, & Hariri, 2008). Functionally, this hyper-reactivity in the amygdala is proposed to be due to weakened serotonin transporter binding (Munafo et al., 2008). There are also two forms of the L allele, L_G and L_A. L_G has been shown to function similarly to the S allele (Hu et al., 2006; Uher & McGuffin, 2008). Therefore, individuals with the L_A/L_A genotype, which is associated with greater serotonin binding (Praschak-Reider et al., 2005), are generally compared to individuals who are heterozygous for 5-HTTTLPR (carriers of either L_G or S) or homozygous for L_G or S (e.g., Kaufman et al., 2006).

Specific to 5-HTTLPR, meta-analyses of GxE studies have provided mixed results, with some finding no overall GxE (Risch et al., 2009, Munafo et al., 2009). In a meta-analysis of 14 studies, Risch and colleagues (2009) found that although the number of stressful life events was

associated with depression, neither 5-HTTLPR nor the interaction between 5-HTTLPR and stressful life events were significantly related to depression. The study by Munafo and colleagues (2009) also included 14 studies and failed to detect a significant interaction effect. The Munafo and colleagues (2009) analysis shared some studies with the Risch and colleagues (2009) analysis, but also included some different studies. However, another meta-analysis which included 56 studies suggested a positive overall GxE (Karg, Burmeister, Shedden, & Sen, 2011), although this study has been criticized for several methodological concerns (Duncan & Keller, 2011). The most notable concern was regarding Karg and colleagues' (2011) inclusion criteria. They included several replication studies with broader definitions of "stressors," studies using several different outcome measurements including both physical and mental distress, and studies using different statistical procedures than the original study by Caspi and colleagues (2003). The differences between these meta-analyses suggest that a GxE interaction might only be detectable when using more relaxed criteria and multiple predictor and outcome variables.

Although the bulk of published 5-HTTLPR studies have revolved around problems within the internalizing spectrum, because of relationships between 5-HTTLPR, amygdala functioning, and emotional regulation, 5-HTTLPR may be related to other problems as well, including externalizing. The limited evidence base for links between 5-HTTLPR and externalizing suggests the presence of both direct and GxE effects. The S allele is found to be overrepresented in individuals who exhibit aggression, violence, drug use, and novelty seeking temperament (Gerra, Garofano, Castaldini, & Donnini, 2005; Retz, Retz-Junginger, Supprian, Thome, & Rösler, 2004). Additionally, the L_G allele, which functions similarly to the S allele, was implicated by findings that 5- to 15-year-old children with either S/S, S/L_G, or L_G/L_G genotypes were more likely to be aggressive than L_A/L_A children (Beitchman et al., 2006).

Interaction effects have also been found between 5-HTTLPR genotype and several other factors, including SES, sex, early institutional care, and maternal unresponsiveness (Aslund et al., 2013; Brett et al., 2015; Davies & Cicchetti, 2014, Hankin et al., 2001). For instance, Hankin and colleagues (2001) found evidence for differential susceptibility moderated by 5-HTTLPR in children aged 9-15 years. Specifically, they found that genetically susceptible children (defined in this study as homozygous S) were more likely to show low levels of positive affect when experiencing unsupportive and negative parenting, and high levels of positive affect when experiencing supportive and positive parenting. Importantly, these effects are not consistently replicated (e.g., Beitchman et al., 2003; Sakai et al., 2007), and the S allele is not consistently found to place individuals at greatest risk. For example, for a sample of 2-year-old children, Davies and Cicchetti (2014) found that the L/L genotype was most sensitive to low maternal unresponsiveness.

Overall, the evidence for specific geneticn effects on externalizing is strongest for DRD4, whereas results are mixed with respect to 5-HTTLPR. Because the development of externalizing behavior is multifactorial and polygenetic, it is likely that both DRD4 and 5-HTTLPR interact with the environment and with other child-related factors (e.g., temperament) to bring about externalizing behavior. Indeed, Hohmann and colleagues (2009) found that 15-year-olds with DRD4-7R had higher levels of externalizing behavior than carriers of other variants, but that those who carried both DRD-7R and two copies of the 5-HTTLPR short allele had the highest aggression scores. This gene-gene interaction, or epistatic effect, highlights the polygenetic nature of externalizing behavior (there are certainly many, many more). For the current study, I examined the contributions of DRD4 and 5-HTTLPR to externalizing behavior.

Current Study

Overall, there is evidence for relationships between sleep problems, externalizing behavior, and temperament traits in children. The relationship between sleep problems and externalizing is well-established, although the bulk of this research has examined sleep and externalizing concurrently, rather than longitudinally. The few longitudinal examinations (e.g.,Gregory et al., 2004; Gregory & O'Connor, 2002) show that early sleep problems are related to externalizing problems in later childhood and adolescence, but these studies did not include examination of potential moderators of these effects. Given the high prevalence of sleep problems in childhood (Owens, 2008), it is important to evaluate other variables that increase or decrease the risk of developing externalizing behaviors in children with early sleep problems.

Given the multiple relationships between temperament traits and both sleep and externalizing, it is possible that temperament moderates the longitudinal relationship between sleep problems and externalizing. Although most research on links between temperament and sleep is limited to studies of infants, sleep problems appear to be related to temperament traits including negative emotionality and overall difficult temperament, in addition to regulatory traits such as rhythmicity (Atkinson, Vetere, & Grayson, 1995; Owens-Stively et al., 1997; Schaefer, 1990). Likewise, externalizing behavior is associated with activity level, negative emotionality, and overall difficult temperament (from Thomas & Chess' model), as well as surgency, effortful control, and negative affectivity (from Rothbart's model). Despite these overlapping temperamental correlates of sleep and externalizing, research on the moderating effect of temperament is scarce.

Goodnight and colleagues (2007) found that temperamental resistance to control moderated the relationship between sleep problems and externalizing behavior between ages 5-9

years, such that this relationship was stronger for children with high resistance to control. A major limitation of that study was the use of retrospective reports of temperament, which may have introduced bias into the results. For the current study, I included similar hypotheses to those from Goodnight and colleagues, but I utilized a prospective longitudinal design with temperament traits measured when children were preschool-aged.

In evaluating the multiple risk factors for externalizing problems, genetic factors are also important to consider. There is overwhelming evidence suggesting that externalizing behavior is heritable. However, although EEG patterns in normal sleep traits were found to be significantly heritable (Ambrosius et al., 2008; De Gennaro et al., 2008), research has not thoroughly addressed the heritability of problematic sleep behaviors. In evaluating the relationship between specific genetic variations and behavioral and emotional functioning, researchers have largely focused on genes related to serotonergic (e.g., 5-HTTLPR) and dopaminergic (e.g., DRD4) neurotransmission. Although the genetic contribution to externalizing behavior almost certainly involves more than two genes, research to date suggests that both 5-HTTLPR and DRD4 are related to problem behavior. Further, 5-HTTLPR and DRD4 variants likely interact with environmental and other factors through diathesis-stress or differential susceptibility models, and possibly interact with each other (epistatic effects). For the current study, I utilized a prospective longitudinal design with a community sample of twins tested at ages 4 and 5 years, and again between ages 7-13 years. I examined longitudinal associations between sleep problems and externalizing problems, as well as ways in which variants of 5-HTTLPR and DRD4 interact with sleep problems in the development of externalizing behavior.

Hypotheses

Hypothesis 1: Heritability of Sleep Problems

Given the biological underpinnings of sleep problems, as well as findings that normal sleep patterns are heritable, sleep problems measured via parent-report questionnaires were expected to be significantly heritable in 4- to 13-year-old children. Sleep problems measured via self-report questionnaires were expected to be significantly heritable in 7- to 13-year-old children.

Hypothesis 2: Longitudinal Effects

A primary goal of this project was to evaluate longitudinal relationships between sleep problems, externalizing problems, and temperament traits. For Hypothesis 2, externalizing problems were used as the dependent variable. Hypothesis 2a examined the stability of externalizing problems over time. Hypotheses 2b and 2c involved exploration of longitudinal relationships between sleep problems, temperament, and externalizing problems (see Figure 1).

Hypothesis 2a. Externalizing problems were expected to be stable over time.

Hypothesis 2b. Early sleep problems (age 5) were expected to be statistically predictive of later externalizing behavior (follow-up ages 7-13).

Hypothesis 2c. Early temperament at age 5 years was expected to be statistically predictive of later externalizing behavior (follow-up ages 7-13). In line with the existing literature (discussed above), age 5 negative emotionality was expected to be positively statistically predictive of follow-up externalizing behavior.

Hypothesis 3: Moderator Models

Given the multifactorial nature of the development of externalizing problems, examination of potential moderators of risk effects is warranted. Thus, hypothesis 3a examined the degree to

which temperament traits affect the relationship between sleep and externalizing. Likewise, hypothesis 3b used genotype as a potential moderator of those effects.

Hypothesis 3a. Temperament was expected to moderate the relationship between early sleep problems and later externalizing problems. Specifically, high negative emotionality (measured at age 5) was expected to interact with age 5 sleep problems to predict follow-up externalizing. Although multiple temperament traits may be associated with externalizing problems, negative emotionality was chosen because of prior research with similar samples suggesting relationships to externalizing (Bersted and DiLalla, 2016), as well as because of the continuous nature of the variable (compared to dichotomous difficult vs. non-difficult temperament comparisons).

Hypothesis 3b. Genotype also was expected to moderate the relationship between sleep and externalizing. This moderation effect was examined in three ways. First, 5-HTTLPR genotype was expected to interact with age 5 sleep problems to predict follow-up externalizing. Children with sleep problems and at least one S or L_G allele were expected to have more externalizing problems, compared to children with sleep problems but without an S or L_G allele. Second, DRD4 genotype was expected to interact with age 5 sleep problems to predict externalizing. Children with sleep problems and the DRD4-7R variant were expected to have more externalizing problems, compared to children with sleep problems but without the 7R variant. Last, 5-HTTLPR genotype and DRD4 genotype were combined into an overall genetic risk score based on the number of "risk" alleles (ranging from 0-4). "Risk" alleles were defined as S/L_G and 7R, for 5-HTTLPR and DRD4, respectively. This overall risk score was expected to interact with age 5 sleep problems to predict externalizing.

CHAPTER 3

METHODS

Participants

Participants for the current study were drawn from a cohort of children from the IRB-approved, longitudinal Southern Illinois Twins/Triplets and Siblings Study (SITSS; DiLalla, Bersted, & Gheyara, 2013). SITSS participants are tested annually between ages 1 to 5 years, within 1 month (at ages 1-4) or 2 months (at age 5) of their birthdays. For the present study, participants included children with previous data within the SITSS database who participated in 5-year-old testing and who would be between ages 7-13 years at follow-up. This potential sample included 170 children (82 families; 74 boys and 96 girls). The final sample for this study included 93 of those children (45 families; 34 boys and 58 girls).

Using Optimal Design software (Raudenbush et al., 2011), statistical power was assessed for this study with nesting at the family level and with 93 participants. Under the assumptions of moderate effect sizes ($\delta = .50$), modest family-level variability ($\sigma^2 = .05$), with 5% of outcome variance explained at the family level, 50% of variance in outcomes predictable from predictor measures, and $\alpha = .05$, power is estimated at .88 to detect intervention effects. Assuming more conservative intervention effects ($\delta = .20$), power is estimated at .25 to detect intervention effects.

For the study sample at follow-up, the median family income was $65,000 to $70,000. 7% of mothers had a high school degree, 9% had some college, 56% had a college degree, and 27% had some form of graduate schooling. 38% of fathers had a high school degree, 5% had some college, 29% had a college degree, and 16% had some form of graduate schooling.

Children were recruited for the original study through several methods, including locally posted flyers, newspaper birth announcements, daycare recruitment, and word of mouth. For the current study, families were contacted via email, mail, and/or phone and asked to participate in this follow-up study (discussed below)

Procedure

The current study utilized a prospective longitudinal design, and data collected for this project were analyzed along with archival data from SITSS. The archival data were collected from preschool-aged children, whereas the new data for this study were collected from school-aged children. Children were eligible for participation in the follow-up data collection if they: 1) were born between July 2005 and August 2009 (to obtain a follow-up sample between ages 7-13); 2) participated in 5-year-old testing at SITSS; and 3) have genetic information (5-HTTLPR and DRD4) within the SITSS database. After all eligible children were identified, those families were contacted via email and asked to participate in the current study (see Appendix A). Families who did not respond within two weeks were contacted again by email, and families that did not respond to the round of emails after another two weeks were contacted by mail (see Appendix B). Families who did not respond within two weeks to the mail contact were contacted by phone (see Appendix C).

All participating families were compensated with ten dollars, split between the two twins in each family (total compensation was fifteen dollars for triplet families). Each child was given five dollars after the testing session and also small gifts including a SITSS baseball hat, a book, and a free food coupon.

Data collection took place in the SITSS laboratory. The measures used for this project were included as part of a larger battery of tests that were administered during an approximately

1-hour-long testing session. Upon arriving at the laboratory, parents provided informed consent (see Appendix D) and children provided assent (see Appendix E). Families were given information about the current study and that the only potential risk was mild anxiety, which was likely during a mood induction procedure (not included in the present study). During the testing session, children were administered questionnaires by trained research assistants while parents completed questionnaires independently. Test measures were administered in three separate rooms (one for each child, one for parents), and privacy was ensured by softly playing an "Ocean Sounds" track in the parent area. For the Likert-style items on the Children's Report of Sleep Patterns (CRSP; Meltzer et al., 2013), the research assistant read items aloud while children provided responses via an "answer card." The answer card (see Appendix F) contained rating descriptions (e.g., not very often, usually) and pictures to which children pointed as the research assistant read through the questionnaires and marked children's responses. Thus, instead of asking the child to tell the research assistant their answers to each item, the answer card allowed children to respond by pointing to pictures corresponding with item ratings. Additionally, the CRSP includes two initial practice items which allowed for the research assistant to ensure adequate understanding of the procedure by children. The answer card method was successfully used previously with this age group, and was chosen for this project due to the sensitive nature of some questionnaire items (e.g., those involving bed-wetting).

Measures

Demographic Information

During previous testing (ages 1-5), all families completed a family information questionnaire (see Appendix G) assessing general demographic information including race, family structure, family income, and parent age, occupation, and education level. Families also

completed the family information questionnaire as part of current testing. SES at each time point was measured by combining five demographic measures. First, parents' education levels were rated on a 5-point scale (1 = no high school degree; 2 = high school degree; 3 = technical certificate; 4 – college degree; 5 = advanced training beyond a college degree). Next, parents' occupations were rated on a seven-point scale of occupation categories using the Hollingshead scheme (Bonjean, Hill, & McLemore, 1967). An occupation rating of one indicates a higher executive, proprietor, or major professional including professors and medical doctors or major business or land owners. An occupation rating of seven indicates an unskilled labor position such as food service. Last, family income was rated on a 19-point scale from "$0 to $5,000" to "over $90,000". The two parent education ratings, two parent occupation ratings (inverted scores), and family income ratings were transformed into z-scores and averaged to obtain an overall SES score. Because SES scores may have changed over time, I calculated a change score by subtracting the SES scores from time 1 (age 4 or 5) from the most recent SES scores.

Genotype

Buccal cell collection for the current study was performed between ages 1-5 years (varies by child). Buccal cells were collected by swabbing the insides of children's cheeks three times over the course of one testing session to ensure adequate DNA collection. Parents were instructed to rub the swab on the inside of their children's cheeks for approximately 20 seconds. Cheek swabs were collected at the beginning of the testing session, after the first twin completed testing, and after the second twin completed testing. The DNA samples were labeled with participant numbers and frozen until they were sent to one of two sites for analysis. Genotyping with respect to 5-HTTLPR and DRD4 followed standard protocol (Anchordoquy, McGeary, Liu, Krauter, & Smolen, 2003). DNA extraction was completed using standard salting out and alcohol

precipitation methods before being resuspended in 0.5 of 50mM Tris-EDTA, pH 8.0. The working samples of DNA were concentrated at 20ng/µl. The analysis of DRD4 and 5-HTTLPR were completed together in a single multiplex reaction. The L_A/L_G determination was completed in a secondary reaction. The polymerase chain reaction (PCR) amplification procedure was completed using a solution including 1 µl of genomic DNA, 200 µM deoxynucleotides with 7-deaza-2'deoxyGTP (Roche Applied Science, Indianapolis, IN), 10 % DMSO, forward and reverse primers, 1 unit of AmpliTaq Gold® polymerase (ABI), and 1x PCR buffer in a total volume of 20 µl. After amplification, the DNA samples were analyzed using electrophoresis with an ABI Prism® 3130 xl DNA sequencer. Allele sizes were scored independently by two investigators at the site where these analyses were performed and disagreements were reviewed and re-run if necessary (Anchordoquy et al., 2003).

For the current study, children's genotypes were coded in several ways in line with existing literature (discussed above). For 5-HTTTLPR, children's genotypes were coded as "risk" if children possessed at least one S or L_G allele (Kaufman et al., 2006; Praschak-Reider et al., 2005). For DRD4, children's genotypes were coded as "risk" if the children possessed at least one DRD4-7R allele (Bakermans-Kranenburg & van IJzendoorn, 2006; DiLalla et al., 2015; Jiang et al., 2013). Additionally, a combined genetic risk score was calculated by summing the risk alleles from 5-HTTLPR and DRD4, resulting in potential scores between 0 (no risk alleles) and 4 (2 risk alleles for both genes, so 4 total risk alleles).

Externalizing Symptoms

Externalizing symptoms were measured using the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) and the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997; 1999). The CBCL is a parent-report questionnaire used as a screening instrument for

emotional and behavioral problems in 4- to 18-year-old children. The CBCL was used with 5-year-old children for this study due to minimal changes between old and new forms (old forms included 4- and 5-year-olds), as well as the use of a compatibility scoring system. It includes 120 problem items that are rated by parents on a 3-point scale based on the presence of the behavior during the past 6 months (0 = not true, 1 = somewhat or sometimes true, 2 = very or often true). Empirically-derived scales on the CBCL include an internalizing broadband score and an externalizing broadband score; the externalizing broadband score were used for the current study. The Externalizing Problems scale includes the Rule-Breaking (e.g., lying or cheating, runs away from home, steals outside the home) and Aggressive Behavior (e.g., temper tantrums or hot temper, argues a lot, physically attacks people) sub-scales. The CBCL was administered to parents of the current sample when children were tested at age 5, and was re-administered to parents during follow-up testing.

CBCL scales were originally derived from factor analyses (Achenbach & Rescorla, 2001). Test-retest reliability (r = .92) and internal consistency (Cronbach's alpha = .94) are excellent for the Externalizing superfactor (Achenbach & Rescorla, 2001). This factor structure of the CBCL is also reliable in several cultural groups, including in American, Australian, Chinese, Dutch, English, and Israeli children (Auerbach & Lerner, 1991; Dedrick, Greenbaum, Friedman, Wetherington, & Knoff, 1997; DeGroot, Koot, & Velhurst, 1994; Heubeck, 2000; Liu et al., 2000). Regarding validity, the CBCL Externalizing Problems scale shows discriminative validity between clinic referred and non-referred children (effect size = 33%). Notably, children from a lower socioeconomic status also tend to have higher Externalizing scores compared to children from a higher socioeconomic status, although this effect (effect size = 2%) is small (Achenbach & Rescorla, 2001). Regarding criterion validity, CBCL Externalizing has been shown to correlate

highly (r = .85 - .88) with the externalizing scale from the Behavior Assessment Scale for Children (BASC; Reynolds & Kamphaus, 1992). In the SITSS sample as a whole, age 5 Cronbach's alpha for the CBCL Externalizing scale is .87.

The SDQ self-report version (Goodman, 1997; 1999) is a brief screening questionnaire for children and adolescents. The SDQ includes 25 items that constitute 5 scales: emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems, and prosocial behavior. The first four of these scales are added together to generate a total difficulties score. According to Goodman and colleagues (1998), the self-report version of the SDQ is suitable for individuals aged 11-16 years. However, Di Riso and colleagues (2010) found that the reliability of the total difficulties scale is sufficient in 8- to 10-year-old children (Cronbach's alpha = .67 - .71). The externalizing scale, which is comprised of the conduct problems and hyperactivity/inattention scales (John & DiLalla, 2013), was used as a measure of self-reported problem behavior at follow-up.

Sleep Problems

Although the CBCL does not include a sleep problems scale, researchers have used sleep-related items from the CBCL to formulate a parent-rated measure of sleep problems. The 6 items that have been used as a measure of sleep problems are: "nightmares," "overtired without good reason," "sleeps less than most kids." "sleeps more than most kids," "trouble sleeping," and "talks or walks in sleep." Becker, Ramsay, and Byars (2015) found these items to have adequate reliability (Cronbach's alpha = .61). In the SITSS sample at age 5, Cronbach's alpha is slightly lower at .52. Regarding external validity, this scale was strongly correlated with the well-validated Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000) and had similar correlations to the CSHQ with social problems and psychopathological symptoms in a

sample of children and adolescents referred to a sleep disorders clinic (Becker et al., 2015). Furthermore, Gregory and colleagues (2011) found that scores on the CBCL sleep items were correlated with other sleep measures, including sleep diaries, actigraphy, and polysomnography. This CBCL measure of sleep problems was used to assess parent-reported sleep problems at age 5.

Because the value of self-report measures increases with children's age, a child-reported measure of sleep was utilized at follow-up, in addition to the parent-reported measure of sleep. In support of including a child-reported measure of sleep, Paavonen and colleagues (2000) found that without children's self-report, one third of sleep problems may remain unidentified in school-aged children. The Children's Report of Sleep Patterns (CRSP; Meltzer et al., 2013) is a 60-item questionnaire including items related to children's sleep habits (see Appendix H). Questions from the CRSP are grouped into 3 modules: Sleep Patterns, Sleep Hygiene Index, and Sleep Disturbance Scale. Sleep Patterns includes items related to bedtimes, wake times, sleep onset, night waking, napping, sleep schedules, and subjective sleep quality. The Sleep Hygiene Index includes items related to caffeine consumption, activities (physical and electronic) close to bedtime, and sleep location. The Sleep Disturbance Scale includes items related to fears and worries surrounding bedtime, restless legs, and other sleep disorder related items. The grouping of items into the scales was achieved through review by 15 pediatric sleep experts, and the three CRSP scales were found to have acceptable reliability in the original article by Meltzer and colleagues (Cronbach's alphas > 0.70). Additionally, the CRSP differentiated between clinical and community samples, as poorer sleep hygiene and more sleep disturbance was reported in children referred to sleep clinics, compared to the community sample (Meltzer et al., 2013). The validity of the CSRP was also highlighted by correlations between child-reported symptoms of

insomnia and actigraph-measured sleep duration. For the current study, the CRSP Sleep Disturbance and Sleep Hygiene scales (the Sleep Patterns section does not yield scale scores) were utilized as both parent- and child-report measures of sleep problems at follow-up. Reliability statistics (Cronbach's alpha) are as follows for the current sample: parent sleep hygiene (.68), parent sleep disturbance (.69), child sleep hygiene (.47), and child sleep disturbance (.75).

Because some of the younger children in the study may be expected to be poor reporters of their own sleep habits/patterns, I modified the CRSP to administer to parents. Meltzer (2013) suggests that child reports of sleep problems may be best utilized in clinical settings as a complementary view to parent reports, rather than relying solely on one or the other. Meltzer also notes that parent reports may be more valid than child reports for specific items/scales, as children might not be aware of some sleep disturbances (e.g., sleepwalking, snoring). Including a parent version of the CRSP in the current study allows for comparisons between the two sources and other associated measures. I modified the CRSP by replacing the word "you" with the words "your child" and making appropriate grammatical changes (see Appendix I). Meltzer (2013) notes that a parent proxy version of the CRSP was compared with the child-report version, and that parent reports of children's sleep disturbances were consistent with child reports of sleep quality (reliability statistics not reported).

Temperament

As discussed earlier, although multiple measures of temperament are likely to be important for my hypotheses, I only included negative emotionality in this project. This decision was made given prior research (discussed above), power concerns, the importance of continuous variables, and age considerations. The Behavioral Styles Questionnaire (McDevitt & Carey,

1978) was used as a measure of negative emotionality at age 5 years. The BSQ is a parent-report measure that assesses the 9 factors originally outlined by Thomas and Chess (1970): activity level, rhythmicity, approach/withdrawal, adaptability, intensity, mood, persistence, distractibility, and sensory threshold. For the current study, negative emotionality was defined as the sum of scores on Adaptability, Intensity, and Mood, as outlined in Bersted and DiLalla (2016). Notably, combinations of scores on BSQ rhythmicity, approach/withdrawal, adaptability, intensity, and mood scales yield diagnostic clusters labeled "easy", "difficult", and "slow to warm up." However, these classifications were not included in analyses due to the categorical nature of the variable.

McDevitt and Carey (1978) found that both test-retest reliability and internal consistency were satisfactory for BSQ scales (Cronbach's alphas range from .84 to .89). Regarding validity, Carey, Fox, and McDevitt (1977) demonstrated relationships between BSQ scales and problem-solving tasks, as well as with teacher's ratings of school adjustment. Bersted and DiLalla (2016) found that internal consistency for the negative emotionality scale was good (Cronbach's alpha = .79). Regarding validity, the negative emotionality scale was significantly correlated with the CBCL Externalizing (r = .59) and Internalizing (r = .49) scales (Bersted & DiLalla, 2016).

Stressful Life Events

Because stressful life events are associated with adverse outcome (e.g., diathesis-stress), I included a measure assessing stressful events within the family. The Social Readjustment Rating Scale (SRRS; Holmes & Rahe, 1967) is a 43-item checklist of possible stressful life events that have occurred within the past year, or are expected to occur in the next year. The SRRS, which was initially developed to predict illness, is one of the most widely used instruments to measure stress in research studies (Scully, Tosi, & Banning, 2000; Hock, 1995).

Within the SRRS are 43 stressful events listed in order of severity, with the most stressful events (e.g., death of spouse, divorce) listed first, and the less stressful events (e.g., vacation, minor violation of the law) listed last. Each item is assigned a point value between 11 and 100 (100 = most severe, 11 = least severe), and the points are summed to obtain a total stressful life events score. If an item is endorsed as occurring more than once, the point value for that item is multiplied by the number of times the item has occurred.

Although the SRRS has been criticized for several reasons, such as the inclusion of both controllable and uncontrollable events, as well as both desirable and undesirable events, the SRRS has been used in hundreds of published research studies and is also used in medical and mental health care intake assessments (Scully et al., 2000). Regarding reliability, Gerst et al. (1978) found acceptable rank-order stability in both a community (r = 0.96 to 0.89) and a clinical (r = 0.91 to 0.70) sample. Regarding validity, Holmes and Rahe (1967) initially tested the predictive validity of SRRS scores during scale development, and they found a positive correlation between SRRS and illness scores (r = 0.118). Paykel and colleagues (1969) found that a modified version of the SRRS predicted depressive symptoms in psychiatrically hospitalized patients.

For the current study, I modified the SRRS (see Appendix J) to assess family stressors rather than individual stressors. This is because it was assumed that most stressors listed on the SRRS are experienced by all children in each family, and that within-family differences in stressors on the SRRS are minimal. Parents indicated the number of times that each event "has occurred to anyone in your home within the past year, or is expected in the near future," and total scores were calculated by summing responses according to the original scoring method (as described above).

CHAPTER 4

RESULTS

Before running primary analyses, I ran correlations between all continuous variables (see Table 1) and checked all variables for skewness and outliers. All descriptive statistics are in Table 2. To correct for skewness, skewed variables were first square rooted or squared, and then log transformed if the variables were still skewed. Parent-rated sleep hygiene, child-rated sleep disturbance, age 5 CBCL sleep, age 5 externalizing, negative emotionality, and follow-up externalizing were skewed and therefore were successfully corrected using either log or square-root transformations (see Table 2). Additionally, I examined relationships between the independent variables and SES and sex. I ran regressions to determine if SES and/or sex were predictive of the externalizing scores from age 5 and follow-up (because externalizing is the only dependent variable for the primary hypotheses) and found that sex was significantly predictive of age 5 externalizing. Therefore, sex was included as a covariate in the primary analyses. Neither age 5 SES, follow-up SES, nor the SES change score (difference between age 5 SES and follow-up SES) were predictive of externalizing, so SES was not included in the primary analyses. Only one family had outlying (greater than 2 standard deviations below mean) SES data, but the family was kept in analyses because their sleep and externalizing data were all within 2 standard deviations of the mean. For one child, there was missing CRSP data contributing to the Sleep Disturbance scale. These item scores were imputed by calculating and entering the mean of the other Sleep Disturbance items for that child.

Hypothesis 1: Heritability of Sleep Problems

Hypothesis 1 stated that sleep problems (both child- and parent-rated) are heritable. For Hypothesis 1, I utilized the twin method to evaluate the heritability (h^2) of sleep problems. As

outlined by DiLalla (2002), the twin method involves comparing pairs of monozygotic (MZ) twins, who share 100% of their genetic makeup, and dizygotic (DZ) twins, who share 50% of their genetic makeup, on average. Triplets were included by entering triplet data as three separate pairs in order to include them in the calculations. Then, correlations between MZ twins (r_{MZ}) and correlations between DZ twins (r_{DZ}) werecalculated for a particular trait. Broadly, if r_{MZ} is close in magnitude to r_{DZ}, that suggests that genetics exert minimal influence on the trait, whereas a higher r_{MZ}, compared to r_{DZ} suggests a genetic influence. Importantly, the correlation between MZ twins depends both on their genetic similarity (h^2) and their shared environmental influences (c^2). Therefore, this correlation can be denoted as

$$r_{MZ} = h^2 + c^2.$$

Likewise, the correlation between DZ twins depends on their shared genetic make-up (50%) and shared environmental influences. This correlation is denoted as

$$r_{DZ} = (1/2)h^2 + c^2.$$

In order to estimate heritability (h^2), the second equation is subtracted from the first and then the difference is doubled:

$$h^2 = 2(r_{MZ} - r_{DZ}).$$

An important assumption that is made when using this estimate of heritability is the "equal environments assumption" (EEA). This assumption states that the degree of similarity between the environments of MZ twins is comparable to the degree of similarity between the environments of DZ twins in relation to the traits of interest. Exceptions are active or evocative gene-environment correlations, which describe how children's environments are, in part, determined by their genetic make-up through niche-picking or through "evoking" responses from the environment based on genetically-determined traits. Regarding the equal environments

assumption, it is true that MZ twins may have more similar environmental influences, including sharing peers, bedrooms, and patterns of dress (Loehlin & Nichols, 1976), compared to DZ twins. However, these differences do not result in more similar psychological traits (Loehlin & Nichols, 1976) or clinical diagnoses (Hettema, Neale, & Kendler, 1995; Kendler, Neale, Kessler, Heath, & Eaves, 1993) in MZ twins.

To analyze sleep problems in preschool-aged children and at follow-up, I computed the correlations between sleep scores of MZ twins (r_{MZ}) and DZ twins (r_{DZ}). In order to determine whether the MZ correlations were significantly different from the DZ correlations, I transformed the r values to z values (Fisher transformation). All correlations and heritability estimates are in Table 3. For the CBCL sleep scale (collected at age 5), the MZ correlation was much higher than the DZ correlation, suggesting dominance genetic effects (the masking of "recessive" traits by a "dominant" allele). When dominance effects occur, the heritability estimate can be roughly estimated as equal to r_{MZ}. For CBCL sleep, r_{MZ} was significantly greater than r_{DZ}, so h^2 was significant. Regarding the CRSP scales, heritability calculations for parent-rated sleep disturbances are indicative of dominance effects (because r_{MZ} is so much greater than r_{DZ}, yielding a significant h^2), whereas parent-rated sleep hygiene did not appear to be significantly heritable. For child-reported sleep problems (only collected at follow-up), neither sleep disturbances nor sleep hygiene were significantly heritable. Overall, these results suggest that sleep hygiene is not significantly heritable when rated by children and parents. However, sleep disturbance does appear to have genetic contributions when rated by parents, although children's ratings of sleep disturbance were not significantly heritable.

Hypothesis 2: Longitudinal Associations

Hypothesis 2 stated that externalizing problems would be stable over time, and that early sleep problems and temperament traits would be predictive of follow-up externalizing problems. For hypothesis 2, I used mixed model multilevel linear regression in order to avoid violating the assumption of independent data due to the hierarchical nature of twin/triplet data. In other words, there are multiple children in each family, which represents a hierarchical structure. Multilevel linear modeling (MLM) allowed me to include a nested factor accounting for siblings within families. Importantly, the dependent variables for Hypothesis 2 (follow-up externalizing) were rated by both parents and children. Thus, all analyses were run twice, once with parent-rated externalizing and once with child-rated externalizing as the dependent variables.

Hypotheses 2a, 2b, and 2c state that externalizing behavior, sleep problems, and temperament at age 5 years would predict follow-up externalizing behavior. To test these hypotheses, I entered variables sequentially and calculated whether the model was improved by adding each variable. For parent-rated follow-up externalizing as the dependent variable (see Table 4), the first model included only age 5 externalizing, which was a significant predictor of follow-up externalizing, $p < .001$. Model 2 added sibship as a random effect, which modeled the hierarchical structure of the data and significantly improved the model fit, χ^2 change (1) = 11.58, $p < .01$. The following models (3 through 6) added random slopes (allowing slopes across families to vary), sex, age 5 CBCL Sleep Problems, and age 5 Negative Emotionality, respectively. None of these variables significantly improved the model fit, p's > .05, indicating that the best fitting model was Model 2, which included age 5 externalizing and sibship status. This result supports the stability of externalizing problems over time (Hypothesis 2a). Parameter estimates for the best model are in Table 5.

For child-rated follow-up externalizing as the dependent variable (see Table 6), the first model included only age 5 externalizing; the parameter estimate for age 5 externalizing indicated that it was not a significant predictor of follow-up externalizing, $p = .22$. Model 2 maintained age 5 externalizing and added sibship as a random effect, which modeled the hierarchical structure of the data and improved the model fit, χ^2 change (1) = 5.00, $p < .05$. Adding random slopes did not improve the model fit, but adding sex did improve the model fit, χ^2 change (1) = 5.29, $p < .05$. Adding age 5 CBCL Sleep Problems and age 5 Negative Emotionality did not improve the model fit, p's > .05, indicating that the best model fit was Model 4, which included age 5 externalizing, sibship, and sex (see Table 7).

Hypothesis 3: Moderator Effects

For hypothesis 3, I used MLM to examine moderator (interaction) effects of temperament traits and genotype. These analyses were based on the best fitting models predicting externalizing behavior from hypothesis 2 and are described in Tables 4 through 6. Thus, in order to test Hypothesis 3 for parent-rated externalizing, I added the genetic variables, centered negative emotionality, and interactions between each genetic variable and negative emotionality to Model 2 (see Table 4, models 1-6), which included age 5 externalizing and sibship. For child-rated externalizing, I added those same variables to Model 4 (see Table 6, models 1-6), which included age 5 externalizing, sibship, and sex. Thus, these were used to test hypothesis 3 in order to determine if temperament or genotype interactions further improved those models.

Hypothesis 3a stated that age 5 negative emotionality would moderate the relationship between early sleep problems and later externalizing problems. To test this hypothesis, I first centered all variables to be included in the interaction terms. Then, I used the best fitting models from hypothesis 2 and added the main effects of negative emotionality and age 5 sleep problems

(Tables 4 and 6; Model 7), followed by the interaction between negative emotionality and age 5 sleep problems (Tables 4 and 6; Model 11) separately as fixed effects. Adding the negative emotionality X sleep problems interaction effect did not significantly improve the prediction of externalizing rated by parents, $\chi^2(1)$ change = 2.95, $p > .05$, or children, $\chi^2(1)$ change = 1.51, $p > .05$.

Hypothesis 3b stated that genotype would moderate the relationship between sleep and externalizing problems. To test this hypothesis, I again used the best fitting models from hypothesis 2 and added the main effects of 5-HTT risk, DRD4 risk, and the combined genetic score, followed by the 3 interaction variables separately as fixed effects. Thus, for 5-HTT, I first entered sleep (centered) and 5-HTT risk (Model 8), and then their interaction (Model 12). For DRD4, I entered sleep (centered) and DRD4 risk (Model 9), and then their interaction (Model 13). For the combined genetic score, I entered sleep (centered) and the combined genetic risk score (Model 10), and then their interaction (Model 14). None of these interaction terms significantly improved the model fit for parent- or child-rated externalizing problem behaviors (see Tables 4 and 6).

Post-Hoc Analyses

I conducted a series of post-hoc analyses to address the potential impacts of stressful life events on externalizing behavior. Given the literature suggesting interactions between stressful life events and genetic factors (Bakermans-Kranenburg & van IJzendoorn, 2006; Karg et al., 2011), I ran MLM analyses to assess possible interaction effects on follow-up externalizing rated both by parents and children. For the parent-rated externalizing analyses, I added main effects and interactions between stressful life events and DRD4, stressful life events and 5-HTTLPR, and stressful life events and the combined genetic risk score to the best fitting model (Model 2) from

hypothesis 2 (see Table 8). First, I added stressful life events and each genetic variable (DRD4, 5-HTTLPR, and combined score) separately as main effects (Models 4-7), and none of them significantly improved the model fit from the model including age 5 externalizing and sibship. Then, to test interaction effects, I added three models with the main effects. Model 8 included age 5 externalizing, sibship, stressful life events, and DRD4 risk. Model 9 included age 5 externalizing, sibship, stressful life events, and 5-HTTLPR risk. Model 10 included age 5 externalizing, sibship, stressful life events, and the combined genetic risk. I then tested three interaction models (Models 11-13) which included interactions between the respective genetic variables and stressful life events.

Comparisons between the interaction models and main effects models showed that neither the 5-HTTLPR nor the combined genetic risk score interacted significantly with stressful life events to predict parent-rated externalizing. However, the DRD4 X stressful life events model did significantly improve the model, $\chi^2(1)$ change = 5.88, $p < .02$. Parameter estimates from that model showed that both DRD4 risk and the DRD4 X stressful life events interaction were significant predictors of parent-rated externalizing (see Table 9). I probed this interaction by grouping children into high- and low-stressful life events at the 50^{th} percentile. Results showed that children without the DRD4 risk allele did not differ on follow-up externalizing as a function of stressful life events; however, if children had at least one risk allele, those with more stressful life events scored significantly higher on follow-up externalizing problems (see Figure 2). Because these interaction analyses are exploratory, they should be interpreted with caution.

For child-rated externalizing, I similarly added main effects and interactions between stressful life events and DRD4, stressful life events and 5-HTTLPR, and stressful life events and the combined genetic risk score to the best fitting model (Model 4) from hypothesis 2 (see Table

10). Regarding main effects, stressful life events significantly improved the model which included age 5 externalizing, sibship, and sex, $\chi^2(1)$ change = 5.54, $p < .05$ (see Table 11 for main effects of the best model). None of the genetic variables significantly improved the model fit compared to the model including age 5 externalizing, sibship, sex, and stressful life events. Interaction analyses also indicated that none of the gene X stressful life events interactions significantly improved that model.

I also conducted exploratory MLM analyses to assess longitudinal predictors of sleep disturbance, rated both by parents and children. For these two analyses, fixed independent variables included age 5 sleep problems (to assess stability), sibship, random slopes, sex, age 5 externalizing, and negative emotionality, which were added to the MLM analyses one at a time. For parent-rated sleep disturbance, none of the independent variables significantly predicted child-rated sleep disturbance (Table 12). For child-rated sleep disturbance, sex significantly improved the model fit, although neither sex nor age 5 sleep problems were significant independent predictors of follow-up child-rated sleep disturbance (Tables 13 and 14).

CHAPTER 5

DISCUSSION

For the current project, I sought to assess longitudinal relationships between sleep problems and externalizing behavior. Additionally, I wanted to assess the heritability of childhood sleep problems, which has not been extensively studied. Last, a focus of this study was the presence of moderators (temperament and genotype), which I hypothesized may be differentially associated with children's externalizing problems based on the presence or absence of early sleep problems. In addition to primary hypotheses regarding longitudinal relationships, heritability, and moderators, I conducted post-hoc analyses to assess sleep problems as a dependent variable in longitudinal relationships, as well as the impact of stressful life events on externalizing behavior. This project was intended to address major limitations to the existing literature on longitudinal relationships between sleep, temperament, and externalizing. First, whereas researchers typically use retrospective accounts of behavior in longitudinal sleep studies (e.g., Bates et al., 1998; Goodnight et al., 2007), I was able to use prospective data which are less biased. Second, existing literature has largely neglected the impact of genetic factors which may affect longitudinal relationships between sleep and externalizing. I assessed genetic variables in multiple ways, including through heritability analyses and the use of specific genetic risk markers. Results from this study have theoretical implications (e.g., risk theories) as well as clinical applications (e.g., early screening and intervention), which are discussed below.

Heritability

Regarding heritability, I hypothesized that sleep problems would be significantly heritable when rated by parents (at age 5 and follow-up) and by children (only at follow-up). Although EEG patterns in normal sleep traits are significantly heritable (Ambrosius et al., 2008; De

Gennaro et al., 2008), research has not previously assessed the heritability of problematic sleep behaviors. In this study, parent-reported children's sleep problems were measured at age 5 by calculating a CBCL sleep scale (Becker et al., 2015), and both parents and children provided reports of sleep hygiene and sleep disturbance at follow-up (using the CRSP). Of these scales, CBCL age 5 sleep ($h^2 = 0.88$) and CRSP sleep disturbance rated by parents ($h^2 = 0.74$) were significantly heritable, whereas the other follow-up sleep measures did not show significant heritability ($h^2 = 0.00 – 0.50$). Because the CBCL age 5 sleep scale contains items primarily related to sleep disturbance rather than to sleep hygiene, it appears that parent ratings of sleep disturbance are heritable both at age 5 and at follow-up ages. However, neither children's nor parents' reports of sleep hygiene were significantly heritable, suggesting that behaviors and routines around bedtime are more related to environmental factors rather than to genetic factors. It was surprising that children's own reports of sleep disturbances were not significantly heritable, which would have strengthened the notion that sleep disturbances are related to genetic factors. However, when considering that many sleep disturbances occur while children are unconscious (e.g., sleep walking, snoring), it is possible that parents are better raters of children's sleep disturbances than the children themselves.

Heritability results are especially useful clinically. For instance, knowledge of parental sleep disturbances may be used to identify children at higher risk for inheriting their own sleep problems. In turn, these children may receive services aimed at preventing the development of problematic sleep, such as education about sleep hygiene. Because many early screening and intervention efforts occur in primary care and school settings, these may be optimal settings in which to identify children who would benefit from prevention and intervention for sleep problems, such as Cognitive Behavioral Therapy for Insomnia (Clarke et al., 2015).

The finding that sleep disturbances are influenced by genetic factors is indicative of underlying specific genes that are related to sleep disturbance. Although there are certainly many, many genes involved in the regulation of children's sleep, my next hypothesis focused in part on two specific genes which regulate the functioning of the neurotransmitters, dopamine and serotonin. Prior research has not assessed genetic contributions to sleep problems, and thus it is unknown whether dopaminergic and/or serotonergic functioning affect, and possibly interact with, childhood sleep problems. Therefore, the finding that sleep disturbances are significantly heritable strengthens other hypotheses related to interactions between sleep and specific genetic factors. However, specific gene analyses (below) did not indicate that either DRD4 or 5-HTTLPR predicted sleep problems. Although that finding is likely related to low power, there are many other genes that likely contribute to sleep regulation and which were not measured in this study. Thus, the finding that sleep disturbances are heritable should inform future research into a broader array of genetic determinants of sleep problems.

Longitudinal Relationships and Moderators

A primary focus of this study was the assessment of longitudinal relationships between early sleep problems and later externalizing problems. Although the development of externalizing problems is caused by many factors, I sought to assess whether preschool sleep problems represent a significant risk factor for later externalizing problems. Whereas sleep has been shown to affect cognitive and emotional functioning in the short-term (Epstein et al., 1998; Fallone et al., 2001; Randazzo et al., 1998), these relationships have not been assessed over longer periods of time. Additionally, research regarding potential moderators of sleep-externalizing relationships is largely lacking, although Goodnight and colleagues (2007) found that temperamental resistance to control moderated the relationship between sleep problems and

externalizing from ages 5-9 years. This moderator effect was such that children who were high in resistance to control and also had sleep problems were more likely to exhibit externalizing behavior.

In the current study, it is important to note that I used multilevel linear modeling (MLM), which accounts for the nested nature of the twin design by adding a sibship variable. Notably, without the use of MLM, the best way to avoid violating the assumption of independent data is to use only one twin from each family, which cuts the sample in half. Thus, the use of MLM allowed me to use the full sample while also taking into account within-family variance.

Regarding Hypothesis 2, I expected that age 5 externalizing problems, sibship, sex, age 5 sleep, and negative emotionality would be significantly predictive of later externalizing problems. The MLM analyses demonstrated that, for predicting parent-rated follow-up externalizing, only age 5 externalizing was a significant predictor. For child-rated follow-up externalizing, only sex was a significant predictor, with boys being more likely than girls to rate themselves as having more externalizing problems. Neither early sleep problems nor negative emotionality were significant predictors of either parent- or child-rated externalizing.

These results suggest that externalizing problems in this sample are somewhat stable over time, at least when rated by parents at both time points (Hypothesis 2a). When children rate their own externalizing at follow-up, however, ratings of externalizing problems are not stable over time. Whereas it is possible that this finding represents children's rater biases about their own behavior, it is also possible that their behavior has truly changed and that the parent ratings are stable simply due to parents' own rating consistencies. Regarding sleep and temperament (Hypotheses 2b and 2c), which were not predictive of later externalizing problems, it is possible that over an extended period of time (up to 7 years from age 5 to follow-up), effects may not be

detectable due to other factors (e.g., peer influences, maturation) impacting externalizing scores. Additionally, there are many others factors that influence externalizing behavior, including parenting (Lovejoy, Graczyk, O'Hare, & Neuman, 2000) and trauma (Perry, Pollard, Blakley, Baker, & Vigilante, 1995). Other factors such as these may be stronger predictors of externalizing problems, thus "washing out" smaller effects of sleep and temperament.

Interestingly, my results differ from those of Eisenberg and colleagues (2009), who found that externalizing problems at age 10 years were predicted by high negative emotionality at age 6 years. Given that the Eisenberg study utilized prospective temperament ratings (unlike most longitudinal studies), it may be that there are important social and developmental changes between age 5 (preschool) and age 6 (school-age). For instance, certain types of externalizing behavior (e.g., behavioral problems associated with ADHD) increase around the time that children begin formal schooling and continue through the school-age years (APA, 2013). Therefore, it is possible that some 5-year-old children in my sample may have developed more behavioral problems at age 6, which would have altered the prediction of later behavioral problems. Additionally, the difference in findings may be due to lower power in my study compared to the Eisenberg study (N = 214).

Regarding moderator effects, neither negative emotionality nor any of the genetic factors (DRD4, 5-HTTLPR, combined genetic risk score) significantly interacted with early sleep problems to improve the prediction of later externalizing problems. The lack of a significant temperament X sleep interaction was surprising given that a similar interaction was found by Goodnight and colleagues (2007). However, a main difference between the Goodnight study and my study is the use of retrospective reporting of temperament by that study, compared to the use of a prospective longitudinal design in my study. Thus, it is possible that differences in our

findings may be due to differences in temperament ratings, as those ratings in the Goodnight and colleagues study were more susceptible to memory decay and recency bias. As with the Eisenberg study, however, the differences could also be reflective of lower power in my study (N=93) compared to the Goodnight and colleagues study (N = 556).

Regarding genetic interactions, it does not appear that sleep interacts with DRD4, 5-HTTLPR, or a combined genetic risk score to predict externalizing problems. Despite the use of a combined score as an effort to remediate concerns about the use of single-gene variables (Roukos, 2010), I did not detect any genetic interaction effects predicting externalizing, which may represent either a lack of true gene X sleep interaction or low power. According to Dick and colleagues (2015), genetic interaction studies as a whole need to be interpreted with caution. They argue that genetic studies with small sample sizes (e.g., N < 1000) are likely to be underpowered to detect genetic influences, especially given the small effect sizes of single gene effects. Given that my study suffered from a sample size of far fewer than a thousand children, as well as existing literature linking DRD4 and 5-HTTLPR to behavioral and emotional problems (including externalizing), it is unlikely that my non-finding indicates a lack of true association between genetic and behavioral variables. Indeed, my post-hoc tests suggest (although cautiously) that DRD4 may interact with stressful life events to predict externalizing problems (described below).

Post-Hoc Tests

Stressful Life Events

Because there is literature suggesting interactions between genetic factors and stressful life events (Bakermans-Kranenburg & van IJzendoorn, 2006; Karg et al., 2011), I ran post-hoc analyses assessing whether these interactions were present in my sample. It is well-established

that stressful life events lead to a plethora of later difficulties, including early death, physical and mental health problems, and poor health behaviors (Felitti, 1998). Stressful events such as parental conflict/divorce, abuse/neglect, household substance use, and parental incarceration are posited to contribute to social, emotional, and cognitive impairment, which in turn leads to high-risk behaviors and eventually more chronic conditions (Felitti, 1998). My study differs from most of the existing literature in this area because the stressful life events included in my measure are generally less severe than those listed above. Additionally, the time span of my study limits conclusions to later in childhood, rather than further into adulthood when many more problems may develop. Thus, it is possible that relationships between stressful life events and externalizing problems in my study represent earlier steps in a larger process that may lead to more severe problems later in life.

The MLM analysis showed that when controlling for age 5 externalizing and sibship, DRD4 risk interacted significantly with stressful life events to predict follow-up parent-rated externalizing. From this analysis it was evident that the children with at least one DRD4 risk allele had differing externalizing scores as a function of stressful life events, whereas children without any DRD4 risk alleles did not differ on externalizing scores regardless of their stressful life events. Specifically, those children who experienced more past-year stressful life events and also had at least one risk allele had more externalizing problems than children who similarly had a risk allele but who experienced fewer stressful life events. This pattern of results is consistent with the differential susceptibility gene X environment interaction theory (Belsky et al., 2007), in which specific gene variations confer malleability to environmental factors such as stressful life events. Further, these results contribute to a body of literature suggesting that DRD4-7R might function as a vulnerability factor that increases children's sensitivity to stressful life events

(Bakermans-Kranenburg & van IJzendoorn, 2006; DiLalla et al., 2015). Future research should consider the effects of trauma across childhood, rather than only in the school-age years, given the developmental impacts of early or chronic childhood trauma on development (Perry et al., 1995).

Sleep as the Dependent Variable

Analyses of sleep problems as a dependent variable were intended to shed light on directional effects of sleep-externalizing relationships. Because early sleep did not significantly predict later externalizing, and because of literature documenting correlations between sleep problems and externalizing behavior (Fallone et al., 2002; Morrison et al., 1992), I sought to evaluate whether early externalizing was related to later sleep problems. These analyses yielded no significant models predicting parent- or child-rated sleep problems. Notably, not even age 5 sleep was significantly predictive of follow-up sleep disturbance, although age 5 sleep approached significance in predicting parent-rated follow-up sleep disturbance. This result is not entirely surprising, given that prior research shows a decrease in sleep problems throughout childhood (Gregory & O'Connor, 2002). Indeed, results regarding sleep problem stability from this study may reflect true instability in sleep problems (i.e., decrease over time) or a lack of power. This research area is particularly relevant for clinical activities, given the high incidence of sleep-related problems in youth with mental health diagnoses (Fallone et al., 2002). Additionally, although parent ratings of sleep problems are correlated with other measures of sleep problems (Meltzer et al., 2013), future longitudinal studies may consider multi-method approaches which utilize objective sleep measures (e.g., polysomnography, actigraphy). The use of actigraphy is well-supported, including by recent research suggesting that early stressful life experiences are associated with actigraph-measured sleep duration and quality (Miadich, Breitenstein, Davis, Doane, & Lemery-Chalfant, 2019)

Strengths and Limitations

This project has several strengths, including the use of prospective rather than retrospective ratings of early childhood behaviors, the use of a twin model, and the inclusion of genetic variables. Whereas most prior longitudinal studies use retrospective ratings of child temperament, which are prone to rater and recall biases, age 5 temperament ratings in my study were obtained at age 5, which contributes to increased validity of those ratings. Additionally, my study captured genetic variables in several ways, including utilization of the twin model to assess heritability, evaluation of two specific genes, and the calculation of a combined genetic risk score. These variables are useful given the combination of genetic and environmental factors affecting externalizing behavior, and the relative lack of genetic considerations in most studies.

In addition to those strengths, there are several limiting factors that should be considered when interpreting the current study. First, because my sample consists primarily of White, middle-class families from a single site in the Midwest, results are not widely generalizable. Second, I did not consider how children's sleep patterns affect each other in this study, which could influence results for children who share a bedroom. Third, although initial power analyses indicated sufficient power for this study, it is possible that power was not high enough to detect smaller effects, which are common in genetic studies, especially when examining single genes (Dick et al., 2015). Fourth, because parents were the raters for many variables in this study (externalizing, sleep problems, stressful life events), rater bias may contribute to findings in multiple ways. Whereas desirability biases could affect both the child- and parent-rated variables, relationships between multiple parent-rated variables (e.g., stressful life events and externalizing) may appear stronger because they were rated by the same person. Last, limitations arise because the participants in this study represent a non-clinical sample, which is not representative of

children with clinically elevated levels of externalizing or sleep problems. Because of this limitation, it is unknown whether results from this study are applicable to children with sleep or conduct disorders. The non-clinical sample also may have impacted results due to the relatively low variability in scores, compared to scores that would be expected from a clinical sample. Because of the above limitations, future studies should use larger, more diverse samples with more variability in sleep and externalizing problems (including children in clinical ranges), which will improve the generalizability and clinical utility of the research. Additionally, future research may be strengthened by the addition of more objective/observational measures to avoid rater bias.

Clinical Implications

As mentioned above, this study has applications for clinical practice. Whereas behavioral interventions are well-established as effective for managing externalizing behavior in children (APA, 2013), psychologists have more recently begun applying behavioral interventions to the treatment of sleep problems. The field of behavioral sleep medicine, which is a relatively new specialty in psychology, focuses on the assessment and treatment of sleep disorders using behavioral, psychological, and physiological principles (Society of Behavioral Sleep Medicine, 2019). Behavioral sleep specialists have additionally begun specializing in pediatric sleep problems, and many health systems now have dedicated pediatric sleep clinics which incorporate integrated behavioral health specialists into their practice.

Given that pediatric behavioral sleep medicine is a relatively young field, the current study adds to a growing body of clinically relevant research. As noted above, the finding that sleep disturbances are heritable is useful for screening and prevention efforts, which can be aimed specifically at children with positive family histories of sleep problems. Whereas sleep problems are frequently encountered in pediatric primary care settings, rates of screening are low and

medical providers report receiving limited sleep training (Honaker & Meltzer, 2015). This study could potentially contribute to standards of care by documenting the heritability of sleep disturbances, which could lead more providers to assess for family histories of sleep problems with pediatric patients.

Regarding externalizing problems, the most clinically salient finding of this study is the interaction between DRD4 and stressful life events predicting externalizing problems at follow-up. The importance of stressful life events is well known due to the Adverse Childhood Experiences (ACEs) Study, which showed that early stressful experiences were related to a host of later behavioral and health problems (Felitti, 1998). Further research into GxE interactions has shown that genetic variants (e.g., DRD4) can serve as vulnerability (e.g., diathesis-stress) or malleability (e.g., differential susceptibility) factors which can increase or decrease the likelihood of maladjustment in the presence of stressful life events. The current study adds to a body of literature supporting the importance of both adverse experiences and genetic factors for healthy development. Clinically, this study can inform screening and intervention efforts aimed at identifying children at highest risk for developing externalizing problems, such as those with several stressful life experiences and "risk" genotypes. Because genetic testing is increasingly used in clinical settings, screening for this type of risk would seem to be feasible in medical settings. Children identified as "at-risk" could then receive further screening and preventative measures, including evidence-based interventions such as The Incredible Years (Reid & Webster-Stratton, 2001).

Conclusions

Overall, this study contributes to existing literature on externalizing and sleep problems in childhood. Whereas prior research has demonstrated the heritability of childhood sleep problems

(Barclay & Gregory, 2012), my heritability analyses add to existing literature by disentangling sleep disturbances (which appear to be heritable) from sleep hygiene (which is not heritable), as well as by assessing heritability over time. Additionally, I found evidence for differential susceptibility with regard to the interaction between stressful life events and DRD4, although that finding should be interpreted with caution due to the post-hoc nature of the analyses. Limitations of this study include low power (especially to detect genetic effects), limited generalizability, and rater bias, although a considerable strength of the study was the use of a prospective longitudinal design. Clinical implications include providing documentation of genetic effects on sleep problems as well as strengthening the importance of GxE interactions for the development of externalizing problems. In addition to considering power, further research should assess mechanisms by which genetic factors affect sleep and externalizing, possibly by using aggregate genetic scores or other methods of assessing genetic risk (e.g., a genetic risk index based on co-twin symptoms). Additionally, using objective measures of sleep patterns, such as actigraphy or polysomnography, may strengthen conclusions drawn about sleep problems.

EXHIBITS

Table 1

Inter-Correlations for Study Measures

	SES5	Ext5	Sleep5	Neg	SESFU	SES Change	pSH	pSD	cSH	cSD	pExt	cEXT
Age 5												
SES (SES5)	-											
Externalizing (Ext5)	-0.28*	-										
Sleep (Sleep5)	-0.05	0.20	-									
Negative Emotionality (Neg)	0.23	0.17	-0.17	-								
Follow-Up												
SES (SESFU)	0.51**	-0.18	0.06	-0.05	-							
SES Change	0.21	-0.10	0.08	0.14	0.70**	-						
Parent Sleep Hygiene (pSH)	-0.37*	0.18	0.08	-0.03	-0.16	0.12	-					
Parent Sleep Disturbance (pSD)	0.00	0.00	0.17	-0.03	-0.01	0.04	-0.10	-				
Child Sleep Hygiene (cSH)	0.04	-0.06	0.29	-0.04	0.18	0.12	0.53**	-0.17	-			
Child Sleep Disturbance (cSD)	0.37*	-0.20	0.06	-0.11	0.13	0.21	-0.23	0.03	0.79**	-		
Parent Externalizing (pExt)	-0.11	0.55**	0.15	0.29*	-0.08	0.22	0.15	0.27*	-0.07	-0.09	-	
Child Externalizing (cExt)	0.18	0.13	-0.01	0.02	0.06	0.06	0.08	-0.02	0.03	0.23*	0.17	-

Note: * < .05, ** < .01

Table 2

Descriptive Statistics and Corrections for Skewness

				Skewness	
	N	Mean	SD	Statistic	Std Error
Parent Sleep Hygiene	93	37.42	7.81	0.76	0.25
Child Sleep Hygiene	93	37.81	6.85	0.25	0.25
Parent Sleep Disturbance	92	27.30	4.12	0.35	0.25
Child Sleep Disturbance	93	28.85	7.21	0.73	0.25
Negative Emotionality	93	2.67	3.55	0.60	0.25
Age 5 Sleep	93	0.77	1.26	2.10	0.25
Age 5 Externalizing	93	5.97	5.06	0.96	0.25
Follow-Up Externalizing	92	5.10	5.39	1.34	0.25
Transformed Variables					
Parent Sleep Hygiene LN	93	1.57	0.09	0.28	0.25
Age 5 Sleep LN	93	0.17	0.24	1.04	0.25
Child Sleep Disturbance SQRT	93	5.33	0.66	0.28	0.25
Negative Emotionality LN	93	1.70	0.54	0.33	0.25
Age 5 Externalizing SQRT	93	2.14	1.18	-0.19	0.25
Follow-Up Externalizing SQRT	92	1.87	1.27	0.21	0.25

Table 3

Heritability Estimates for Parent- and Child-Rated Sleep Problems

	*r*MZ	*r*DZ	h^2	*z*	*p* value
CBCL Age 5 Sleep LN	.881	.117	0.881	3.91	<.001
CRSP Child Sleep Hygiene	.631	.590	0.082	0.2	.42
CRSP Child Sleep Disturbance SQRT	.585	.337	0.496	0.99	.16
CRSP Parent Sleep Disturbance	.740	.063	0.740	2.75	.003
CRSP Parent Sleep Hygiene LN	.931	.952	0.000	-0.58	.281

Table 4

Mixed Model Multilevel Linear Regression Modeling Results, with Parent-Rated Externalizing as the Dependent Variable (Model 2 is Best)

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
Hypothesis 2					
1 – Age 5 Externalizing (H2a)	271.65 (3)	285.21	-	-	-
2 – Model 1 Plus Sibship	260.06 (4)	278.15	1	11.58 (1)	<.01
3 – Model 2 Plus Random slopes	260.06 (5)	282.67	2	0.00 (1)	ns
4 – Model 2 Plus Sex	260.02 (5)	282.63	2	0.04 (1)	ns
5 – Model 2 Plus Age 5 Sleep (H2b)	260.02 (5)	287.15	2	0.04 (1)	ns
6 – Model 2 Plus Negative Emotionality (H2c)	259.00 (5)	290.65	2	1.06 (1)	ns
Hypothesis 3					
7 – Model 2 Plus Sleep & Negative Emotionality	259.04 (6)	286.17	2	1.02 (2)	ns
8 – Model 2 Plus Sleep and 5-HTTLPR	257.88 (6)	285.02	2	2.18 (2)	ns
9 – Model 2 Plus Sleep and DRD4	260.01 (6)	287.14	2	0.05 (2)	ns
10 – Model 2 Plus Sleep and Combined Genes	258.95 (6)	286.08	2	1.11 (2)	ns
11 – Model 7 Plus Sleep X Negative Emotionality (H3a)	256.09 (7)	292.26	7	2.95 (1)	ns
12 – Model 8 Plus Sleep X 5-HTTLPR (H3b)	256.97 (7)	297.67	8	0.91 (1)	ns
13 – Model 9 Plus Sleep X DRD4 (H3b)	257.38 (7)	298.08	9	2.63 (1)	ns
14 – Model 10 Plus Sleep X Combined Genes (H3b)	257.87 (7)	298.56	10	1.08 (1)	ns

Table 5

Mixed Model Multilevel Linear Regression Modeling Parameter Estimates for Best Model from Table 4 (Model 2), Predicting Parent-Rated Externalizing

	Estimate	95% CI	*p* value
Fixed Effects			
Intercept	1.86	1.75; 1.97	.000
Age 5 Externalizing	0.60	0.50; 0.70	.000
Random Effects			
Within-family effect	0.51	0.33; 0.69	.005

Table 6

Mixed Model Multilevel Linear Regression Modeling Results, with Child-Rated Externalizing as the Dependent Variable (Model 4 is Best)

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
Hypothesis 2					
1 – Age 5 Externalizing (H2a)	481.72 (3)	495.31	-	-	-
2 – Model 1 Plus Sibship	476.73 (4)	494.86	1	5.00 (1)	<.05
3 – Model 2 Plus Random slopes	476.73 (5)	494.86	2	0.00 (1)	ns
4 – Model 2 Plus Sex	471.44 (5)	494.05	2	5.29 (1)	<.05
5 – Model 4 Plus Age 5 Sleep (H2b)	471.38 (6)	498.51	4	0.06 (1)	ns
6 – Model 4 Plus Negative Emotionality (H2c)	471.44 (6)	498.57	4	0.00 (1)	ns
Hypothesis 3					
7 – Model 4 Plus Sleep and Negative Emotionality	471.37 (7)	503.03	4	0.07 (1)	ns
8 – Model 4 Plus Sleep and 5-HTTLPR	468.41 (7)	500.07	4	3.03 (1)	ns
9 – Model 4 Plus Sleep and DRD4	471.03 (7)	502.69	4	0.41 (1)	ns
10 – Model 4 Plus Sleep and Combined Genes	469.50 (7)	501.16	4	1.94 (1)	ns
11 – Model 7 Plus Sleep X Negative Emotionality (H3a)	469.86 (8)	478.77	7	1.51 (1)	ns
12 – Model 8 Plus Sleep X 5-HTTLPR (H3b)	468.25 (8)	508.94	8	0.16 (1)	ns
13 – Model 9 Plus Sleep X DRD4 (H3b)	468.17 (8)	508.86	9	2.86 (1)	ns
14 – Model 10 Plus Sleep X Combined Genes (H3b)	468.58 (8)	509.28	10	0.92 (1)	ns

Table 7

Mixed Model Multilevel Linear Regression Modeling Parameter Estimates for Best Model from Table 6 (Model 4) Predicting Child-Rated Externalizing

	Estimate	95% CI	*p* value
Fixed Effects			
Intercept	6.37	5.91; 6.83	.000
Age 5 Externalizing	0.30	-0.02; 0.62	.347
Sex	0.81	0.08; 1.54	.265
Random Effects			
Within-family effect	2.73	1.16; 4.30	.082

Table 8

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Results, with Parent-Rated Follow-Up Externalizing Problems as the Dependent Variable (Model 11 is Best)

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
1 – Age 5 Externalizing	271.65 (3)	285.21	-	-	-
2 – Model 1 Plus Sibship	260.06 (4)	278.15	1	11.58 (1)	<.01
3 – Model 2 Plus Sex	260.02 (5)	282.63	2	0.04 (1)	ns
4 – Model 2 Plus Stressful Life Events (SRRS)	256.97 (5)	279.58	2	3.09 (1)	ns
5 – Model 2 Plus DRD4 Risk	260.02 (5)	282.63	2	0.04 (1)	ns
6 – Model 2 Plus 5-HTTLPR Risk	257.94 (5)	280.55	2	2.12 (1)	ns
7 – Model 2 Plus Combined Genetic Risk	258.96 (5)	281.57	2	1.10 (1)	ns
8 – Age 5 Externalizing, Sibship, SRRS, DRD4 Risk	256.76 (6)	283.90	2	3.30 (2)	ns
9 – Age 5 Externalizing, Sibship, SRRS, 5-HTTLPR Risk	254.26 (6)	281.39	2	5.80 (2)	ns
10 – Age 5 Externalizing, Sibship, SRRS, Combined Genetic Risk	256.20 (6)	283.33	2	3.86 (2)	ns
11 – Model 8 Plus DRD4 X SRRS	250.88 (7)	282.53	8	5.88 (1)	<.02
12 – Model 9 Plus 5-HTTLPR X SRRS	252.98 (7)	284.63	9	1.28 (1)	ns
13 – Model 10 Plus Combined Genes X SRRS	252.92 (7)	284.57	10	1.34 (1)	ns

Table 9

Mixed Model Multilevel Linear Regression Modeling Parameter Estimates for for Best Model from Table 8 (Model 11), Predicting Parent- Rated Follow-Up Externalizing

	Estimate	95% CI	*p* value
Fixed Effects			
Intercept	2.80	1.75; 1.97	.000
Age 5 Externalizing	0.53	0.50; 0.70	.000
DRD4 Risk	-0.98	-1.41; -0.55	.026
SRRS	0.00	0.01; 0.00	.124
DRD4Risk X SRRS	0.00	0.00; 0.01	.016
Random Effects			
Within-family effect	0.44	0.28; 0.60	.006

Table 10

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Results, with Child-Rated Follow-Up Externalizing Problems as the Dependent Variable (Model 4 is Best)

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
1 – Age 5 Externalizing (	481.72 (3)	495.31	-	-	-
2 – Model 1 Plus Sibship	476.73 (4)	494.86	1	5.00 (1)	<.05
3 – Model 2 Plus Sex	471.44 (5)	494.05	2	5.29 (1)	<.05
4 – Model 3 Plus Stressful Life Events (SRRS)	465.89 (6)	493.02	3	5.54 (1)	<.05
5 – Age 5 Externalizing, Sibship, Sex, SRRS, DRD4 Risk	465.19 (7)	496.86	4	0.70 (1)	ns
6 – Age 5 Externalizing, Sibship, SRRS, 5-HTTLPR Risk	463.10 (7)	494.75	4	2.79 (1)	ns
7 – Age 5 Externalizing, Sibship, SRRS, Combined Genetic Risk	462.93 (7)	494.58	4	2.96 (1)	ns
8 – Model 5 Plus DRD4 X SRRS	464.97 (8)	501.14	4	0.92 (2)	ns
9 – Model 6 Plus 5-HTTLPR X SRRS	463.06 (8)	499.23	4	2.83 (2)	ns
10 – Model 7 Plus Combined Genes X SRRS	461.10 (8)	498.17	4	3.90 (2)	ns

Table 11

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Parameter Estimates for Best Model from Table 10 (Model 4), Predicting Child-Rated Externalizing

	Estimate	95% CI	*p* value
Fixed Effects			
Intercept	7.69	6.98; 8.40	<.001
Age 5 Externalizing	0.53	0.22; 0.83	.102
Sex	0.53	-0.18; 1.25	.465
Stressful Life Events	-0.01	-0.01; 0.01	.020
Random Effects			
Within-family effect	2.08	0.65; 3.51	.148

Table 12

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Results, with Follow-Up Parent-Rated Sleep Disturbance as the Dependent Variable

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
1 – Age 5 Sleep	521.40 (3)	495.31	-	-	-
2 – Model 1 Plus Sibship	521.18 (4)	539.31	1	0.22 (1)	ns
3 – Model 2 Plus Random Slopes	521.18 (5)	539.31	2	0.00 (1)	ns
4 – Model 2 Plus Sex	515.47 (5)	538.08	2	5.72 (1)	ns
5 – Model 2 Plus Age 5 Externalizing	520.12 (5)	542.78	2	1.07 (1)	ns
6 – Model 2 Plus Negative Emotionality	521.16 (5)	543.83	2	0.02 (1)	ns

Table 13

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Results, with Child-Rated Sleep Disturbance as the Dependent Variable (Model 4 is Best)

Model	χ^2 (df)	BIC	vs. Model	χ^2 (df)	*p* value
1 – Age 5 Sleep	183.08 (3)	196.68	-	-	ns
2 – Model 1 Plus Sibship	182.97 (4)	196.57	1	-0.11 (1)	ns
3 – Model 2 Plus Random Slopes	182.97 (5)	196.57	2	0.00 (1)	ns
4 – Model 2 Plus Sex	173.47 (5)	196.08	2	9.47 (1)	<.01
5 – Model 4 Plus Age 5 Externalizing	172.38 (6)	199.51	4	1.09 (1)	ns
6 – Model 4 Plus Negative Emotionality	172.95 (6)	200.08	4	0.52 (1)	ns

Table 14

Post-Hoc Test: Mixed Model Multilevel Linear Regression Modeling Parameter Estimates for Best Model from Table 13 (4), Predicting Child-Rated Sleep Disturbance

	Estimate	95% CI	*p* value
Fixed Effects			
Intercept	5.32	5.23; 5.41	<.001
Age 5 Sleep	0.48	0.19; 0.77	.099
Sex	-0.02	-0.16; 0.12	.861
Random Effects			
Within-family effect	0.11	0.05; 0.17	.084

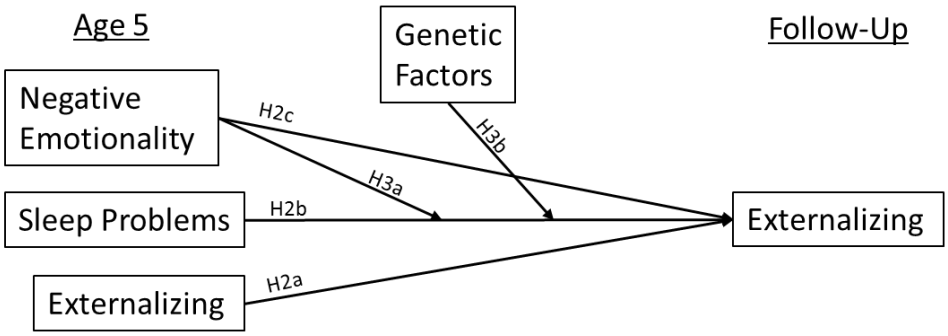

Figure 1. Hypothesized longitudinal relationships. Note: H = Hypothesis (e.g., H2a = Hypothesis 2a).

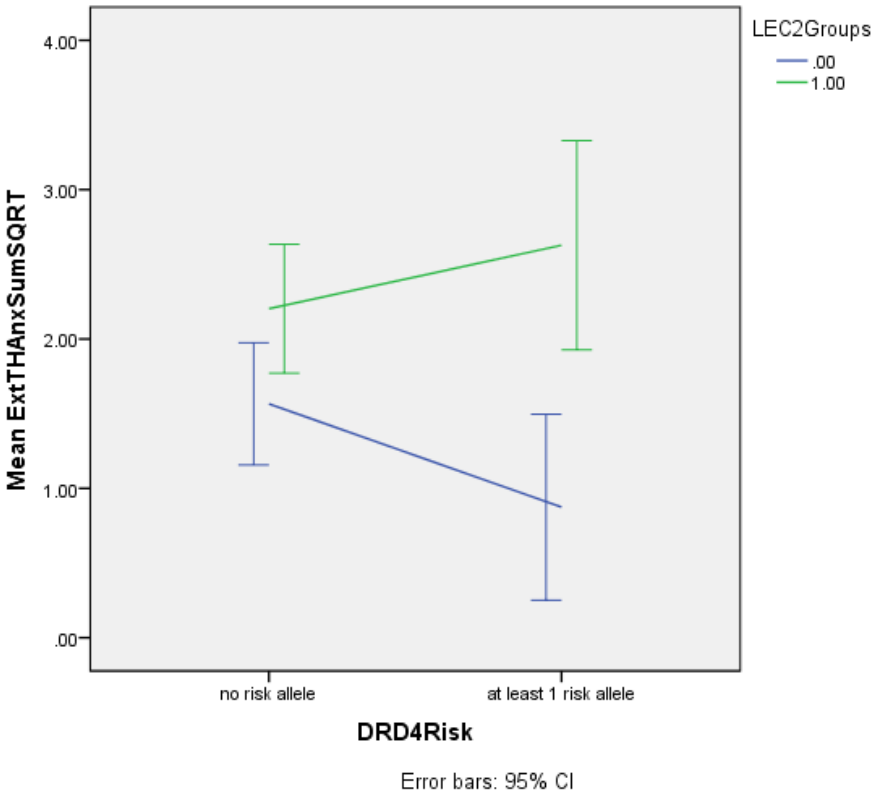

Figure 2. Interaction between DRD4 Risk and Stressful Life Events Predicting Follow-Up Parent-Rated Externalizing Problems.

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APPENDIX A

RECRUITMENT EMAIL

From: Southern Illinois Twins/Triplets and Siblings Study (sitss.siu@gmail.com)

Subject: Research Request

Dear Parent:

Thank you for having participated in Dr. Lisabeth DiLalla's Southern Illinois Twins/Triplets and Siblings Study (SITSS) in previous years! Your e-mail address was obtained from your previous participation in SITSS. A blind copy format was used for this email so that the list of recipients does not appear in the header.

We are now beginning a new study that we hope will interest you! It involves bringing your twins or triplets to the SITSS lab to complete a testing session. During this testing session, your children will be asked questions about their strengths and weaknesses, including sleep habits and worries. We will also provide additional forms for you to complete during the testing session. Your children were selected to participate in this study because they have participated in SITSS in the past.

The testing session will take approximately one hour to complete. All your responses will be kept confidential within reasonable limits. Only people directly involved with this project will have access to the questionnaires you complete.

We do not foresee any significant risks involved with this project. In fact, we expect that this experience should be an interesting and fun one for you and your child. The only possible risk is that your child may feel anxious during a worry task we will use during testing. If this happens, we will stop and use calming techniques to relax him or her. We will use calming techniques at the end of testing for everyone, as it is a pleasant way to end the session.

If you are willing to participate, please respond to this email indicating your interest. You may also call us at (618) 453-1397. After we hear back from you, we will call you to schedule the testing session. To thank you and your children for your participation, the first 100 children to participate will earn $5, which will be given to them on the day of testing. In addition, all children will receive a small gift for participating in the study.

Questions about this study can be directed to us or to our supervising professor, Dr. Lisabeth DiLalla, SIUC School of Medicine, Department of Family and Community Medicine, Carbondale, IL 62901 . Her phone number is (618) 453-1855.

If you would like for your name and email address to be removed from our mailing list, please respond to this email asking us to do so. If you do not respond to this email or return the opt-out message, you will be contacted again with this request in two weeks.

Thank you for taking the time to assist us in this research.

Megan McCrary

SITSS Graduate Assistant

(618) 453-1397

megan.mccrary@siu.edu

Gabe Casher

SITSS Graduate Assistant

(618) 453-1397

gabriel.casher@siu.edu

This project has been reviewed and approved by the SIUC Human Subjects Committee. Questions concerning your rights as a participant in this research may be addressed to the Committee Chairperson, Office of Sponsored Projects Administration, SIUC, Carbondale, IL 62901-4709. Phone (618) 453-4533. E-mail: siuhsc@siu.edu

APPENDIX B

RECRUITMENT LETTER TO ELIGIBLE SITSS FAMILIES

Dear Parent,

Thank you for having participated in Dr. Lisabeth DiLalla's Southern Illinois Twins/Triplets and Siblings Study (SITSS) in previous years! We are now beginning a new study that we hope will interest you! It involves bringing your twins or triplets to the SITSS lab to complete a testing session. During this testing session, your children will be asked questions about their strengths and weaknesses, including sleep habits and worries. Also, we will provide additional forms for you to complete during the testing session. If you would like more information before committing to participate in this study or have questions that are not answered in this letter, please call us at (618) 453-1397 or email us at sitss.siu@gmail.com. We would love to have your help in this study!

If you are willing to participate, please complete and sign the enclosed Contact Form and return it in the pre-stamped, pre-addressed, enclosed envelope. You may also call us to indicate your interest. After we hear back from you, we will call you to schedule the testing session, which will last approximately one hour. To thank you and your children for your participation, the first 100 children to participate will earn $5, which will be given to them on the day of testing. In addition, they will receive a small gift for participating in the study.

We do not foresee any significant risks involved with this project. In fact, we expect that this experience should be an interesting and fun one for you and your child. The only possible risk is that your child may feel anxious during a worry task we will use during testing. If this happens, we will stop and use calming techniques to relax him or her. We will use calming techniques at the end of testing for everyone, as it is a pleasant way to end the session.

All information that we receive from you will be held as strictly confidential. All questionnaires and data will be identified only by an identification number that is assigned to your family. Your children's names will never be placed on the questionnaires or on any data that we receive. Your name, address, and phone number will be maintained in a confidential file on a password-protected computer in order to contact you. All information will be kept in a locked file cabinet and only trained researchers on this project will have access to that file.

We hope that you will join us for this exciting opportunity! We look forward to hearing from you. If you have any questions or concerns, feel free to contact Dr. Lisabeth DiLalla (618-453-1855; sitss.siu@gmail.com), Megan McCrary (618-453-1397; megan.mccrary@siu.edu), or Gabe Casher (618-453-1397; gabriel.casher@siu.edu)

Sincerely,

Megan McCrary
SITSS Graduate Assistant

Gabe Casher
SITSS Graduate Assistant

APPENDIX C

RECRUITMENT PHONE SCRIPT

"Good morning/afternoon, my name is _____and I am a graduate student completing a dissertation research project through the Southern Illinois Twins/Triplets and Siblings Study at Southern Illinois University. Do you have a minute to talk now?"

If no: "Okay, is there another time that you would prefer for me to contact you about this study?"

If yes: "Great, when would you like for me to call you back?" [Set up day and time].

"Is this a good number to reach you?" [Obtain best phone number].

"Thank you, I look forward to speaking with you soon"

If yes: "Did you receive an (email/letter) from us? That is what I'm following up on. This study will involve bringing your twins (or triplets) to the SIU SITSS lab at a convenient time for you and having you and your children answer some questionnaires. The questionnaires will be similar to some of the questionnaires you have completed in the past. Your children will also be shown videos of social interactions and will be asked to answer some questions about them afterwards. We are interested in studying children's overall strengths and weaknesses, sleep habits, worries, health, and empathy. In addition, we will use DNA samples that we collected from your children during an earlier phase of testing to get information about whether your children are identical or fraternal, as well as information about specific genes that we believe may be related to their behavior. All information that you provide will be kept completely confidential, and results from this study will be reported as group data in my dissertation.

This study requires you to bring the twins[triplets] to the lab one time for about an hour. We will give each of your twins[triplets] SITSS hats and coupons to local restaurants to thank them for participating. [If the children are one of the first 100 participants]: We are also able to pay each of your twins/triplets $5.

Do you think you might be interested in participating in the study?"

***If they do not want to participate*:** "Thank you for your time. Goodbye."

***If they are willing to participate*:** "Great, then all I will need is for you to select a date and time that is best for you to bring the twins[triplets] to SIU. [Set up day and time]. I also will need your address so that I can mail you a map to get here. What is your current address?

_____

Do you have any questions for me? [Answer any questions the parent may have]. Thank you very much for your time, and I look forward to talking with you on _____(repeat scheduled date and time). Goodbye."

***If someone asks who is supervising your research, and if you are a student*:** "Yes, I am a student, and Dr. Lisabeth DiLalla is my supervisor. Her phone number is 618-453-1855 if you'd like to speak with her directly."

If someone asks whether your research has been approved by the Human Subjects Committee, have this information available to read to them:

This project has been reviewed and approved by the SIUC Human Subjects Committee. Questions concerning your rights as a participant in this research may be addressed to the Committee Chairperson, Office of Sponsored Projects Administration, SIUC, Carbondale, IL 62901-4709. Phone (618) 453-4533. E-mail: siuhsc@siu.edu

APPENDIX D

CONSENT FORM WITH COMPENSATION

PARENT CONSENT FORM

This research project is a study of normally developing children's social behaviors, perceptions, and attitudes. The purpose is to better understand why some children experience certain behaviors more than others. After being in this study, each of your children will be given $5 for participating. Additionally, they will each be given a small gift after testing.

During testing, your children will be asked a series of questions including questions about their overall strengths and weaknesses, health, and empathy. Another set of questions will be about your child's normal sleeping habits. After answering these questions, your child will complete a worry task, during which your child will be shown videos of social situations and asked questions about them. During the testing session, you will also be asked to complete a questionnaire about your child's behavior. If at any time your child becomes upset or unwilling to complete the session, we will stop immediately. There will be no penalty for this and you and your children will still receive compensation in order to thank you for participating. Also, we will use DNA samples that we collected from your children during past testing to obtain information about whether your children are identical or fraternal, as well as information about specific genes that we believe may be related to their behaviors.

We do not foresee any significant risks involved with this project. In fact, we expect that this experience should be an interesting and fun one for you and your child. The only possible risk is that your child may feel anxious during the worry task. If this happens, we will stop and use calming techniques to relax him/her. We will use calming techniques at the end of testing for everyone, as it is a pleasant way to end the session.

All the questionnaires will be identified only by an identification number that is assigned to your family. Your names will never be placed on the questionnaires. Your name, address, and phone number will be maintained in a confidential file. All information is strictly confidential and will never be shared with anyone outside of this laboratory. It will be kept in a locked file in the lab and only Dr. DiLalla or trained research assistants will have access to that file. The confidential list of names will be maintained so that we can contact families again in the future for follow-up studies. We will take all reasonable steps to protect your identity.

Under Illinois law, an exception to confidentiality is incidents of child abuse or neglect. If, in the course of this research, we develop reasonable cause to believe such an incident has occurred, we are required to contact the Department of Children and Family Services (DCFS).

If you have any further questions about this research project, please feel free to contact the Lab Director, Dr. Lisabeth DiLalla, at the SIUC School of Medicine, Department of Family and Community Medicine, (618) 453-1855. If you agree to participate in this project and to have your child participate, please fill out the section(s) below:

I have read the material above, and any questions I asked have been answered to my satisfaction. I understand I will receive a copy of this form for the relevant information and phone numbers. I agree to participate in this activity and realize that I may withdraw without prejudice at any time.

_____ _____

Parent's Signature Date

Consent Form Without Compensation

PARENT CONSENT FORM

This research project is a study of normally developing children's social behaviors, perceptions, and attitudes. The purpose is to better understand why some children experience certain behaviors more than others. After being in this study, each of your children will be given a small gift.

During testing, your children will be asked a series of questions including questions about their overall strengths and weaknesses, health, and empathy. Another set of questions will be about your child's normal sleeping habits. After answering these questions, your child will complete a worry task, during which your child will be shown videos of social situations and asked questions about them. During the testing session, you will also be asked to complete a questionnaire about your child's behavior. If at any time your child becomes upset or unwilling to complete the session, we will stop immediately. There will be no penalty for this and you and your children will still receive compensation in order to thank you for participating. In addition, we will use DNA samples that we collected from your children during an earlier phase of testing to obtain information about whether your children are identical or fraternal, as well as information about specific genes that we believe may be related to their behavior.

We do not foresee any significant risks involved with this project. In fact, we expect that this experience should be an interesting and fun one for you and your child. The only possible risk is that your child may feel anxious during the worry task. If this happens, we will stop and use calming techniques to relax him/her. We will use calming techniques at the end of testing for everyone, as it is a pleasant way to end the session.

All the questionnaires will be identified only by an identification number that is assigned to your family. Your names will never be placed on the questionnaires. Your name, address, and phone number will be maintained in a confidential file. All information is strictly confidential and will never be shared with anyone outside of this laboratory. It will be kept in a locked file in the lab and only Dr. DiLalla or trained research assistants will have access to that file. The confidential list of names will be maintained so that we can contact families again in the future for follow-up studies. We will take all reasonable steps to protect your identity.

Under Illinois law, an exception to confidentiality is incidents of child abuse or neglect. If, in the course of this research, we develop reasonable cause to believe such an incident has occurred, we are required to contact the Department of Children and Family Services (DCFS).

If you have any further questions about this research project, please feel free to contact the Lab Director, Dr. Lisabeth DiLalla, at the SIUC School of Medicine, Department of Family and Community Medicine, (618) 453-1855. If you agree to participate in this project and to have your child participate, please fill out the section(s) below:

I have read the material above, and any questions I asked have been answered to my satisfaction. I understand I will receive a copy of this form for the relevant information and phone numbers. I agree to participate in this activity and realize that I may withdraw without prejudice at any time.

_____ _____

Parent's Signature Date

APPENDIX E

CHILD ASSENT FORM

I am (NAME) from the SIU Twin Lab. I'm going to ask you some questions about things you are good at or not good at, and things like sleeping, worrying, and friends. Remember, you can stop at any time if you don't like the questions, and you will still get your prize for being here today. All you have to do if you need to stop is tell me.

Is this okay? Are you ready to play?

_____ _____

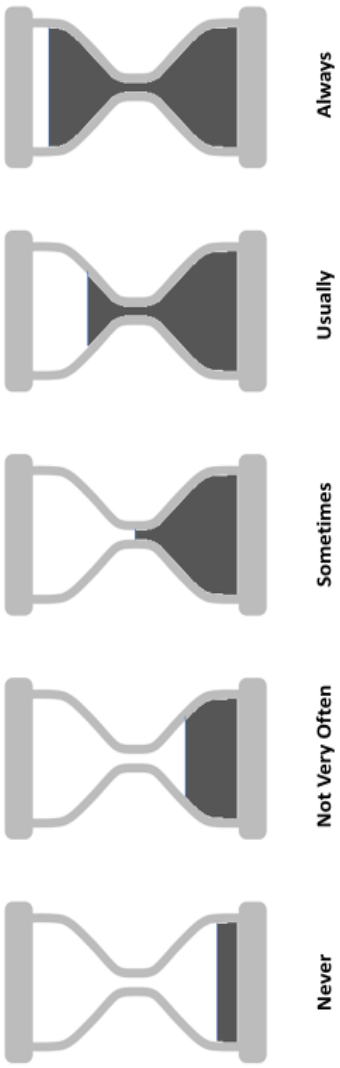
Assenting Child Date

_____ _____

Parent's Signature Date

APPENDIX F

CHILDREN'S ANSWER CARD



APPENDIX G

FAMILY INFORMATION SHEET

Date_____ ID Number_____

Age of Child/ren _____ DOB child/ren_____

Your Relationship to the child/ren (mother or father; please note if adoptive parent):_____
Your Age: _____

Marital Status:
Single, never married_____ Married_____ Divorced/Separated _____
Widowed _____ Living with a significant other_____

Approximate Total Family Income:
___ less than $5,000___ $20,000 - 25,000 ___ $40,000 - 45,000 ___ $60,000 - 65,000 ___$80,000 - 85,000

___ $5,000 - 10,000 ___ $25, 000 - 30,000 ___ $45,000 - 50,000 ___ $65,000 - 70,000 ___ $85,000 - 90,000

___ $10,000 - 15,000 ___ $30,000 - 35,000 ___ $50,000 - 55,000 ___ $70,000 - 75,000 ___ over $90,000

___ $15,000 - 20,000 ___ $35,000 - 40,000 ___ $55,000 - 60,000 ___ $75,000 - 80,000

Race of Child's Parents: Mother _____ Father _____

Race of Children in Study: _____

	Occupation (JOB TITLE)			Years of College (undergraduate & graduate)	College Degrees (AA, BA, etc.)
Self					
Spouse or Partner if Living in Home with Children					

Please list the birthdates of all siblings of the children in this study, and please note if they are half-siblings, step-siblings, or adopted siblings of the children in this study:

_____

_____

_____

Please list everyone living in your household and their relation (e.g., father, grandmother, etc.) to the children in the study. (First names only, example: Ben – grandfather)

_____

_____

_____

We are interested in whether changes in the family, such as divorce or remarriage, affect children's behaviors. If applicable, please indicate if you have ever been divorced or remarried and the year this occurred.

Not applicable_____ Divorced _____ Remarried _____

Year_____ Year_____

Year_____ Year_____

APPENDIX H

CHILDREN'S REPORT OF SLEEP PATTERNS (CRSP)

Instructions
This form asks questions about you and your sleep. Please answer the questions as honestly as you can. There are no right or wrong answers. Do not spend too much time on any question; your first answer is usually the best. Choose from each question the answer that best describes you by putting a mark like this ☒ next to your answer. Put the mark in the box next to the answer that you pick. Only choose one answer for each question.

First are some questions about your sleep. Please think about LAST NIGHT when answering these questions.

1) What time did you go to bed last night?
_____ ☐ PM ☐ AM

2) Once you turned your light off, how long did it take you to fall asleep last night?
☐ No time at all, I fell asleep very quickly
☐ A few minutes (5-10 minutes)
☐ A little while (10-30 minutes)
☐ A long time (more than 30 minutes)

3) Did you take any medication to help you sleep last night?
☐ Yes ☐ No
If yes, what medication did you take?
_____ ☐ I Don't Know

4) After you fell asleep, did you wake up during the night?
☐ Yes ☐ No

5) How long did it take you to go back to sleep after you woke up during the night?
☐ I did not wake up last night
☐ No time at all, I went back to sleep very quickly
☐ A few minutes (5-10 minutes)
☐ A little while (10-30 minutes)
☐ A long time (more than 30 minutes)

6) What time did you wake up today?
_____ ☐ PM ☐ AM

7) How did you wake up this morning?
☐ I woke up by myself
☐ I woke up with an alarm clock
☐ Someone in my family woke me up
☐ My pet woke me up

8) How well did you sleep last night?
☐ I had a great night of sleep
☐ I had a good night of sleep
☐ I had an okay night of sleep
☐ I had a poor night of sleep

(go to next page)

The next set of questions has to do with your usual schedule on WEEKDAYS when you go to school.

9) What time do you usually go to bed on weekdays?

- ☐ Before 7:00 pm
- ☐ 7:00-7:29 pm
- ☐ 7:30-7:59 pm
- ☐ 8:00-8:29 pm
- ☐ 8:30-8:59 pm
- ☐ 9:00-9:29 pm
- ☐ 9:30-9:59 pm
- ☐ 10:00-10:29 pm
- ☐ 10:30-10:59 pm
- ☐ 11:00-11:29 pm
- ☐ 11:30pm-11:59 pm
- ☐ After 12:00 am

12a. How often do you usually go to bed at this time?

- ☐ Every night
- ☐ Several times a week (3-4 nights during the week)
- ☐ Every now and then (1-2 nights during the week)

10) Once you turn your light off on weekdays, how long does it usually take you to fall asleep?

- ☐ No time at all, I fall asleep very quickly
- ☐ A few minutes (5-10 minutes)
- ☐ A little while (10-30 minutes)
- ☐ A long time (more than 30 minutes)

11) What time do you usually wake up on weekdays?

- ☐ Before 5:00 am
- ☐ 5:00-5:29 am
- ☐ 5:30-5:59 am
- ☐ 6:00-6:29 am
- ☐ 6:30-6:59 am
- ☐ 7:00-7:29 am
- ☐ 7:30-7:59 am
- ☐ 8:00-8:29 am

The next set of questions has to do with your usual schedule on WEEKENDS or during the summer when you don't go to school.

12) What time do you usually go to bed on weekends?

- ☐ Before 8:00 pm
- ☐ 8:00-8:29 pm
- ☐ 8:30-8:59 pm
- ☐ 9:00-9:29 pm
- ☐ 9:30-9:59 pm
- ☐ 10:00-10:29 pm
- ☐ 10:30-10:59 pm
- ☐ 11:00-11:29 pm
- ☐ 11:30-11:59 pm
- ☐ 12:00-12:29 am
- ☐ 12:30-12:59 am
- ☐ After 1:00 am

15a. How often do you usually go to bed at this time?

- ☐ Both weekend nights
- ☐ One weekend night

13) Once you turn your light off on weekends, how long does it usually take you to fall asleep?

- ☐ No time at all, I fall asleep very quickly
- ☐ A few minutes (5-10 minutes)
- ☐ A little while (10-30 minutes)
- ☐ A long time (more than 30 minutes)

14) What time do you usually wake up on weekends?

- ☐ Before 6:00 am
- ☐ 6:00-6:29 am
- ☐ 6:30-6:59 am
- ☐ 7:00-7:29 am
- ☐ 7:30-7:59 am
- ☐ 8:00-8:29 am
- ☐ 8:30-8:59 am
- ☐ 9:00-9:29 am
- ☐ 9:30-9:59 am
- ☐ 10:00-10:29 am
- ☐ 10:30-10:59 am
- ☐ After 11:00 am

The next set of questions has to do with your usual sleep on most days (including both weekdays and weekends)

15) After you have gone to sleep at night, how often do you usually wake up during the night?

- ☐ Almost every night (5-7 times/week)
- ☐ Several times a week (1-4 times/week)
- ☐ Every now and then (2-3 times/month)
- ☐ I almost never wake up during the night

16) How long does it usually take you to go back to sleep after you wake up during the night?

- ☐ I usually don't wake up during the night
- ☐ No time at all, I go back to sleep very quickly
- ☐ A few minutes (5-10 minutes)
- ☐ A little while (10-30 minutes)
- ☐ A long time (more than 30 minutes)

17) Some kids take naps in the daytime every day, others never do. Do you nap?

- ☐ I never nap
- ☐ I never nap unless I am sick
- ☐ I sometimes nap
- ☐ I nap almost every day

18) Most nights, do you feel you get...

- ☐ too much sleep?
- ☐ the right amount of sleep?
- ☐ too little sleep?

19) Most nights, do you consider yourself to be...

- ☐ a great sleeper?
- ☐ a good sleeper?
- ☐ an okay sleeper?
- ☐ a poor sleeper?

(go to next page)

Think about how often the following things happen during a *regular week for you (not if you were sick or on vacation)*. For each one, please circle the answer that describes how often each question is true about you.

Circle Never if it never happens
Circle Not very often if it happens less than once a week
Circle Sometimes if it happens once or twice a week
Circle Usually if it happens 3-5 times a week
Circle Always if it happens every day

Here are two practice questions for you:

How often do you...					
A) Ride a bicycle	Never	Not very often	Sometimes	Usually	Always
B) Go to the library	Never	Not very often	Sometimes	Usually	Always

How often do you drink the following beverages...					
23) Regular or diet soda with caffeine (Coke, Pepsi, Dr. Pepper, Mountain Dew)	Never	Not very often	Sometimes	Usually	Always
24) Iced tea or hot tea (with caffeine)	Never	Not very often	Sometimes	Usually	Always
25) Coffee (with caffeine)	Never	Not very often	Sometimes	Usually	Always

During the hour before you go to bed how often do you....					
26) Have activities (sports, dance, music, other activities)	Never	Not very often	Sometimes	Usually	Always
27) Email or text with friends	Never	Not very often	Sometimes	Usually	Always
28) Watch television or movies	Never	Not very often	Sometimes	Usually	Always
29) Play video games or computer games	Never	Not very often	Sometimes	Usually	Always
30) Take a bath or shower	Never	Not very often	Sometimes	Usually	Always
31) Read books or magazines	Never	Not very often	Sometimes	Usually	Always

How often do you fall asleep at night in the following locations...					
32) Your sister's or brother's bed	Never	Not very often	Sometimes	Usually	Always
33) Your parents' bed	Never	Not very often	Sometimes	Usually	Always
34) Couch or other place (not your bed)	Never	Not very often	Sometimes	Usually	Always

When you are trying to fall asleep at night...					
35) Is a television on in your room?	Never	Not very often	Sometimes	Usually	Always
36) Are you listening to music?	Never	Not very often	Sometimes	Usually	Always
37) Is there a light on in your room (other than a nightlight)?	Never	Not very often	Sometimes	Usually	Always
38) Are you scared?	Never	Not very often	Sometimes	Usually	Always
39) Are you upset or worried?	Never	Not very often	Sometimes	Usually	Always
40) Are you thinking about that day or the next day which makes it hard to fall asleep?	Never	Not very often	Sometimes	Usually	Always

(go to next page)

Just a reminder...Circle **Never** if it never happens
Circle **Not very often** if it happens **less than once a week**
Circle **Sometimes** if it happens **once or twice a week**
Circle **Usually** if it happens **3-5 times a week**
Circle **Always** if it happens **every day**

How often do you wake up in the morning in the following locations...					
41) Your sister's or brother's bed	Never	Not very often	Sometimes	Usually	Always
42) Your parents' bed	Never	Not very often	Sometimes	Usually	Always
43) Couch or other place (not your bed)	Never	Not very often	Sometimes	Usually	Always

How often do you...					
44) Have funny feelings in your legs at bedtime or during the night (creepy-crawly, tingling, or soda bubbles)	Never	Not very often	Sometimes	Usually	Always
45) Feel like your legs bother you at bedtime or during the night	Never	Not very often	Sometimes	Usually	Always
46) Feel like you have to move your legs at bedtime or during the night	Never	Not very often	Sometimes	Usually	Always
47) Have trouble falling asleep at bedtime	Never	Not very often	Sometimes	Usually	Always
48) Wake up during the night	Never	Not very often	Sometimes	Usually	Always
49) Wet the bed	Never	Not very often	Sometimes	Usually	Always
50) Have bad dreams	Never	Not very often	Sometimes	Usually	Always
51) Wake in the morning very thirsty	Never	Not very often	Sometimes	Usually	Always
52) Wake in the morning with a headache	Never	Not very often	Sometimes	Usually	Always

How often do you feel sleepy or fall asleep when you are...					
53) Eating	Never	Not very often	Sometimes	Usually	Always
54) Talking with someone else	Never	Not very often	Sometimes	Usually	Always
55) At school	Never	Not very often	Sometimes	Usually	Always
56) Playing	Never	Not very often	Sometimes	Usually	Always
57) Riding in a car or bus for a short time (less than 20 minutes)	Never	Not very often	Sometimes	Usually	Always

Has anyone ever told you that...			
58) You snore	All the time	Sometimes	Never
59) You kick your legs when you are sleeping	All the time	Sometimes	Never
60) You move a lot in your sleep	All the time	Sometimes	Never
61) You talk in your sleep	All the time	Sometimes	Never
62) You walk around or cry out when you sleep	All the time	Sometimes	Never

THANK YOU FOR FILLING OUT THIS QUESTIONNAIRE!!

APPENDIX I

CHILDREN'S REPORT OF SLEEP PATTERNS (CRSP) – PARENT FORM

Instructions: this form asks questions about your child's sleep. Please answer questions as honestly as you can. <u>There are no right or wrong answers.</u> Do not spend too much time on any questions; your first answer is usually the best. Choose from each question the answer that best describes your child by putting a mark like this [X] next to your answer. Put the mark in the box next to the answer that you pick. <u>Only choose one answer for each question.</u>

1. **What time did your child go to sleep last night?** _____ am pm

2. **Once your child turned the light off, how long did it take your child to fall asleep <u>last night?</u>**

[ ] No time at all, my child fell asleep very quickly
[ ] A few minutes (5-10 minutes)
[ ] A little while (10-30 minutes)
[ ] A long time (more than 30 minutes)

3. **Did your child take any medication to help them sleep <u>last night?</u>** YES NO

If yes, what medication did your child take? _____

4. **After your child fell asleep, did your child wake up during the night?** YES NO

5. **How long did it take your child to go back to sleep after your child woke up during the night?**

[ ] My child did not wake up last night
[ ] No time at all, my child went back to sleep very quickly
[ ] A few minutes (5-10 minutes)
[ ] A little while (10-30 minutes)
[ ] A long time (more than 30 minutes)

6. **What time did your child wake up today?** _____ am pm

7. **How did your child wake up this morning?**

[ ] My child woke up by themselves
[ ] My child woke up with an alarm clock
[ ] Someone in the family woke them up
[ ] A pet woke them up

8. **How well did your child sleep last night?**

[ ] Great
[ ] Good
[ ] Okay
[ ] Poor

9. **What time does your child <u>usually</u> go to bed on weekdays?** _____ am pm

10. How often does your child usually go to bed at this time?

[ ] Every night
[ ] Several times a week (3-4 nights)
[ ] Every now and then (1-2 nights)

11. Once your child turns the light off on weekdays, how long does it <u>usually</u> take your child to fall asleep?

[ ] No time at all, my child falls asleep very quickly
[ ] A few minutes (5-10 minutes)
[ ] A little while (10-30 minutes)
[ ] A long time (more than 30 minutes)

12. What time does your child <u>usually</u> wake up on weekdays? _____ am pm

13. What time does your child <u>usually</u> go to bed on weekends? _____ am pm

14. How often does your child usually go to bed at this time?

[ ] Both weekend nights
[ ] One weekend night

15. Once your child turns the light off on weekends, how long does it usually take your child to fall asleep?

[ ] No time at all, my child falls asleep very quickly
[ ] A few minutes (5-10 minutes)
[ ] A little while (10-30 minutes)
[ ] A long time (more than 30 minutes)

16. What time does your child <u>usually</u> wake up on weekends? _____ am pm

17. After your child has gone to sleep at night, how often does your child <u>usually</u> wake up during the night?

[ ] Almost every night (5-7 times/week)
[ ] Several times a week (1-4 times/week)
[ ] Every now and then (2-3 times/month)
[ ] My child almost never wakes up during the night

18. How long does it <u>usually</u> take your child to go back to sleep after your child wakes up during the night?

[ ] My child usually doesn't wake up during the night
[ ] No time at all, my child went back to sleep very quickly
[ ] A few minutes (5-10 minutes)
[ ] A little while (10-30 minutes)
[ ] A long time (more than 30 minutes)

19. Some kids take naps in the daytime every day, others never do. Does your child nap?

[ ] My child never naps
[ ] My child never naps unless my child are sick
[ ] My child sometimes naps
[ ] My child naps almost every day

20. <u>Most nights</u>, do you feel your child gets…

[ ] too much sleep
[ ] the right amount of sleep
[ ] too little sleep

21. <u>Most nights</u>, do you consider your child to be…

[ ] A great sleeper
[ ] A good sleeper
[ ] An okay sleeper
[ ] A poor sleeper

Instructions: Think about how often the following things happen during a *<u>regular week for you child (not if s/he is sick or on vacation).</u>* For each one, please circle the answer that describes how often each question is true about your child.

Circle **Never** if it **never happens**
Circle **Not very often** if it happens **less than once a week**
Circle **Sometimes** if it happens **once or twice a week**
Circle **Usually** if it happens **3-5 times a week**
Circle **Always** if it happens **every day**

<u>How often does your child drink the following beverages…</u>					
23) Regular or diet soda with caffeine (Coke, Pepsi, Dr. Pepper, Mountain Dew)	Never	Not Very Often	Sometimes	Usually	Always
24) Iced tea or hot tea (with caffeine)	Never	Not Very Often	Sometimes	Usually	Always
25) Coffee (with caffeine)	Never	Not Very Often	Sometimes	Usually	Always

<u>During the hour before your child goes to bed how often does s/he…</u>					
26) Have activities (sports, dance, music, other activities)	Never	Not Very Often	Sometimes	Usually	Always
27) Email or text with friends	Never	Not Very Often	Sometimes	Usually	Always
28) Watch television or movies	Never	Not Very Often	Sometimes	Usually	Always
29) Play video games or computer games	Never	Not Very Often	Sometimes	Usually	Always
30) Take a bath or shower	Never	Not Very Often	Sometimes	Usually	Always
31) Read books or magazines	Neve	Not Very Often	Sometimes	Usually	Always

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How often does your child fall asleep at night in the following locations…					
32) A sibling's bed	Never	Not Very Often	Sometimes	Usually	Always
33) Parents' bed	Never	Not Very Often	Sometimes	Usually	Always
34) Couch or other place (not your child's bed)	Never	Not Very Often	Sometimes	Usually	Always

When your child is trying to fall asleep at night...					
35) In a television on in your child's room?	Never	Not Very Often	Sometimes	Usually	Always
36) Is your child listening to music?	Never	Not Very Often	Sometimes	Usually	Always
37) Is there a light on in your child's room (other than a nightlight)?	Never	Not Very Often	Sometimes	Usually	Always
38) Is your child scared?	Never	Not Very Often	Sometimes	Usually	Always
39) Is your child upset or worried?	Never	Not Very Often	Sometimes	Usually	Always
40) Is your child thinking about that day or the next day which makes it hard to fall asleep?	Never	Not Very Often	Sometimes	Usually	Always

How often does your child wake up in the morning in the following locations					
41) A sibling's bed	Never	Not Very Often	Sometimes	Usually	Always
42) Parents' bed	Never	Not Very Often	Sometimes	Usually	Always
43) Couch or other place (not your child's bed)	Never	Not Very Often	Sometimes	Usually	Always

How often does your child					
44) Have funny feelings in her/his legs at bedtime or during the night (creepy-crawly, tingling, or soda bubbles)	Never	Not Very Often	Sometimes	Usually	Always
45) Feel like her/his legs bother her/him at bedtime or during the night	Never	Not Very Often	Sometimes	Usually	Always
46) Feel like s/he has to move her/his legs at bedtime or during the	Never	Not Very Often	Sometimes	Usually	Always

night					
47) Have trouble falling asleep at bedtime	Never	Not Very Often	Sometimes	Usually	Always
48) Wake up during the night	Never	Not Very Often	Sometimes	Usually	Always
49) Wet the bed	Never	Not Very Often	Sometimes	Usually	Always

How often does your child					
50) Have bad dreams	Never	Not Very Often	Sometimes	Usually	Always
51) Wake in the morning very thirsty	Never	Not Very Often	Sometimes	Usually	Always
52) Wake in the morning with a headache	Never	Not Very Often	Sometimes	Usually	Always
How often does your child fall asleep when s/he is…					
53) Eating	Never	Not Very Often	Sometimes	Usually	Always
54) Talking with someone else	Never	Not Very Often	Sometimes	Usually	Always
55) At school	Never	Not Very Often	Sometimes	Usually	Always
56) Playing	Never	Not Very Often	Sometimes	Usually	Always
57) Riding in a car or bus for a short time (less than 20 minutes)	Never	Not Very Often	Sometimes	Usually	Always

Has anyone ever told your child that s/he…			
58) Snores	All The Time	Sometimes	Never
59) Kicks her/his legs when s/he is sleeping	All The Time	Sometimes	Never
60) Moves a lot in her/his sleep	All The Time	Sometimes	Never
61) Talks in her/his sleep	All The Time	Sometimes	Never
62) Walks around or cries out when she/he sleeps	All The Time	Sometimes	Never

APPENDIX J

LIFE EVENTS CHECKLIST

<u>Directions</u>: If an event mentioned below has occurred to anyone in your home within the past year, or is expected in the near future, write the number of times the event has occurred in the "# of times" column.

Event	# of times
Death of spouse	
Divorce	
Marital Separation	
Jail Term	
Death of close family member	
Personal injury or illness	
Marriage	
Fired at work	
Marital reconciliation	
Retirement	
Change in health of family member	
Pregnancy	
Sex difficulties	
Gain of a new family member	
Business readjustment	
Change in financial state	
Death of a close friend	
Change to a different line of work	
Change in number of arguments with spouse	
Mortgage over $20,000	
Foreclosure of mortgage or loan	
Change in responsibilities at work	
Son or daughter leaving home	
Trouble with in laws	
Outstanding personal achievement	
Spouse begins or stop work	
Begin or end school	
Change in living conditions	
Revisions of personal habits	
Trouble with boss	
Change in work hours or conditions	
Change in residence	
Change in schools	
Change in recreations	
Change in church activities	
Change in social activities	
Mortgage or loan less than $20,000	

Change in sleeping habits	
Change in number of family get-togethers	
Change in eating habits	
Vacation	
Christmas/Other Holiday approaching	
Minor violation of the law	

VITA

Graduate School
Southern Illinois University

Gabriel A. Casher

Gabriel.casher@gmail.com

Hope College
Bachelor of Arts, Psychology, May 2013

Southern Illinois University Carbondale
Master of Arts in Psychology, May 2016

Special Honors and Awards:

Sigma Xi Rose and Essie Padgett Research Award, Southern Illinois University	2017
Doctoral Fellowship, Southern Illinois University Graduate School	2017-2018
Outstanding Graduate Teaching Assistant Award, Southern Illinois University	2016
Doctoral Fellowship, Southern Illinois University Graduate School	2016-2017
Master's Fellowship, Southern Illinois University Graduate School	2015-2016

Dissertation Paper Title:

> Longitudinal Relationships Between Sleep Problems and Externalizing Behavior in Children: Investigating Genetic and Temperamental Moderators

Major Professor: Dr. Lisabeth Dilalla

Publications:

Casher, G.A., Gagnon, K., & Simpson, S.A. (2018). Integrated behavioral health in the pediatric emergency department: Program development, implementation, and future directions. *Journal of the American Association of Emergency Psychiatry, Winter.*

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