

EXPLORING SOURCES OF THE ASSOCIATION BETWEEN SLEEP AND RISK-  
TAKING FROM LATE-CHILDHOOD INTO ADOLESCENCE

Erika J. Bagley

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Dissertation committee members:

Dr. Martha Cox (chair)

Dr. Vonnie C. McLoyd

Dr. Jean-Louis Gariepy

Dr. Cecelia Valrie

Dr. Susan Ennett

Dr. Rita Fuchs Lokensgard

## ABSTRACT

ERIKA J. BAGLEY: Exploring Sources of the Association between Sleep and Risk-taking  
from Late-Childhood into Adolescence  
(Under the direction of Martha Cox, Ph.D.)

This doctoral dissertation seeks to provide a deeper understanding of the association between sleep variables (sleep duration, variability and problems) and risk-taking behaviors (general delinquency, substance use and sexual risk-taking) from late childhood into adolescence. The study examines the change over time in sleep and risk-taking separately and the association between these variables over time. The influences of pubertal development and parental monitoring on the association between sleep and risk-taking are considered. Finally, executive functioning and impulse control are explored as possible mediators that may explain the association. Data for the study are drawn from the National Institutes of Child Health and Development Study of Early Child Care and Youth Development (NICHD SECCYD) sixth grade and 15 year data collections.

As predicted, sleep variables demonstrated changes from sixth grade to 15 years that reflected worsening sleep with decreased sleep durations, increased variability, and increased sleep problems. Earlier pubertal development and greater parental monitoring were related to greater increases in sleep problems, but not other sleep variables. General risk-taking and substance use also increased over the period from sixth grade to 15 years. Greater parental monitoring was predictive of less increase in general risk-taking and substance use behaviors. Pubertal development was not predictive of change in risk-taking.

All of the sleep variables were associated with general risk-taking, but not other risk-taking variables. Parental monitoring, but not pubertal development, moderated the effect of sleep on risk-taking. Executive functioning did not function as a mediator of the relationship between sleep and risk-taking. Impulse control did, however, play a mediating role, partially explaining the effect of sleep on risk-taking behaviors. Developmental systems theory frames the discussion of results, limitations and future directions.

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## INTRODUCTION

In recent years the interest in adolescent sleep has grown, spurred by research showing the negative effects that poor or inadequate sleep and irregular sleep patterns can have on cognitive and behavioral functioning (Gregory & O'Connor, 2002 & Friedman, Corley, Hewitt & Wright, 2009). Among this growing body of literature are findings from psychology, psychiatry and public health that show an association between sleep and risk-taking behaviors in adolescents (Mindell & O'Brien, 2006). Researchers have suggested that the deleterious effects of sleep on cognitive abilities, particularly executive functioning, may provide a mechanism by which sleep and risk-taking behaviors are related (Wong, Brower, Nigg & Zucker, 2010). Indeed, prominent models of risk-taking which are informed by advances in developmental neuroscience, suggest that there are deficits in self-regulatory abilities and reward processing that may explain the increases in risk-taking that occur during adolescence (Steinberg, 2010). However, although it may be possible that links between sleep and risk-taking are mediated by cognitive or reward-processing abilities, little work in the area has sought to explore this or other possible mediators, moderators and third variables that could explain the relationship. This is somewhat surprising given the overlap in known predictors of both poor sleep and risk-taking. Further, few distinctions have been made in previous research between measures of sleep and different types of risk-taking behaviors (Gregory, Ely, O'Connor & Plomin, 2004). This omnibus approach to understanding how poor sleep is related to risk-taking may obscure important information that could provide insights into the underlying mechanisms at work. The goal of the proposed research is to

expand our understanding of the associations between sleep and risk-taking by: (a) considering the role of cognitive, biological and family level variables as possible mediators and moderators of the relationship, (b) using multiple measures of risk-taking and sleep, and (c) using longitudinal design that will take into account changes across development.

Research in the area of sleep and behavior is surprisingly young and has lacked a guiding framework necessary to allow for efficient accumulation of knowledge on the topic. This shortcoming has not gone without notice. In a recent review of the literature looking at the relationship between sleep and daytime behavior in children, Sadeh (2009) concluded that the majority of research in the area has been atheoretical, partially given to the fact that there is still little agreement on the actual function of sleep. Modest attention has been paid to issues such as bidirectionality and potential timing effects on the association of sleep and behavior. By relying on single time point analyses, cascades overtime, in which poor sleep begets risk-taking and in turn leads to worsening sleep, may have been overlooked. The correlational work to date may also ignore the cumulative influence of sleep on daytime behavior, the importance of other maturational processes, or the particular susceptibility of developing biological, cognitive and regulatory systems to the negative effects of inadequate or poor sleep. The proposed study will allow for testing of the relations between sleep and risk-taking behavior at sixth grade and 15 year data collection time points. While it may be preferable to have more intervening time points, the proposed study captures an important transition from a period when sleep is generally considered good (sixth grade) to a period in which sleep patterns are undergoing many changes and sleep durations are often inadequate (15 year). The study design allows for testing the influence of the change over time in sleep, presenting a significant advantage over single time-point analyses.



Little has been done to date to consider possible mediators or moderating contextual factors that bear on the association between sleep and risk-taking behavior. Within the literature review of this paper, I will pull from a number of separate literatures to suggest that executive functioning, impulse control, pubertal timing and parental monitoring may have influences on the association. There are a number of possible roles that these variables may play and, without inclusion in analyses, our understanding of the true relationship between sleep and risk-taking is likely biased. Broadly, I describe these factors as possible third variables; they may be best understood as mediators, moderators and/or co-variates. The goal of mediational analyses in the proposed study will be to explain why sleep and risk-taking are associated. Evidence of mediation supports a causal model in non-experimental research, such as this, although one cannot fully rule out other alternatives. Moderational analyses in the proposed study will provide insights into the conditions under which the association between sleep and risk-taking is strongest. It is entirely possible that the previous research that has not considered moderation has obscured the influence of contextual factors on the relationship between sleep and risk-taking to the extent that it may only exist under certain conditions or for certain individuals. Finally, by including a variable as a covariate one suggests that its effect biases our understanding of the true relationship between sleep and risk-taking and, therefore, should be controlled for. If controlling for a factor eliminates the relationship between sleep and risk-taking, and it is not better understood as a mediator or moderator, then one might conclude that the common association drove the relationship between sleep and risk-taking and that a causal link does not exist.

Not unrelated to the goal of uncovering mediational and moderational processes, the proposed study will investigate the extent to which different sleep indices reveal different

pieces of information about the relationship between sleep and risk-taking behaviors. Across the reviewed studies, sleep has been measured objectively using polysomnography and actigraphy and measured subjectively using self and parent report measures. These measures can be and have been used to create a large number of variables that describe sleep quantity (duration), quality (sleep problems, daytime sleepiness, difficulty falling/staying asleep), and variability (day to day or weeknight to weekend differences in onset, mid-sleep or wake time). Previous research has rarely tested the separate influences of different aspects of sleep on risk-taking behavior within a single study. As a result, many questions are raised about the relative importance of particular sleep parameters with very practical significance. For example, if research shows that sleep duration is most predictive of behavior, above and beyond all other aspects of sleep, then practitioners would be wise to suggest that the optimal sleep duration be reached nightly, regardless of the time of sleep onset or wake time. On the other hand, if research finds that variability in sleep patterns is most predictive of negative daytime behavior, it would have much different clinical implications. Further, by gaining a better understanding of the unique effects of aspects of sleep on particular behaviors it is possible that mechanisms may be revealed. To this end, the proposed study will significantly contribute to the current literature by examining multiple sleep variables and risk-taking in the context of the real-world and laboratory setting.

This document will provide background on the existing correlational and experimental literature that considers the association between sleep and risk-taking. I will review the few studies that have explored possible mediators of the relationship but also discuss commonalities across the two separate literatures on problem sleep and risk-taking regarding predictors and correlates of these behaviors. Specifically, I will address executive

functioning, impulse control, pubertal timing, and parental monitoring as mediators or moderators that may account for the relationship between sleep and risk-taking during adolescence. I will provide an overview of the proposed study, which will investigate possible mediators and moderators and describe the data source and measures to be used. Finally, I will detail the hypotheses for this study and analytic plans for the data.

*Co-occurrence of inadequate and problematic sleep and risk-taking*

Adolescence marks a period of biological, cognitive and social transitions. Researchers interested in this stage of development have often tried to understand the interaction between the changes that occur on various levels and ways in which those interactions lead to the unique behaviors that epitomize what it is to be a teenager. This paper and proposed study will focus on two common phenomena of adolescence, the tendency to receive inadequate or problematic sleep and increases in risk-taking. Separately, poor sleep and risk-taking are of great interest to those in the areas of public health, medicine, and psychology. It is estimated that almost half of adolescents receive inadequate sleep on weeknights (Carskadon, Mindell, & Drake, 2006), potentially leading to chronic sleep deprivation and having effects on cognitive abilities, mental and physical health. Risk-taking during adolescence is common in many forms and, according to a recent CDC report, is directly or indirectly responsible for 72% of deaths in individuals aged 10-24 (Eaton et al., 2008). However, it is only recently that researchers have seriously started to investigate the possibility that problems with sleep and increased risk-taking might be causally related to each other during adolescence, rather than just occurring at a similar time in development.

Sleep research with adolescents suggests that changes in sleep patterns occur in this period involving primarily a decline in quality of sleep from childhood (Sheldon, Spire &

Levy, 1992, Iglowstein et al., 2003). The early work that examined normative changes in sleep patterns during adolescence has revealed that duration, daytime sleepiness, and sleep problems together pose significant influences on daytime functioning of adolescents (Carskadon, 1990, Wolfson & Carskadon, 1998). In particular, research has suggested that adolescents who report problems falling asleep and maintaining sleep show more inattentive, depressed and conduct disordered behaviors compared to those who report no, or occasional, sleep problems (Morrison, McGee, & Stanton, 1992, Carskadon, 1996). Links between general externalizing behavior problems and sleep have been established in clinical populations of adolescents with ADHD and sleep apnea (Cortese, Konofal & Yatemen, 2006, Alfano & Gamble, 2009). Most convincing findings from this work regarding the association between sleep and daytime behavior have demonstrated decreases in externalizing behavior problems resulting from interventions that improved sleep duration or quality (Chervin et al., 2006).

Much less work has specifically considered the relationship between sleep and risk-taking behavior, although prior research certainly supports the hypothesis that they would be related. Research by O'Brien and Mindell (2005) represents the first published study that primarily examined the relationship between sleep and risk-taking specifically in adolescents. In this study, 388 high school students responded to the Sleep Habits and Youth Risk Behavior surveys. O'Brien and Mindell found that students with the greatest differential between sleep times on weekdays versus weekends (weekend sleep delay) reported the highest use of tobacco, alcohol, and marijuana, and the highest participation in risky sexual behaviors. Further, scores on the Sleep-Wake Behavior Problem subscale of the Sleep Habits survey were significantly and positively associated with risky sexual behaviors. This

research substantiated a relationship between aspects of sleep and risk-taking behavior, but raised questions about the extent to which this relationship is driven by a direct effect of sleep on risk-taking or vice versa rather than another third variable. For example, it is entirely possible that the relationship exists because sexual risk-taking often occurs during weekend nighttime hours, when one might be otherwise sleeping. It also raised questions about how sleep may be functioning given that significance was not reached for relations between total sleep time and risk-taking behaviors, but only for variability in sleep patterns and problems.

Another recent study has suggested that sleep patterns, and not simply sleep duration, may shed additional light on the sleep-risk-taking link. Pasch, Laska, Lytle and Moe (2010), using self-report surveys of sleep habits, truancy and substance use in a largely white sample of ninth to eleventh grade adolescents, found that later weekend bedtimes/wake times were positively associated with substance use and truancy. Longer weekday (but not weekend) sleep durations, on the other hand, were associated with less alcohol use and fewer reports of drunkenness. Individuals with greater variability in sleep patterns between weekdays and weekends in their study showed the highest odds ratio of risk-taking behavior. The authors concluded that weekday sleep may have protective effects on adolescents leading to fewer risk-taking behaviors around substance use.

Finally, Caterett and Gaultney (2009) used the nationally-representative Add Health dataset to examine concurrent and longitudinal relations between symptoms of insomnia and risk-taking across middle school and high school. Because sleep problems and depression often co-occur and both depression and poor sleep have been linked with risk-taking behaviors, the authors were interested in understanding whether the relations between sleep problems and risk-taking would be significant even after accounting for the association of

depressive symptoms and other demographic variables such as gender and age with risk-taking. They found within wave support for the significant association between symptoms of insomnia and smoking and delinquency, but failed to find these relationships longitudinally. Catrett and Gaultney found no support for the possibility that the association was the result of underlying depressive symptoms, but just as in O'Brien and Mindell's study, many other possible third variables remain unexplored.

In addition to these three correlational studies, evidence of the association between sleep and risk-taking has also come from researchers who have used clustering strategies to understand patterning of a broad range of health-compromising and delinquent behaviors, including receiving inadequate sleep. The fact that risk-taking behaviors are likely to co-occur is not new (Jessor, 1992), however it is more recent that researchers have used both clustering and latent class analytical techniques to describe the co-occurrence of problem behaviors and understand differences in patterns of clustering based on a variety of demographic variables (such as age or gender). In some cases, these studies have revealed that problem behaviors with which sleep is associated may change across development. Van Nieuwenhuijzen and colleagues (2009) ran separate cluster analyses for young adolescents (12-15 years) and late adolescents (16-18 years). Their findings showed that short sleep durations clustered with alcohol, tobacco and drug use during early adolescence, but clustered with a broader category of delinquency during late adolescence. In a comparable study that sampled among slightly older individuals (18-25 years), Laska, Pasch, Lust and Story (2009) used latent class analysis to identify mutually exclusive patterns of health risk behavior, focusing on gender as a moderator. Despite finding homogenous classes that described unique patterns of health risk taking for male and female participants, it was

notable that inadequate sleep was likely to occur within all of the risk groups regardless of gender. Similarly, Sullivan, Childs and O'Connell's (2010) latent class analysis based on risk-taking behaviors of high school students found that average hours of sleep on school nights was a significant correlate of every risk group.

The findings from clustering approaches have added to this literature by describing distinct, naturally occurring patterns of risk-taking and changes in those patterns across development. Importantly for the focus of this paper, this type of descriptive analysis is additional confirmation that sleep problems or inadequate sleep often co-occur with high-risk behaviors. What has not been revealed by these strategies are the reasons for the association and whether or not sleep is playing a central role in the development of risk-taking behaviors.

*Sleep and substance use.* The previous section reviewed research that considered associations between various aspects of sleep and broad categories of risk-taking behaviors. However, another line of research has focused on the association between sleep and substance use. In fact, a significant association between sleep and substance use in adulthood has long been acknowledged, leading to the conclusion that there may be important direct and indirect, reciprocal influences contributing to a dangerous cycle of abuse. Brower (2001) has suggested that in alcoholics this cycle can often start with self-medicating for sleep problems with alcohol, which with increasing tolerance leads to the need for more alcohol to achieve desired effects, and in turn brain systems responsible for regulation of sleep are disrupted. However, most models that describe the relationship between sleep and substance use are based on findings from adult populations only. Far fewer studies have considered these relationships in adolescent populations.

In one of the few studies to do so, Johnson and Breslau (2001) used the US National Household Survey on Drug Abuse 1994-1996, a nationally-representative sample of over 13,000 adolescents, to examine the association between sleep problems and substance use while controlling for a variety of demographic variables. Their findings are consistent with adult work showing a significant relationship between self-reported sleep problems and various forms of substance use (tobacco, alcohol and illicit drugs). Significant interactions were found for gender, indicating that the relationships between sleep problems and substance use were stronger for females in the sample. With the exception of illicit drug use at the most frequent level, the relationships between sleep problems and substance use remained significant, albeit reduced, after controlling for externalizing and internalizing behavior problems. Interestingly, though, the inclusion of externalizing and internalizing behavior problems as co-variates eliminated the significant interactions for gender. In sum, the findings from Johnson and Breslau's work suggest that the relationships between sleep problems and substance abuse are complex, moderated by gender and influenced by other existing behavior problems. However, the use of cross-sectional data in this study leaves many questions about the role of sleep in substance use unanswered.

A recent study by Wong, Brower and Zucker (2009) made use of longitudinal data to address similar questions about the relationship between sleep problems in childhood (3-8 years old) and substance use during adolescence (15-17 years old). The ability to look at the association between sleep and substance use prospectively represents a significant advantage over previous studies. However, the sample from this study perhaps limits the generalizability of findings in that it was drawn from an ongoing longitudinal study of Caucasian families in which the father was identified as an alcoholic. Taking into account the



limitations of their high-risk sample, the authors were able to provide evidence for the association between childhood sleep problems and multiple forms of adolescent substance use (tobacco, alcohol and marijuana), but only for males in the sample. This gender finding seems to stand in contrast to the earlier work of Johnson and Breslau and may be the result of unique characteristics of the sample or of the timing of sleep problems in girls versus boys. In terms of the role of externalizing and internalizing behavior problems, the authors found that although childhood sleep problems were related to internalizing and externalizing behavior problems during adolescence, the association between childhood sleep problems and substance use was not affected when steps were taken to adjust for behavior problems.

One innovative approach to understanding the role of sleep problems in substance use has been to study children of alcoholics, who themselves are at high risk of developing alcoholism across their lifetime. It is thought that unique characteristics of children of alcoholics might reveal underlying factors that contribute to the onset and maintenance of alcohol use. This method allows researchers to isolate contributing factors that may occur before the onset of substance use and thereby circumvent problems related to understanding the reciprocal relations between sleep and substance use. In a recent publication, Tarokh and Carskadon (2010) compared polysomnography data from 9 to 10 year old children of alcoholics and normal controls. They found significant differences in the EEG's of children of alcoholics that indicate impaired ability to maintain sleep. The authors hypothesize that these differences may lead to disrupted sleep later in life and may be an underlying biological mechanism that could explain the heritability of alcoholism. The authors chose to look at a sample of prepubescent youth to eliminate confounds related to changes in sleep that occur during puberty. However, it may also be important to consider that the vulnerability of sleep

during the biological and social transitions of puberty might exacerbate preexisting abnormalities in the sleep of children of alcoholics.

Taken together, the cross-sectional, longitudinal and prospective studies all suggest that there is indeed a significant link between sleep and substance use during adolescence. In the long run, the research that looks at the potentially bi-directional relationship between sleep and substance use may lead to a more nuanced understanding of the conditions under which the relationship is the strongest and for whom. The limited research available does suggest that this relationship should be considered in the context of other behavioral problems and in consideration of gender. It is unknown whether the work in this area will be specific to mechanisms that underlie substance use only, or may possibly expand our understanding of the sleep risk-taking relationship as a whole. This work does raise important questions about the wisdom of looking at risk-taking as a single construct. If the relationship between sleep and substance use is driven by the effects, or perceived effects, of those substances on sleep, then the same mechanisms would not equally apply to other types of risk-taking.

#### *Explanations for the sleep and risk-taking link*

The research that has been summarized to this point has documented an association between sleep and risk-taking behaviors during adolescence. A much more difficult task lays in explaining the association. In the discussion that follows, I take two approaches to address this issue. First, I will discuss the possibility of a mediational model suggesting that sleep indirectly affects risk-taking through some effect that sleep has on functioning which in turn increases risk-taking. Secondly, I discuss the possibility that a third variable, important to sleep and risk-taking, may be an important moderator to consider.

*Executive functioning and impulse control.* As suggested in the introduction of this paper, the effect of sleep on cognitive performance has been implicated as a mediator that explains the relationship between sleep and risk-taking (Wong, Brower, Nigg & Zucker, 2010). Specifically, the deleterious effects of inadequate sleep can have on activity in the prefrontal cortex, primarily executive functioning, has been central to many of the proposed mediational models. Although few empirical studies are available to support these models, they have great appeal in light of the current thinking about adolescent risk-taking (Steinberg, 2010) and the fact that much of the research that has considered the effect of sleep on functioning has concluded that the functioning of the prefrontal cortex may be particularly susceptible to sleep loss (Killgore, Balkin & Wesenten, 2006). In the following section, I briefly review these two separate findings and summarize the very few studies that have tested mediational models linking sleep and risk-taking.

One of the most prominent theories of risk-taking posits that risk-taking behaviors of adolescents can be understood as a result of the co-occurrence of a heightened socio-emotional system seeking rewards and a still immature cognitive control system to keep the reward seeking in check (Steinberg, 2008). Some of the strongest evidence for this argument, at least in terms of the cognitive control pathway, comes from a growing understanding of the structural and functional maturation of the prefrontal cortex throughout adolescence (Van Leijenhorst, Moor, Macks, Rombouts, Westenberg & Crone, 2010, Casey, Getz & Galvan, 2008, Crone, et al., 2006, Casey, Giedd & Tomas, 2000, Spear, 2000). This work uses advances in fMRI and other brain imaging technologies to suggest that adolescent risk-taking and the subsequent decline in risk-taking that is observed into adulthood are a result of changes in executive functioning, which comes about as the prefrontal cortex matures.

A recent study by Van Leijenhorst and colleagues (2010) looked cross-sectionally at developmental changes in brain activation in individuals aged 8 to 26 (n=58) under conditions that demanded high or low risk taking on a gambling, lab-based task. Their study tested the 2 pronged theory that activity in reward related areas of the brain (prefrontal cortex and ventral striatum) would show an inverted U-shape from childhood into early adulthood, with a peak during adolescence and that a linear pattern would be observed in cognitive control areas of the brain (dorsal lateral prefrontal cortex) across time. By comparing brain activity when individuals were engaged in low and high reward tasks and the effect of age on risk-taking behaviors, the findings of this study suggest that during adolescence the prefrontal cortex appears more sensitive to reward and only moderately able to regulate behavior, leading to risky decision making. This study provides strong evidence for the basis of the dual-pathway theory of adolescent risk taking.

Indeed, evidence from neurobiological and behavioral studies in support of the dual systems model has been accumulating over the past few years. Gianotti and colleagues (2009) used resting state EEG and brain imaging to observe differences in cortical activity of the prefrontal cortex, finding that participants who displayed cortical hypoactivity in the right prefrontal cortex had higher propensities for risk-taking on a lab-based task. They concluded from this research that hypoactivity in this area of the brain may represent a disposition towards poor regulation and risk-taking. An intriguing pair of studies has experimentally modulated (Fecteau, Knoch, Fregni, Sultani, & Boggio, 2007) and suppressed (Knoch, Gianotti, Regard & Brugger, 2006) activity in the prefrontal cortex using transcranial brain stimulation. The results of these studies showed that externally inducing changes in

prefrontal activity lead to changes in risk-taking behavior on a lab-based task in the hypothesized directions.

Possibly related to these findings of increased reward sensitivity and poor cognitive control during adolescence, other research has suggested that executive functioning may have indirect effects on real-world risk-taking by affecting the tendency to act impulsively (Romer, Betancourt, Giannetta & Brodsky, 2009). Impulsivity, as a personality trait, has long been considered a predictor of risk-taking behaviors (Caspi et al., 1997, Zuckerman & Kuhlman, 2000), but it is more recent that impulse control has been integrated into models of risk-taking as a correlate of executive function. In a community sample of almost 400 preadolescents, Romer and colleagues (2009) found support for a correlation between executive functioning (as measured by the Stroop task) and impulse control, defined as sensation seeking with lack of planning. Further, using structural equation modeling, impulse control was found to be significantly and positively related to risk-taking initiation (including alcohol use, gambling, and fighting), although executive functioning was not directly related to risk-taking. Certainly, more work needs to be done to operationally define the construct and clarify the components of executive function that contribute to impulse control, but even as the way in which impulsivity is conceptualized changes from fixed personality trait to a malleable characteristic, it remains an important consideration in prominent models of adolescent risk-taking.

The second set of findings that suggest a mediational model linking sleep and risk-taking through executive functioning concerns the effects of sleep loss on cognitive performance. Prior research provides convincing evidence that insufficient sleep has negative effects on cognitive performance, particularly in domains considered part of executive

functioning. A recent review of over twenty studies that used functional neuroimaging to understand the effects of sleep loss found that substantial evidence exists to conclude that sleep is critical for optimal functioning in the domains of working memory, inhibition, and attention (Chee & Chuah, 2008). In studies of acute sleep deprivation, attention and decision making appear to be greatly affected, even when participants did not report feeling sleepy (Harrison & Horne, 2000, Van Dongen et al., 2003, Killgore, Balkin & Wesensten, 2006). However it should be noted that the bulk of work that has looked at sleep and cognitive functioning in humans has been conducted using adults and using experimental designs that induce acute sleep deprivation (of up to 49 hours).

The models that suggest that sleep is related to risk-taking through effects on executive functioning are more convincing when paired with evidence showing that even modest sleep loss can have effects for children and adolescents. A study by Sadeh, Gruber and Raviv (2003) provides such evidence. In one of the few experimental studies of the consequences of sleep loss in children, researchers attempted to answer the question of “what difference does an hour make?”. The normal sleep patterns of 77 children (ages 10-12) were assessed using actigraphy for two consecutive nights. On the second day, neurobehavioral functioning was assessed through a series of tasks, including reaction time, sustained attention, processing speed and working memory tests. On the third evening, half the sample was asked to restrict sleep by 1 hour and the other half was asked to extend sleep by 1 hour. Finally, on day 6, 3 days after the sleep duration manipulation started, the neurobehavioral functioning tasks were repeated. The experimental manipulation of sleep resulted in an average of 86 minutes difference in duration between the restricted and extended groups, approximately 40 minutes +/- individual baseline levels. In a comparison of performance

between sleep restricted and sleep extended groups, the researchers found that tasks that required quick reaction were particularly affected by this modest sleep restriction. The continuous performance test, which assesses sustained visual attention, response inhibition, and motor speed, was one of the tests that was most sensitive sleep restriction and extension. The findings of this study suggest that even one hour difference in sleep can make a considerable difference in cognitive performance, particularly on tasks that are related to executive functioning. In conclusion, the authors believed that the results supported the suggestion that the prefrontal cortex of children is uniquely vulnerable to small changes in sleep duration.

Taken together, the research suggesting an important role of the prefrontal cortex in risk-taking and the particular sensitivity of the prefrontal cortex to sleep loss has led some to the conclusion that sleep and risk taking are related through negative effects on the activity of the prefrontal cortex (Dahl, 2006, Wong, Brower, Nigg & Zucker, 2010). Surprisingly, only one study has tested this mediational model empirically using sleep data gathered during childhood and real-world risk-taking outcomes during adolescence. Using a high risk sample drawn from a study of families in which the father was reported as an alcoholic, Wong, Brower, Nigg and Zucker (2010) sought to examine the extent to which childhood sleep problems predicted response inhibition problems in adolescence (assessed using a stopping task) and secondly, to test the extent to which response inhibition mediated the relationship between childhood sleep problems and substance use during adolescence. Controlling for age, parental alcoholism, and adolescent sleep measures, childhood sleep problems predicted binge-drinking and other alcohol related problems, as well as illicit drug use, and other drug related problems during adolescence. Using two-level mixed models to account for clustering

of data, Wong et al. found support for the proposed meditational model through response inhibition and also for a direct association between sleep and substance use. The authors conclude that this study provides evidence for the possibility that relationships between sleep problems and self-regulatory abilities persist overtime and have important long-term implications for risk-taking during adolescence. Certainly, this study fails to address questions about the uniqueness of these findings for substance use outcomes and the generalizability to non-high risk populations, however it is significant as the first to empirically test a meditational model linking sleep and risk-taking through a component of executive function. In an attempt to address these concerns, the proposed study will similarly test a meditational model through executive function, as well as impulse control, using a wider range of risky behaviors as outcomes within a non-high risk sample.

Meditational models are not the only way of explaining the association between sleep and risk-taking during adolescence. It is quite possible that a common factor might explain the association less directly, such that sleep and risk taking are related as a result of being influenced by a common factor that does not mediate the association between them. The following two sections will discuss the evidence that suggests either pubertal timing (a biological factor) or parental monitoring (a family factor) may be important third variables to consider.

*The role of pubertal timing.* The importance of puberty and pubertal timing to the experience of adolescence cannot be overstated. Of particular interest here are the effects of puberty on sleep and risk-taking; significant literatures exist in both areas to suggest a connection. Below I summarize the current understanding of the effects of pubertal status on



sleep and risk-taking and discuss the possibility that pubertal status is a third variable that explains the sleep-risk taking link.

It has been recognized that during puberty significant shifts in sleep onset and duration occur. A variety of studies across cultures and across times have reported that with advancing age children go to bed later and achieve shorter sleep durations (Goodlin-Jones, Sitnick, Tang, Lui & Anders, 2008, Mindell, Meltzer, Carskadon & Chervin, 2008, Lui, Lui, Owens & Kaplan, 2009). Self-reported and objectively assessed pubertal development is correlated with preferences for later sleep onset, a process called the circadian shift (Carskadon, Veira & Acebo, 1992, Carskadon & Acebo, 2005). Carskadon has hypothesized that changing sensitivity to light occurs during puberty, affecting melatonin secretion. However, data to support this idea are limited. Research in animal models suggests that hormones are playing a pivotal role in circadian changes. Studies in rats and monkeys, in which hormones (testosterone and estrogen) were administered or gonadectomies were done, showed subsequent effects on circadian properties of sleep (Hagenaur, 2009). Regardless of the underlying causes, the shift to later sleep onset during adolescence has negative consequences on total sleep duration when paired with demands placed on adolescents to get to school early in the morning (Carskadon, 2002, Fuligni and Hardway, 2006, Adam, Snell and Pendry, 2007). The result from the coming together of biological changes that lead to later sleep onset and external demands to rise early is striking. Forty-five percent of adolescents in the United States are considered sleep deprived (Carskadon, Mindell & Drake, 2006).

Just as research has shown that pubertal onset brings with it change in sleep patterns, the onset of puberty has also been shown to usher in a time of high levels of risk-taking in

many domains. Studies have found pubertal onset to be related to substance use, specifically, the initiation of tobacco use (Harrell et al., 1998, Negriiff, Dorn & Huang, 2010), alcohol use (Dick, et al., 2000), marijuana use (Stattin & Magnusson, 1990). Pubertal onset has also been shown to be related to sexual risk taking, particularly for females (Ellis, 2004, Halpern, Kaestle & Hallfors, 2007, Steinberg, 2008) and aggressive risk taking, particularly for males (Byrnes et al., 1999, Udry, 2000).

Only one study to date has considered the possible relations between sleep and risk-taking in the context of pubertal development. In this study, Holm and colleagues (2009) used fMRI technology to understand differences in reward processing (thought to be critical for risk-taking and decision-making) between pre/early pubertal and mid/late pubertal adolescents, all ages 11-12 years old, who typically received adequate versus inadequate sleep. In an initial stage of the study, sleep was objectively and subjectively assessed for 4 days using actigraphy and self-report. Naturally occurring differences in sleep quality and quantity were used to draw conclusions about the effects of sleep on reward processing and risk-taking behavior. Rather than focusing on the activity in the prefrontal cortex, as other neuroimaging studies of risk-taking have, researchers in this study were looking for differences, based on sleep and pubertal status, in the caudate. The caudate, part of the ventral striatum, is thought to be a center of reward processing that undergoes maturational changes during puberty that lead to lower reactivity in response to rewarding stimuli. Lower reactivity of the caudate during mid/late puberty has been hypothesized to explain risk-taking behaviors through compensations that adolescents make in seeking higher levels of stimulation or excitement. The authors of this study believed that pubertal development would be associated with poorer sleep quality and quantity and that inadequate sleep would

have similar dampening effects on caudate activation. Results revealed that more advanced pubertal development was associated with shorter sleep duration and in turn that sleep was associated with lower levels of reward processing during both anticipation and outcome phases of their lab-based risk taking task. This finding describes a mediational model such that changes in sleep patterns resulting from advancing pubertal development indirectly explains the link between pubertal development and risk-taking. The authors believe their findings suggest that sleep and puberty may have synergistic effects on reward processing which result in increases in adolescent risk-taking behaviors.

There is work to suggest that the relationships between sleep and pubertal maturation may be even more complex than it may first appear. Using evolutionary-developmental perspectives, Ellis and Boyce (2005) have suggested that environmental stressors influence timing and tempo of pubertal maturation. If inadequate sleep or poor sleep is included as a stressor, as has been suggested by McEwen (1998), then it is possible that sleep could serve as a signal that leads to the onset of puberty or increases the rate of pubertal development. If this was the case, the relationship between sleep and risk-taking could be mediated through pubertal development.

Important for the proposed study, the fact that the onset of puberty is the beginning of significant changes in sleep patterns and it also is marked by an increase in risk-taking behaviors must be taken into consideration for hypothesis development and analysis strategies. Pubertal status may be acting as a third variable or might be an important moderator of the relationship between sleep and risk-taking. It is possible that controlling for pubertal status may reduce the relationship between sleep and risk-taking behaviors to a non-significant level but this has never been empirically tested in prior research. On the other

hand, pubertal status may act as a moderator such that for those individuals who experience poor sleep and have already reached mid/late puberty, the association with risk-taking might be stronger. Further, as suggested by the existing literature on the effect of pubertal status on risk taking, gender differences may exist, particularly when looking at risk taking in separate domains (i.e. sexual risk taking versus aggressive risk taking). Finally, based on the suggestion that poor or inadequate sleep is perceived as a stressor, it is possible that the relationship between sleep and risk-taking could be mediated by pubertal development. Testing these possibilities will be a basis of the proposed study and will be exploratory given that very little prior research has looked at the relationship between sleep and risk taking with consideration of pubertal status.

*The role of parental monitoring.* In addition to the biological factor of pubertal development, a family factor, parental monitoring may influence both sleep and risk-taking. Parental monitoring, the involvement and knowledge that parents have about their child's plans and activities, is an important component of Jessor's (Jessor, 1992, Jessor & Jessor, 1977) influential Problem Behavior Theory that attributes risk behaviors to poor social control and inadequate shaping of conventional and prosocial behaviors. There is a substantial, established literature that shows the importance of parental monitoring in protecting youth from exhibiting a wide spectrum of risk-taking behaviors (Dishion, et al, 1991, Dishion & McMahon, 1998). Greater parental monitoring has been shown to be associated with later initiation or lower levels of tobacco use (Childcoat & Anthony, 1996) and alcohol and drug use (Dishion et al., 1995). A recent review of the literature on the parental monitoring and marijuana use (Lac & Crano, 2009) concluded with a rather convincing file-drawer analysis that showed it would take 7,358 studies of null findings to

render the association non-significant. In the area of sexual risk taking, several studies have similarly found parental monitoring to be protective in terms of initiation of sexual activity, number of partners and risky sexual behaviors (Luster & Small, 1994, Rodgers, 1999, Huebner & Howell, 2003). Even while disagreement exists about the mechanisms that underlie the protective effects of parental monitoring or how it should be measured (Stattin & Kerr, 2000, Fletcher, Steinberg & Williams-Wheeler, 2004), it seems indisputable that this construct remains relevant for the understanding of adolescent risk-taking behaviors.

The role of parental monitoring for sleep during adolescence is less clear. Theoretically, a lessening of parental influence over their children's actions is one consequence of the transition from childhood into adolescence and parental influence over sleep behaviors should show this pattern. Indeed, this is the case for parental influence over bedtimes. From age 10 to 13 there are significant changes in the percentage of children who report that their parents set bedtimes and are responsible for wake up times (Carskadon, 1979, Acebo & Carskadon, 1997). However, while parental influence over bedtime decreases greatly around puberty, parental involvement in waking, at least on weekdays, continues at high levels through high school (Carskadon, 2002). Research also shows that the turning over of decisional control concerning sleep among early adolescents (9-13 years old) is associated with fewer hours of sleep and daytime sleepiness (Teufal, Brown & Birch, 2007), suggesting that, at least in this age group, self-management of sleep is detrimental to sleep hygiene. Likewise, in a study by Adam, Snell and Pendry (2007), parental control was found to be predictive of sleep duration on weekdays for adolescents. So, although parental influence is waning across the adolescent years, parents appear to continue to shape sleep behaviors. More broadly, researchers have found that positive home atmosphere and familial

support were predictive of sleep habits for a sample of 15 year old adolescents (Tynjala Kannas, Levalathi, & Valimaa, 1999). The possibility exists that there may be more subtle, less direct ways in which parents influence sleep habits in older children, for example through setting curfew, through restricting television/computer use, or providing structure to daily activities. Although much more research needs to be done looking at the influence of parents, there is enough research to suggest that the construct of parental monitoring might be usefully applied to understanding adolescent sleep habits.

Given what is known about the importance of parental monitoring for risk-taking behaviors and the suggestion that parents continue to exert influence on sleep patterns into adolescence, one might reasonably conclude that sleep and risk-taking behaviors might be related through their common association with parental monitoring. If parents do a good job of monitoring their children's whereabouts they are likely to also be involved in setting limits that would shape positive sleep habits. Conversely, it may be that parents are unable to provide structure and supports for positive daytime behavior and emotional regulation are also less likely to provide structure and support for development of positive sleep habits at night. Therefore, parental monitoring may serve as a protective factor that would lessen the strength of the relationship between sleep and risk-taking behaviors. Again, these are important considerations that have yet to be empirically tested and are another major focus of the proposed research study.

### *Overview of study*

The purpose of the proposed study is to first, replicate results showing an association between sleep and risk-taking and then to investigate possible mediators and moderators of

the association between these two behaviors from late childhood into adolescence. This research adds significantly to the existing literature by making use of longitudinal data and multiple measures of both sleep and risk taking, while taking into account co-existing behavior problems, family factors and possible gender differences. Because important distinctions between different sleep variables (sleep problems, sleep duration, sleep schedule variability) have not been consistently made in previous research, one overarching aim of this study is to analyze the relationship of each of these variables to risk-taking behaviors separately. Because prior research has relied on single time point analyses, little evidence exists about the unique importance of changes in sleep during adolescence. In order to address this limitation, this study also includes change from sixth grade to 15 years in each sleep variable.

Similarly, risk taking has been operationally defined in a variety of ways in previous research, sometimes measured by self-report of real-world risky behaviors and other times measured through lab-based tasks that assess risk-taking propensities. A second aim of this proposal is to assess the association between sleep and “real-world” risk taking as well as “lab-based” risk taking. Further, it may be that certain types of risk taking are more or less related to sleep such that the findings of the association may be driven by one type of risk taking (substance use, for example). It is then important to consider sub-types of risky behaviors. Therefore, in order to address the concerns related to the various measures of sleep and risk-taking, the hypotheses for this study are written in a way that allow for testing these relationships across available sleep variables and risk-taking measures.

The NICHD Study of Early Child Care and Youth Development (SECCYD) data set is uniquely suited to achieve the goals of the proposed study in ways that would be very

difficult and expensive to achieve through other means. First, the dataset includes detailed measurement of sleep at sixth grade and 15 years, providing data that can be used to look at changes in sleep duration, sleep problems and sleep schedule variability across a critical developmental period. Secondly, the dual measurement of risk-taking behavior, both self-report of real world risk-taking and lab based tasks to measure risk-taking propensity, allows the analysis to examine the sleep-risk association broadly. The assessment of pubertal development by clinicians is the rarely achieved, gold standard and avoids the problems inherent with parent or self-reported pubertal development measures. Another important benefit of this dataset is the availability of measures of depressive symptoms at baseline (sixth grade). This allows the examination of the sleep-risk-taking link to be conducted while controlling for depressive symptoms, as has been done by other researchers (Carterett & Gaultney, 2009). Finally, the use of the SECCYD dataset has the advantage of having a large number of important family, contextual and health variables that can be examined in follow-up studies.

The first set of hypotheses in the proposed study (H1, a-d) pertain to change over time, from sixth grade to 15 years, for the sleep and risk-taking variables. Given the age span covered and the likelihood that the transition from pre-pubertal to post-pubertal status are captured, it is hypothesized that all sleep variables will change over time in a way that reflects worsening sleep. Specifically, it is expected that with an increase in age sleep problems will increase, sleep duration will shorten, mid-sleep will be later and sleep schedules will become less regular. The effect of parental monitoring and pubertal status on the change in sleep variables are explored. Similarly, change over time in risk-taking behaviors are examined, with the hypothesis that increases will be seen across all domains of



risk taking. The effect of parental monitoring and pubertal status on the change in risk-taking behaviors are explored.

The second set of hypotheses (H2, a-h) examine the association between sleep and risk-taking variables within and across time. The role of parental monitoring and pubertal status as moderators or third variables are explored. Further, analyses will examine the extent to which including depressive behavior problems as a co-variate will affect the relationships between study variables. Given that multiple measures of sleep and of risk-taking are available, all combinations of sleep variables, including change in sleep from sixth grade to 15 years, and risk-taking behaviors, are systematically tested for each of the hypotheses.

Finally, the third set of hypotheses (H3, a-b) involves the testing of mediational models that may explain the link between sleep and risk-taking within and across time. Specifically, executive functioning and impulse control are examined as mediators of the association between sixth grade sleep variables and 15 year risk-taking behaviors. Again, all combinations of sleep variables, including change in sleep from sixth grade to 15 years, and risk-taking behaviors are systematically tested for each of the hypotheses.

### *Hypotheses*

Hypothesis 1a: Sleep problems and sleep variability will increase from sixth grade to 15 years. Sleep duration will decrease across this period.

Hypothesis 1b: The increase in sleep problems and variability in sleep patterns and the decrease in sleep duration from sixth grade to 15 year will be greater for youth who experience lower levels of parental monitoring and earlier pubertal timing.

Hypothesis 1c: Risk-taking will increase from sixth grade to 15 years.

Hypothesis 1d: The increase in risk-taking will be greater for youth who experience lower levels of parental monitoring and earlier pubertal development.

Hypothesis 2a: Sleep and risk-taking will be associated at both sixth grade and 15 years.

Hypothesis 2b: Sleep problems, sleep duration and sleep variability during sixth grade and change in sleep variables from sixth grade to 15 year will predict risk-taking behaviors at 15 years.

Hypothesis 2c: The relationship between sixth grade sleep and change in sleep variables from sixth grade and 15 year and 15 year risk-taking will be moderated by pubertal development.

Hypothesis 2d: Changes in sleep from sixth grade to 15 year will mediate the association between pubertal development and risk-taking.

Hypothesis 2e: The relationship between sixth grade sleep and change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be moderated by parental monitoring.

Hypothesis 2f: Changes in sleep from sixth grade to 15 year mediate the association between parental monitoring and risk-taking.

Hypothesis 3a: The relationship between sixth grade sleep and change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be mediated by executive functioning.

Hypothesis 3b: The relationship between sixth grade sleep and change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be mediated by impulse control.

## METHODS

### *Data source and sample*

The sample was drawn from the NICHD Study of Early Child Care and Youth Development (NICHD SECCYD), a comprehensive longitudinal study aimed at understanding the relationship between childcare, family and school experiences, child characteristics, and developmental outcomes. Families in the study were recruited during the first 11 months of 1991 from 24 hospitals in the vicinity of 10 data collection sites across the United States. Further details regarding the recruitment and selection of child participants in the NICHD SECCYD are described in several documents that are publicly available, to which interested readers are referred (<http://secc.rti.org>). A total of 1,364 families with healthy newborns were ultimately enrolled in the study, with approximately equal numbers of families at each site. The study sample was demographically similar to the population of families with young infants in the communities from which it was recruited. As has been previously reported in studies using this data set, most of the attrition occurred within the first few years of data collection and by the sixth grade time point, the sample is stable.

The analysis sample for the current study consisted of 1077 children (at the sixth grade data collection) with approximately equal numbers of boys and girls. The sample was primarily Caucasian but also included a variety of other ethnic minorities (11% African American, 6% Hispanic and 5% other). About 30% of the mothers in the sample have a high school education or less. Table 1 provides summary baseline information on the sample from

the sixth grade time point, including means and standard deviations for included covariates and tested moderators (pubertal development and parental monitoring).

### *Measures*

All study variables in the present study are summarized in Table 2. This table includes details about which time point the data was collected and specifies the reporter.

*Sleep variables- duration, problems, and variability.* At sixth grade and 15 year time point, youth were asked to complete a questionnaire designed to measure their sleep habits and possible difficulties with sleep. The “My Sleep” questionnaire included questions in which youth rated the frequency of sleep problems on a 5 point scale (never, hardly ever, sometimes, most of the time, always) and indicated the time of day they typically go to sleep and wake up on weeknights and weekends. From these responses the three variables of interest were created (total sleep duration, total sleep problems, and sleep schedule variability). The total sleep duration was calculated in three steps; first the sleep duration for weeknights and weekend nights were calculated separately, next the sleep durations were weighted by multiplying the weeknight total duration by .71 and weekend night total by .29 (for proportion of the week each represented), and finally these totals were added to produce the total duration average for the week. Total sleep problems was a sum of the responses on 5 items that asked youth to rate the frequency they experience a variety of sleep problems (i.e. daytime sleepiness, trouble falling asleep, and trouble maintaining sleep). Answers ranged from 5-35. Sleep schedule variability was created by computing the difference between bed time reported for weeknights and weekends.

*Risk-taking.* Risk-taking was assessed using the Youth Self-report, Child Behavior Checklist, and “Thing I Do” Questionnaire. These questionnaires include scales of general delinquency, sexual risk-taking, and substance use risk-taking. “Things I Do” is a 61 item self-report measures that assesses the frequency with which adolescents engage in a wide range of risky behaviors. The general delinquency score is a sum of responses to the 61 items and has high internal reliability (Chronbach’s  $\alpha=.89$ ). Sexual risk-taking behavior was assessed using 3 items from the “Things I Do” questionnaire that asked youth to report how many times in the last year they have had oral sex, had sexual intercourse, or have been told they have an STD (3 items, Chronbach’s  $\alpha=0.72$ ). Substance use risk-taking was assessed using 4 items from the “Things I Do” questionnaire that asked youth to report how many times in the last year they smoked cigarettes, drunk a bottle of beer or other alcohol, and smoked marijuana.

*Lab-based risk-taking.* The Balloon Analogue Risk Task (BART), a computerized, laboratory based measure which assesses risk-taking propensity, was administered at the 15 year home visit to study youth. BART was developed to model risk taking in the natural environment in which risk taking up to a certain point leads to positive consequences, with further excessive risk taking leading to greater negative consequences that outweigh the positive (Lejuez, Read, Kahler, Richards, Ramsey, Stuart, Strong & Brown, 2002). During the task, the computer screen showed a small, simulated balloon accompanied by a balloon pump, a reset button, and a display of points earned. With each pump of the balloon, youth earned 1 point but all accumulated points would be erased if the balloon popped. Each balloon was programmed to have a different possibility of exploding. Youth were instructed that all balloons would explode at some point and could occur as early as the first pump. An

index of riskiness was computed from the average number of pumps only on balloons that were not exploded. Higher values on this index indicate a higher propensity for risk-taking. The BART has been shown to identify a variety of real-world risk-taking behaviors in adults and adolescents (Lejuez, Aklin, Daughters, Zvolensky, Kahler & Gwadz, 2007).

*Executive function.* Executive function was assessed through two tasks, the Tower of London (TOL) and Stroop task. The TOL is believed to measure spatial planning, and behavioral and response inhibition (Berg & Byrd, 2002; Asato, Sweeney & Luna, 2006). The TOL task was administered as part of the 15 year lab visit. The activity is a computerized, puzzle-like game in which youth were asked to move three balls on the screen from their starting positions so that they match the target positions that appear on the screen. Each problem can be solved, although the number of minimum moves ranges from one to seven. Summary scores across 23 trials were computed to reflect success (percent of trials solved and percent of trials with perfect solutions), efficiency (the average number of moves made beyond the minimum number of moves necessary for an optimal solution), and planning time (time from the problem presentation to the completion of the first move).

The Stroop task was also administered to youth during the 15 year lab visit. The Stroop task is one of the most frequently used measures of inhibitory control (MacLeod, 1991) and is also associated with cognitive flexibility and resistance to interference from outside stimuli. In this activity, youth were asked to press a button that matches the color of a word, while ignoring what the word says. Youth participated in 96 trials, 48 included incongruency between word and color (e.g. the word red written in blue) and 48 trials were neutral (e.g. the word math written in blue). A measure of interference was computed by subtracting the average reaction time for neutral trials from the average reaction time for

incongruent trials. The final interference score was then adjusted for baseline differences in reaction time by dividing the difference score by the average reaction time for neutral trials, so that lower scores indicate less interference and better performance.

*Parental monitoring.* Parental monitoring was assessed at sixth grade and 15 year using the “Keeping Tabs” questionnaire, an 11-item self-report measure that asked youth questions regarding parental supervision and monitoring. Responses are on a range from 1-4 (“doesn’t know at all” to “knows everything”) and indicate the extent to which the parent is thought to know about different aspects of the child’s day to day experience. Sample questions include: “how much does a parent know about... who you spend time with?”, “...how you spend your money?”, “...where you go after school?”. The parental monitoring score is the total of the responses to the 11 items (Chronbach’s  $\alpha=.77$ ).

*Impulse control.* Impulse control was assessed at 15 year using a 8-item impulse control subscale from the Weinberger Adjustment Inventory (WAI; Weinberger & Schwartz, 1990). The measure asks participants to rank the frequency (range 1-5) in which their behavior matches a series of statements. Sample statements include: “I’m the kind of person who will try anything once, even if it is not that safe” and “I do things without giving them enough thought”. The items have a moderate internal reliability (Chronbach’s  $\alpha=.82$ ).

*Pubertal development.* Study youth participated in an annual Health and Physical Development Assessment (HPDA) starting at age 9<sup>1/2</sup> which was completed by a doctor or nurse practitioner. The purpose of the physical examination was to provide direct observational data on the adolescent’s physical growth and development. Tanner staging, an assessment of development of secondary sexual characteristics, for girls was based on instructions from the American Academy of Pediatrics manual. Breast and pubic hair



development were scored on a 5 point scale, ranging from no development to mature adult development. Tanner staging for boys was based on Tanner's original criteria and included assessment of genital and pubic hair development on a similar 5 point scale.

*Depressive behavior problems.* At sixth grade and 15 years, participants completed the Children's Depression Inventory (CDI) (Kovacs, 1992) to assess level of depressive symptoms over the previous two weeks. The CDI consists of ten sets of three statements that the participants have to choose from that best fit how they have felt over the past two weeks. Sample sets include, "0=I am sad once in a while; 1=I am sad many times; 2=I am sad all the time". The items measure dysphoric mood, lack of pleasure, and low self-esteem. Items are summed to create the Child Depression Score, (10 items, Chronbach's  $\alpha=0.76$ ).

*Family socioeconomic status.* The income-to-needs ratio was derived from two separate assessment measures administered on a yearly basis to the families, the "Household Grid" and the "Family Education and Income" questionnaire. The "Household Grid" was used to determine the number of adults and children living in the household and the "Family Education and Income" questionnaire provided total family income. These variables were combined to compute the family income-to-needs ratio. Data from the sixth grade data collection were used as a control variable in the study.

## RESULTS

### *Preliminary analyses*

Prior to testing the study hypotheses, preliminary analyses were conducted. These preliminary analyses included checking data for outliers, assessing normality of distributions, and conducting scale diagnostics. For study variables that used a limited range of response options (pubertal development, parental monitoring, impulse control, sleep problems), no outliers were found. For these same variables, there was no evidence of unacceptable levels of skew (above 7) or kurtosis (above 2) (Cohen, Cohen, Aiken & West, 2003). A limited number of outliers were found for sleep duration ( $n=13$ ) and variability ( $n=16$ ) and were removed from subsequent analyses by changing them to missing values. Real world risk-taking variables were found to be left-skewed and were then square root transformed to normalize the distribution, as has been done by previous researchers using this dataset. Means and bivariate correlations for all study variables were calculated. Bivariate correlations, means and standard deviations for study variables are presented in Table 3.

Given that this study sought to understand the unique influence of sleep duration, sleep variability and sleep problems on later risk-taking behaviors, it was important to describe the relationship between these sleep variables. Intercorrelations for within each time point can be seen in Table 3. Although the three sleep variables were significantly correlated at both the sixth grade and 15 year time points, the correlations within each time point were

modest (-.27 to .23). These findings confirmed the assertion that each sleep variable should be considered separately in the subsequent analyses as proposed.

*Co-variables.* Collection site and family income-to-needs ratio were included as control variables in regression analyses, as has been done in previous studies looking at risk using national datasets (Johnson & Breslau, 2001). When conducting analyses that did not include specific hypotheses about gender, it was also included as a control variable. In some models, depressive behavior problems were also included as a co-variate.

#### *Change over time in sleep and risk-taking*

Hypothesis 1a: Sleep problems and sleep variability will increase from sixth grade to 15 years. Sleep duration will decrease over the same period.

As has been reported by previous research, it is expected that (1) sleep duration will decrease, (2) sleep problems will increase and (3) sleep patterns will be more variable at the 15 year time point compared to the sixth grade time point. A paired t-test was conducted to determine if the difference between sixth grade and 15 years was significant.

Results showed that there was a significant change in all sleep variables across the two time points. Sleep duration decreased from sixth grade ( $M=9.48$ ,  $SD=.79$ ) to 15 years ( $M=8.28$ ,  $SD=1.00$ ),  $t(868)=33.31$ ,  $p<.000$ . Sleep variability increased from sixth grade ( $M=1.39$ ,  $SD=.1.07$ ) to 15 years ( $M=1.69$ ,  $SD=1.26$ ),  $t(826)=-6.10$ ,  $p<.000$ . Likewise, sleep problems increased from sixth grade ( $M=19.53$ ,  $SD=.5.09$ ) to 15 years ( $M=24.04$ ,  $SD=5.55$ ),  $t(946)=-23.23$ ,  $p<.000$ . Data from the paired sample t-tests, along with confidence intervals and effect sizes, are presented in Table 4.

Hypothesis 1b: The increase in sleep problems and variability in sleep patterns and the decrease in sleep duration from sixth grade to 15 year will be greater for youth who experience lower levels of parental monitoring and earlier pubertal timing.

The influences of parental monitoring and pubertal development on the *change* in sleep variables from sixth grade to 15 years were examined by using linear regression. To accomplish this, first a change variable was created for each sleep outcome (sleep duration, sleep problems, and sleep variability). In the regression, depressive symptoms and demographic covariates were entered as controls.

The results showed that pubertal development at sixth grade significantly predicted change in sleep problems from sixth grade to 15 years,  $\beta=.67$ ,  $t(826)=3.34$ ,  $p<.001$ , such that participants with more advanced pubertal development at sixth grade were more likely to experience greater increases in sleep problems across the transition to adolescence. Pubertal development explained a significant proportion of variance in change in sleep problems,  $R^2=.11$ ,  $F(1, 827)=11.05$ ,  $p<.001$ . Pubertal development did not have these effects on sleep duration or sleep variability.

The influence of parental monitoring on the change in sleep variables from sixth grade to 15 years was also examined. Contrary to the hypothesis, greater parental monitoring at sixth grade was predictive of greater change in sleep problems,  $\beta=1.86$ ,  $t(898)=3.37$ ,  $p<.001$ , explaining a significant proportion of variance in changes in sleep problems,  $R^2=.13$ ,  $F(1, 898)=5.56$ ,  $p<.001$ . Parental monitoring did not predict changes in sleep duration or sleep variability.

Hypothesis 1c: Risk-taking will increase from sixth grade to 15 years.

It was expected that self-reported risk-taking would increase from sixth grade to 15 years. Paired t-test was used to determine if the difference from sixth grade to 15 years was significant. General risk-taking and substance use were measured at sixth grade and 15 years and showed increases across this time span. General risk-taking at 15 years ( $M=6.14$ ,  $SD=5.67$ ) was significantly greater than general risk-taking at sixth grade ( $M=2.26$ ,  $SD=1.91$ ),  $t(932) = 23.95$ ,  $p<.000$ . Likewise, substance use increased from sixth grade ( $M=.04$ ,  $SD=.25$ ) to 15 years ( $M=.64$ ,  $SD= 1.33$ ),  $t(932) = 13.74$ ,  $p<.000$ . Data from the paired sample t-tests, along with confidence intervals and effect sizes, are presented in Table 4.

Hypothesis 1d: The increase in risk-taking will be greater for youth who experience low levels of parental monitoring and earlier pubertal development.

Similar to the analyses for H1b, a change variable was created for each risk-taking outcome (self-reported general risk-taking and substance use). Next, this hypothesis was tested by using linear regression predicting the change in risk-taking variables (from sixth grade to 15 year) considering sixth grade parental monitoring and sixth grade pubertal development separately as predictors.

Results showed that the influence of pubertal development on changes in risk-taking was insignificant. Parental monitoring, however, at sixth grade was found to significantly predict changes in general risk-taking  $\beta=-1.63$ ,  $t(780)=-3.58$ ,  $p<.000$  and substance use  $\beta=-.19$ ,  $t(783)=1.97$ ,  $p<.001$ . Parental monitoring explained a significant proportion of variance in change in general risk-taking,  $R^2=.04$ ,  $F(1, 780)= 6.10$ ,  $p<.000$ , and substance use,  $R^2=.03$ ,

$F(1, 783) = 6.13, p < .000$ . Greater parental monitoring at sixth grade was predictive of less increase in general risk-taking and substance use across sixth grade to 15 years.

*Association between sleep and risk-taking over time*

Hypothesis 2a: Sleep and risk-taking will be associated at both sixth grade and 15 years.

It was hypothesized that sleep variables and risk-taking variables would be associated within each time point. Differences in the association between sixth grade and 15 year were evaluated using the comparison of correlation coefficients. This was done using the Fisher  $r$ -to- $z$  transformation to assess significance of the difference between two correlation coefficients (i.e. the correlation between sleep and risk taking variables at sixth grade versus 15 year).

Results from these analyses showed that all sleep variables and all risk-taking variables (except for the BART lab-based task) were associated at sixth grade and at 15 years (Table 3). Specifically, sleep problems and sleep variability were positively correlated with general risk-taking and substance use at sixth grade and 15 years. Sleep duration, on the other hand, was negatively correlated with general risk-taking and substance use at sixth grade and at 15 years. Likewise, sleep problems and variability were positively associated with sexual risk-taking (not measured at sixth grade) at 15 years. All correlations were significant at  $p < .01$ . Fisher's  $R$  to  $Z$  transformation indicated no significant differences between the level of association at across time points.

Hypothesis 2b: Sleep problems, sleep duration, and sleep variability during sixth grade and change in these sleep variables from sixth grade to 15 years will predict risk-taking behaviors at 15 years.

To test this hypothesis, a series of linear regressions were run testing the extent to which the sleep variables at sixth grade predicted self-reported and lab based risk-taking behaviors at 15 years. By conducting a similar regression using 15 year sleep variables as the predictor while controlling for sixth grade sleep variables, the extent to which the *change* in sleep from sixth grade to 15 year predicted 15 year risk-taking was also tested.

Results showed that while controlling for gender, income to needs ratio, and sixth grade depression scores, 15 year general risk-taking was predicted by all sleep variables: sleep duration  $\beta = -.05$ ,  $t(891) = -3.22$ ,  $p < .001$ , sleep variability  $\beta = .03$ ,  $t(781) = 2.47$ ,  $p < .01$ , and sleep problems  $\beta = .05$ ,  $t(891) = 1.94$ ,  $p < .05$ . Greater sleep durations predicted lower levels of risk-taking, while sleep variability and sleep problems predicted greater risk-taking. Sixth grade sleep variables did not predict substance use, sexual risk-taking nor performance on the BART lab-based risk-taking task. The data predicting risk-taking from sixth grade sleep variables are summarized in Table 5.

In addition, results showed that change in sleep variables significantly predicted general risk-taking: change in sleep duration  $\beta = -.13$ ,  $t(734) = -2.62$ ,  $p < .05$ , change in sleep variability  $\beta = .02$ ,  $t(735) = 3.67$ ,  $p < .05$ , and change in sleep problems  $\beta = .03$ ,  $t(734) = 2.91$ ,  $p < .05$ . Data from the regression analysis of the effect of sleep and changes in sleep on risk-taking are presented in Table 5. Substance use, sexual risk-taking and lab-based risk-taking were not predicted by change in any sleep variables. The data predicting risk-taking from the change in sleep variables are summarized in Table 5.

Hypothesis 2c: The relationship between sixth grade sleep variables or change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be moderated by pubertal development.

The moderating effects of pubertal development on the relationship between sixth grade sleep and 15 year risk-taking were tested. In order to test the hypotheses related to moderating effects on the relationship between sleep and risk-taking variables (Hypotheses 2c and 2e), the product of the focal predictor and moderator (the interaction term) was entered as an additional predictor into the regression model. The effect of the moderator on the relationship between the focal predictor and the outcome was considered significant if the change in variance explained ( $R^2$ ) when the interaction term is added to the model is significant (Cohen, Cohen, West & Aiken, 2003). To understand the effect of the moderator, the *pick-a-point* approach (Aiken & West, 1991) for probing interactions was used wherein representative values (high, moderate, low) of the moderator values are used to estimate the effect of the focal predictor at those values. An application created by Hayes and Matthes (2009) for use with SPSS statistical analysis software that employs this procedure was used.

No significant two-way interactions for pubertal development as a moderator were found. To further explore possible gender effects, I also tested for interactions for pubertal development with male and female samples separately. Again, no significant two-way interactions with pubertal development were found.

Parental monitoring moderated the relationship between changes in sleep problems and general risk-taking, but not the relationship between the other sleep variables or risk-taking variables. As can be seen in Figure 1, the interaction term between parental



monitoring and change in sleep problems explained a significant increase in variance in general risk-taking,  $\Delta R^2 = .05$ ,  $F(1,734) = 23.06$ ,  $p < .001$ . The relationships between other sixth grade sleep variables and substance use, sexual risk-taking or the lab-based BART task were not significantly moderated by the interaction of change in sleep variables and parental monitoring.

Hypothesis 2d: Changes in sleep from sixth grade to 15 year will mediate the association between pubertal development and risk-taking.

Hypothesis 2d represents an alternative explanation for the relationship between sleep, risk-taking and pubertal development. Instead of considering pubertal development as the moderator, changes in sleep were tested as possible mediators of the relationship between pubertal development and risk-taking. Mediation was tested using the Sobel test (Sobel, 1982), which allows one to directly test the significance of indirect effects in large samples and is considered a more rigorous approach than the commonly applied Baron and Kenny (1986) procedure. Given the sample size, it is appropriate to employ the Sobel test of mediation rather than more complicated bootstrapping techniques that are useful for smaller samples.

The Sobel test was carried out using an application created by Preacher and Hayes (2004) for SPSS data analysis software. The application provides estimation of indirect effects, defined as the product of the  $X \rightarrow M$  path ( $a$ ) and the  $M \rightarrow Y$  path ( $b$ ), or  $ab$ . In the case of simple mediation, as is tested in this study,  $ab = (c - c')$ , where  $c$  is the main effect of  $X$  on  $Y$ , not controlling for  $M$ , and  $c'$  equals  $X \rightarrow Y$  path after adding  $M$  as a control to the model (see figure below). In order to determine significance, the indirect effect value is

divided by the standard error of the indirect effect and compared to the critical value for the desired alpha level.

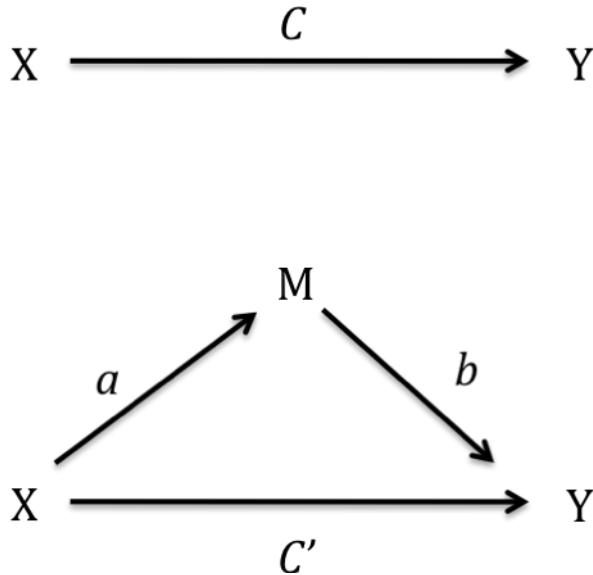


Figure 1. Indirect effect model

In testing Hypothesis 2d, none of the  $c'$  paths were significant, which suggested that changes in sleep did not mediate the relationship between pubertal development and risk-taking.

Hypothesis 2e: The relationship between sixth grade sleep variables or changes in sleep variables from sixth grade to 15 year and 15 year risk-taking will be moderated by parental monitoring.

Similar to the analyses for Hypothesis 2c, the moderational effects of parental monitoring on the association between sleep variables or changes in sleep variables from sixth grade to 15 years and risk-taking behavior were tested using a liner regression predicting risk-taking from sleep variables. In a following step, parental monitoring was

entered as an interaction term. It was expected that higher levels of parental monitoring would be associated with weaker relations between sleep and risk-taking.

Results showed that parental monitoring moderated the relationship between changes in sleep problems and general risk-taking, but not the relationship between the other sleep variables or risk-taking outcomes. As can be seen in Figure 1, the interaction term between parental monitoring and change in sleep problems explained a significant increase in variance in general risk-taking,  $\Delta R^2 = .05$ ,  $F(1,734) = 23.06$ ,  $p < .001$ . Higher levels of parental monitoring weakened the relationship between changes in sleep problems and general risk-taking. The relationship between other sixth grade sleep variables and substance use, sexual risk-taking or the lab-based BART task were not significantly moderated by the interaction of change in sleep variables and parental monitoring.

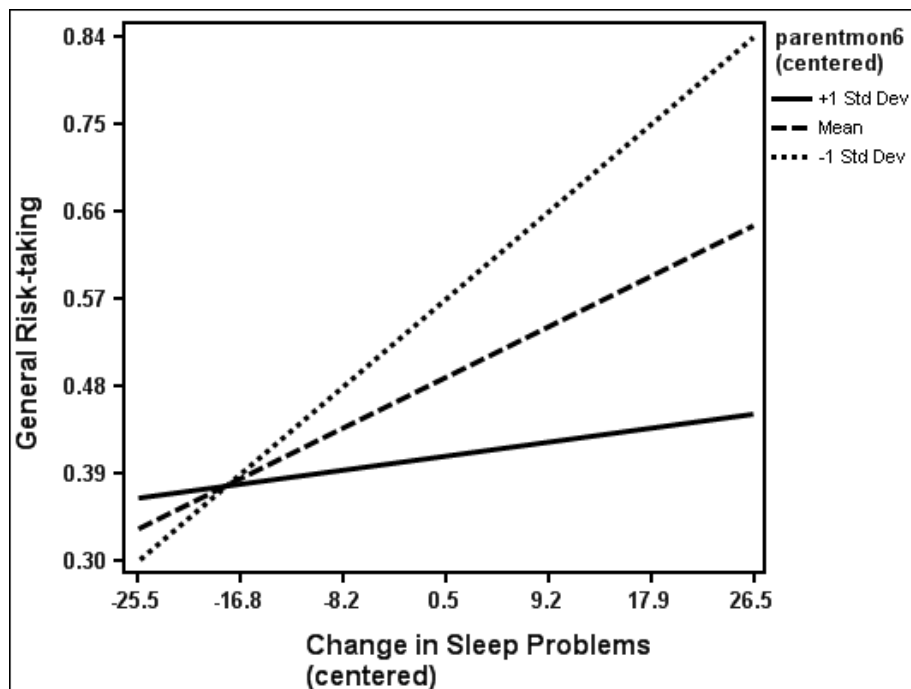


Figure 2. Parental monitoring moderates the effect of change in sleep problems on general risk-taking

Hypothesis 2f: Changes in sleep from sixth grade to 15 year will mediate the association between parental monitoring and risk-taking.

Just as Hypothesis 2d represented an alternative explanation for the relationship between sleep, risk-taking and pubertal development, Hypothesis 2f represents an alternative explanation for the relationship between sleep, risk-taking and parental monitoring. A model in which higher levels of parental monitoring would be predictive of risk-taking behaviors through the effects on sleep variables was tested by running a regression with parental monitoring predicting changes in sleep and a separate regression in which parental monitoring and changes in sleep predict risk-taking behaviors. Then, as described above, the Sobel test was employed to calculate whether the indirect effect of pubertal development on risk-taking via changes in sleep is significantly different from zero. Results from this indirect path analysis showed that changes in sleep did not mediate the relationship between parental monitoring and risk-taking.

#### *Mediation of the sleep-risk association*

Hypothesis 3a: The relationship between sixth grade sleep variables or change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be mediated by executive functioning, particularly for lab-based risk tasks.

This hypothesis tested a model in which sleep was predictive of risk-taking behaviors through the effects on executive functioning. This was tested by running separate regressions with sleep variables or change in sleep variables predicting executive functioning and a separate regression in which sleep and executive functioning predict risk-taking behaviors.

Then the Sobel test is employed to calculate whether the indirect effect of sleep on risk-taking via changes in executive functioning is significantly different from zero. Just as in the analyses for Hypotheses 2d and 2f, the Sobel test was carried out using an application created by Preacher and Hayes (2004) for SPSS data analysis software.

In testing the model that executive functioning mediated the relationship between sleep and risk-taking variables, no significant relationships between executive functioning as measured by the Tower of London or the Stroop task and sleep variables were found. Despite significant relations between executive functioning and risk-taking variables, the lack of any significant *a* paths suggested that executive functioning does not act as a mediator between sleep and risk-taking. Further analyses were conducted using sub-scores for these executive functioning tasks which are thought to measure working memory, planning and inhibition separately. Again, no significant associations between executive functioning sub-scores and sleep variables, *a* paths, were found.

Hypothesis 3b: The relationship between sixth grade sleep variables or change in sleep variables from sixth grade to 15 year and 15 year risk-taking will be mediated by impulse control, particularly for self-reported risk-taking.

This hypothesis tested a model in which advanced sleep would be predictive of risk-taking behaviors through the effects on impulse control using the same technique as above. A regression was run with sleep predicting changes in impulse control and a separate regression in which sleep and impulse control predict risk-taking behaviors. Then the Sobel test will be employed to calculate whether the indirect effect of sleep on risk-taking via impulse control is significantly different from zero.

In all cases where the direct effect of the sleep variable on risk-taking was significant, impulse control mediated the association. Table 6 provides standardized regression coefficients and standard errors for  $a$ ,  $b$ , and  $c'$  paths and the results of the Sobel tests. The indirect path is considered significant when the standardized regression coefficient between sixth grade sleep duration and general risk-taking decreased substantially when controlling for the indirect path through impulse control. Other conditions for mediation were also met; sleep variables significantly predicted risk-taking and impulse control and impulse control was a significant predictor of risk-taking.

Specifically, greater sleep durations were predictive of higher levels of impulse control, which was in turn related to decreased risk-taking. Greater sleep variability, on the other hand, was predictive of less impulse control, which was then related to greater risk-taking. Similarly, greater sleep problems were predictive of lower levels of impulse control, which was then related to greater risk-taking.

## DISCUSSION

The period between late-childhood and adolescence is significant for the swift biological, behavioral and social changes that occur across this transition. In examining the association between sleep and risk-taking behaviors, this dissertation employed a developmental systems framework that assumes changes at each level potentially have influences on each other and are sensitive to context and timing effects (Gottlieb, 1991, Thelen & Smith, 1998). The current study's focus on sleep and risk-taking addressed questions raised by previous studies showing an association between these behaviors, but this research also provided an excellent test case to explore multiple levels of influence on a relationship between emerging and dynamic behaviors over a critical developmental period. The results from this study reveal a more nuanced description of the association between sleep and risk-taking than has been reported by addressing questions related to: (1) change over time in sleep and risk-taking, (2) the association between sleep and risk-taking over time, and (3) possible mediators of the association. Further, the results of this study suggest many avenues for future research.

As predicted, the period from late childhood into adolescence was marked by poorer sleep as measured by shorter sleep durations, greater variability in sleep patterns and more self-reported sleep problems. Over the same period, participants in this study reported increases in risk-taking. These findings are consistent with previous research and lay the basis for one goal of this study, which was to explore the extent to which the co-occurrence

of poorer sleep and increased risk-taking from late childhood into adolescence are causally related or simply influenced by common factors.

In examining the effect of pubertal development on *changes* in sleep, it appears that child experiencing earlier pubertal development in late-childhood may experience a greater increase in sleep problems into adolescence as compared to children experiencing later pubertal development. This finding is particularly interesting since depressive symptoms were controlled in the regression analyses, suggesting that mood changes are not solely responsible for the relationship between pubertal development and sleep problems. While this study is unable to address the underlying cause for the relationship, it might be that the biological changes to circadian rhythm that are known to be ushered in by the onset of puberty (Carskadon & Acebo, 2002, Hagenauer, Perryman, Lee & Carskadon, 2009) may be particularly out of sync with external forces that shape sleep habits when the onset of puberty comes early. For example, bedtimes set by parents based on the chronological age of the child may not be in accord with the child's changing sleep preferences. The mismatch between the youth's internal clock and environmental demands and constraints has been proposed as a cause for sleep problems in typically developing youth (Wolfson & Carskadon, 2005) and may be exacerbated when pubertal development is early.

In addition to biological influences on the change in sleep variables from late childhood into adolescence, the effect of parental monitoring on change in sleep was also examined. Parental monitoring did not predict changes in sleep duration or sleep variability, however greater parental monitoring at sixth grade was found to be predictive of greater increases in sleep problems from sixth grade into 15 years. This finding was contrary to the prediction and recent research (Short, Gradisar, Wright, Lack & Carskadon, in press) that



parental monitoring would minimize or buffer changes in sleep into adolescence. There are a few possible explanations for this unexpected finding. First, it may be that the measure of parental monitoring does not include enough information that is specific to the monitoring and shaping of sleep habits. If the measure was focused on parental behaviors regarding bedtimes and wake times, restriction of television and media in the bedroom, and other socialization around healthy sleep behaviors, the findings may have been in the expected direction. On the other hand, it may be that parental monitoring is a response to pre-existing behavior problems (including sleep problems) and when measured at sixth grade, high levels of parental monitoring are reflective of youth who have demonstrated problems with sleep or behavioral regulation earlier in development. These youth might, therefore, be at greater risk for increased sleep problems across the transition to adolescence.

Finally, the possibility exists that greater parental monitoring at sixth grade prevents youth from developing their own strategies for sleep regulation leading to greater problems when more autonomy is granted regarding sleep. This idea may be the developmentally appropriate extension of research that considered the role of parents in bedtime routines for school-aged children. Using the National Sleep Foundation's "Sleep in America" survey, Mindell and colleagues (2008) showed that parental involvement in sleep routines through behaviors that are used to initiate sleep onset is associated with longer times to get to sleep and more night wakings. Findings like these have led the NSF to recommend that parents encourage children to "self-soothe" and become independent sleepers. This study's finding that parental monitoring led to greater increases in sleep problems across the transition to adolescence may be further support for encouraging independent sleep regulation beyond early childhood.

In looking at the relationship between sleep and risk-taking, this dissertation tested the influence of sleep duration, sleep variability and sleep problems and changes in those variables on risk-taking behaviors separately, an approach that was justified by the low intercorrelations between the variables. Although all of the sleep variables and change in sleep variables predicted general risk-taking, they did not equally predict other sexual risk-taking or substance use. Further the proportion of variance explained by the sleep variables differed. From this data, it is not entirely clear if some of these sleep variables are merely more reliable or sensitive than others or if each sleep variable provides different pieces of information about underlying processes that are related to risk-taking. For example, the fact that the *change* in sleep variables from sixth grade to 15 years explained more variance in risk-taking at 15 years compared to sixth grade sleep variables is worthy of note. It is entirely possible that this was true simply because of the temporal proximity to the outcome measurement. Alternatively, it may also be that greater change in sleep duration, variability and problems are signals that the individual is not managing the transition to adolescence well. From studies of children dealing with marital conflict, researchers have suggested that sleep may provide valuable insights for parents and clinicians regarding a child's ability to cope with stressors (El-Sheikh, Buckhalt, Mize & Acebo, 2006). Likewise, it may be that greater changes in sleep (all reflecting worsening sleep) occur in individuals who are really struggling with the transition to adolescence and risk-taking behaviors are another maladaptive way of dealing with this transition.

Pubertal development was not found to moderate the relationship between sleep and risk-taking. Unfortunately, the timing of data collection with the two time points nearly three years apart, may have affected the ability to detect this relationship. Since the biological and

social changes that occur with pubertal development can unfold rather quickly, it may be that a more proximal measure of pubertal development would have shown a significant moderating effect on the relationship between sleep and risk-taking.

Parental monitoring, however, did moderate the relationship between changes in sleep problems and general risk-taking. It appears from this data that higher levels of parental monitoring may buffer against the effects that changing sleep habits have on risk-taking. If adolescents are staying up later as a result of changing circadian rhythm and sleep preferences, then parental monitoring may serve to restrict their access to “late night dangers”, such as delinquent peers and unsupervised activities.

One goal of this dissertation was to uncover possible mediating processes that might explain the association between sleep and risk-taking. Executive function was considered a good candidate mediator because research has found that insufficient sleep can affect executive functioning and executive functioning has been considered an important cognitive component of risk-taking and decision-making. As the previous research would suggest, a significant relationship between poor executive functioning and higher risk-taking was observed. However, no evidence was found for mediation of the model linking sleep problems to risk-taking. It may have been that the two tasks, the Stroop test and the Tower of London, which were used to assess executive functioning, were not sensitive enough to allow detection of a mediating effect. It is also possible, however, that the timing of data collection had a detrimental effect on the ability to detect a relationship because sleep variables were measured nearly three years before executive functioning was assessed. Most of the prior research on the effects of sleep on executive functioning found the relationship in more immediate conditions. For example, sleep deprivation over the previous night (Killgore,

Balkin & Wesensten, 2006) or week (Sadeh, Gruber & Raviv, 2003) has been shown to lead to decreases in executive function abilities. The differences between information about sleep and its effects on daytime functioning that are derived under acute versus chronic/persistent conditions might suggest coping processes that unfold across time.

An indirect effect of impulse control on the relationship between all sleep variables separately and general risk-taking was found. This indirect effect suggests that, individually, shorter sleep duration, greater variability and greater sleep problems at sixth grade were predictive of poorer impulse control at 15 years, which was, in turn, associated with greater risk-taking at that time point. It is interesting to note that this potential mediating effect was significant, although executive functioning was not. Presumably, executive functioning abilities underlie the ability to control one's impulses. However, it may be that the impulse control taps into processes that are more pertinent for the specific outcome of risk-taking. Sternberg's theory of risk-taking suggests that the increase in risk-taking seen during adolescence is the net effect of poor regulatory systems and particular sensitivity to rewards during this period. Perhaps, the direct of measure of executive functioning only is tapping into one component of this phenomenon (poor self-regulation). The measure of impulse control, on the other hand, is providing more information about how the individual has behaved in situations that require impulse control, a product of their sensitivity to reward and regulatory abilities.

In whole, the findings of this study are a significant addition to our current understanding of the association between sleep and risk-taking. Previous findings of an association were replicated, but importantly, they were replicated while controlling for potential third variables (child depression and income to needs ratio) that had not been

controlled in previous research and were replicated across a critical developmental transition. Pubertal timing, which has been linked to sleep changes and risk-taking in prior research, predicted changes in sleep but did not moderate the relationship between sleep and risk-taking. Parental monitoring, which has been shown to be an important influence on risk-taking, appeared to have a buffering effect on the relationship between sleep problems and risk-taking across the transition from late-childhood to adolescence. Finally, this study suggests that sleep's effects on impulsive behavior may partially explain the relationship between sleep and risk-taking during adolescence. All of the significant findings raise additional questions about timing, reciprocity, and explanatory mechanisms that deserve closer investigation, particularly in light of the building evidence that sleep and risk-taking are potentially causally related.

### *Limitations*

This dissertation sought to broaden the current understanding of the association between sleep and risk-taking behavior by utilizing an existing large, longitudinal dataset. The NICHD SECCYD dataset included many variables that were relevant to the study. However, there are a number of limitations that are the result of the fact that the data was not collected directly for the purposes of this study. Of primary importance to the central questions of this study is the measurement of sleep variables. Although previous research has found that self-report methodologies for obtaining information about sleep duration may be reliable (Sadeh, 2008; Sadeh, Gruber, & Raviv, 2002), the measurement of the sleep variables using self-report surveys is not optimal, especially for measuring sleep quality. That said, the significant findings in this study related to sleep while using self-report measures underscores the importance of gathering objective data, which would likely lead to more

robust findings. Objectively collected sleep data would not only be a more reliable account of sleep habits but provides information about sleep that is not within an individual's conscious awareness. Recent research has suggested that understanding sleep quality is perhaps just as or more important than hours slept for predicting daytime behavior (El-Sheikh, Buckhalt, Keller, Cummings & Acebo, 2007), so objective sleep measurement, which captures this information, would be of great benefit to the field.

In this study, I had no direct measure of sleep quality and instead sleep problems were assumed to reflect quality of sleep. Interestingly, the sleep problems variable produced a number of significant findings. First, sleep problems were found to be significantly influenced by both internal and external forces (pubertal development and parental monitoring). In addition, sleep problems explained a significant amount of variance in later risk-taking behaviors. Therefore, given the importance of self-reported sleep problems to understanding the relationships between study variables, the question of what exactly is being measured by this self-report survey is critical. I did control for child depressive symptoms, at least in part, to deal with concerns over reporter bias on the self-report instrument, but it is still possible that individuals who were more likely to report a high level of sleep problems also have cognitive and behavioral tendencies to focus on negative aspects of their experience. The addition of objectively measured sleep quality would certainly allow insights into extent to which the survey actually provides information about an individual's awareness of their sleep quality. Further, it would be important to compare, within one study, the self-reported and objectively measured data as they may provide different pieces of information about sleep and relate differently to risk-taking behavior.

The multimodal measurement of risk-taking was seen as an advantage of this study over others. The measurement of “real world” risk in this study was done using the Risky Behavior Questionnaire, a widely used self-report measure. It was hoped that adding the Balloon Analogue Risk-Taking, a lab-based task of risk-taking, to this study would provide more information about risk-taking propensity, isolated from the social and environmental factors that may or may not support risk-taking behavior in the real world. If, for example, the sleep and risk-taking association exists because youth who have poor sleep habits are more likely to be exposed to late-night dangers (older peer groups, less monitoring, etc.) and not because of the effects of poor sleep on decision-making, then one might have expected to see that sleep significantly predicted real world risk-taking and not lab-based risk-taking. In this study, the BART lab-based task did not produce any significant findings. It may be that the lack of significant findings reflects the fact that social and environmental influences are solely responsible for the association between sleep and risk-taking. However, it may be more likely that there was some flaw in the administration or scoring of this task. The fact that the BART measure did not correlate with any of the real-world risk-taking measures puts into doubt the external validity of the task. Previous research using the BART has found moderately strong associations between this lab-based measure and real-world risk-taking in adolescents (Lejuez, Aklin, Daughters, Zvolensky, Kahler & Gwadz, 2007).

Although one advantage of this study over previous research is the timing of data collections that captures an important transition, marking developmentally normative changes in sleep and risk-taking, additional data collections would have improved this study. For example, it would have been very helpful, especially for understanding possible mediators of the sleep and risk-taking association, to have an intermediating data collection

time point. As Cohen et al. (2003) point out, when testing mediational models with only two time points and the proposed mediator measured at time 2, one risks the possibility that the effect of the mediator may be in part due to the causal effect of the outcome on the mediator, rather than vice versa as the model assumes. In this study, impulse control was measured at the same time as risk-taking (at 15 years), so the finding that the relationship between sleep and risk-taking was mediated by impulse control may be overestimated. By adding an intervening data collection at which point the possible mediating variables would have been measured, concerns about overestimation or underestimation (in the case of the mediator being measured at time 1) would be addressed. Furthermore, the timing of this study leaves many unanswered questions about what was happening in terms of sleep and risk-taking during the three years between data collections. Additional intervening time points would have allowed for testing of reciprocal influences across time, using cross-lagged models.

Ultimately, the greatest limitation of this study may be the inherent difficulty in describing complex and dynamic interactions overtime with simple regression models. This limitation is certainly not unique to this study. If one accepts the developmental systems framework that emphasizes the importance of placing behavior in context and defines “context” on individual, family and community levels, simple approaches may seem akin to modeling the Sistine Chapel with toothpicks and Play-Dough. The tools and materials simply are not up to the demands of the task. However, given the lack of research that has been done in the area, the analyses conducted for this dissertation are a necessary and important first step. I was able to make use of an existing dataset to examine biological, cognitive, behavioral and family level influences on the association between sleep and risk-taking. While controlling for individual and family level factors that have not been previously



accounted for, this research provides more detailed evidence of the relationship between sleep and risk-taking across a significant developmental transition in a large, national sample. It suggests a number of areas for future research, which are discussed in detail in the following section.

### *Future directions*

Research on the subject of sleep during adolescence and the influence sleep may have on still developing systems is in its infancy. The changes to sleep that occur around puberty have only started to be viewed as significant in light of research questioning the wisdom of early school start times for high school students (Wolfson & Carskadon, 2005, 2003). As studies are starting to show that poor sleep can have detrimental effects on cognitive functioning, health, and behavior in young people and adults, it is likely that there will much more work done to understand sleep's influence in the coming years. A necessary complement to this line of work will be research that seeks to understand the factors and processes that shape sleep changes.

This dissertation showed changes in sleep duration, variability and problems across the transition to adolescence that were all reflective of worsening sleep. At the same time, this study also showed that there are considerable individual differences in those changes but was unable to provide much information about the source of those differences outside of the two proposed moderators, pubertal development and parental monitoring. Although researchers have made great strides in understanding the biological mechanisms that underlie changes in sleep and sleep preferences (Hagenauer, Perryman, Lee & Carskadon, 2009), far less is known about the behavioral, social and cultural influences on sleep that may

exacerbate or buffer against those biologically driven changes. Further, understanding what contributes to individual differences in the change in sleep overtime could prove to be helpful in explaining the link between sleep and risk-taking as common forces might influence both behaviors.

The impact of included covariates on the sleep and risk-taking also deserves further investigation. Of particular note, child depressive symptoms at sixth grade were included as a covariate in all regression analyses and, although not the focus of this research, were found to explain a significant amount of variance in sleep and in the relationship between sleep and risk-taking variables over time. First, this finding suggests that future studies examining the association between sleep and risk-taking should take into account the influence of depressive symptoms. More importantly, future research should seek to elucidate the role of depressive symptoms as they may initiate, maintain, or be a consequence of poor or inadequate sleep and have a role in problem daytime behaviors. A recent review of research regarding the consequences of loneliness by Hawkey and Cacioppo (2010) suggests that the poor quality of sleep in lonely individuals interferes with the restorative effects of sleep leading to a negative cycle of daytime and nighttime dysfunction. Similarly, it may be that depressive symptoms (quite possibly the result of loneliness during adolescence) and sleep problems are operating to exacerbate each other. Research using objective measurement of sleep and daily sampling methods could prove very useful in understanding this potentially negative feedback loop.

In addition to future research on links between mental health and sleep, our rapidly expanding understanding of the importance of sleep behaviors for overall wellbeing suggests that this might also be a fruitful area of inquiry. Poor sleep has been linked to chronic

inflammation, immune function, and chronic diseases like diabetes and obesity (Foster & Wulff, 2005). In addition, epidemiologic research with adult samples has shown significant socioeconomic and ethnic differences in sleep patterns and the suggestion has been made that these sleep differences may be a source of the socioeconomic and ethnic health disparities in America (Patel, Grandner, Xie, Branas & Gooneratne, 2010; Hale & Do, 2007). Therefore, examination of what shapes sleep behaviors may be of particular interest to researchers in the area of public health. While understanding the proximal consequences of poor sleep during adolescence is worth investigation in itself, the long-term consequences of adolescent sleep changes on adult sleep patterns may be even more significant to understanding quality of life and longevity (Shonkoff, Boyce & McEwen, 2009). Future research might seek to understand the extent to which the changes that occur during adolescence to sleep are merely temporary or, rather, set trajectories for adult behavior patterns. Transitions in development are often periods of increased plasticity; if this is the case with sleep patterns, adolescence might be a particularly potent time to intervene on problematic sleep behaviors before they are canalized in development.

The application of a developmental systems framework suggests a number of other avenues for future research that might allow us to gain a better understanding of the sleep risk-taking association. Sleep and risk-taking are clearly very complex behaviors, understanding their association is exponentially more complex; a point illustrated by the fact that models tested in this study, while significant, explained only a modest proportion of variance in the relationship between key variables. The developmental systems framework emphasizes dynamic and reciprocal influences on behavior that extend from genes to culture and there exists solid evidence that sleep (Jenni & O'Connor, 2005) and risk-taking (Conner,

Hellemann, Ritchie & Noble, 2010) are particularly good illustrations of the “interplay between biology and culture”. As such, future research should explore possible sources of the association between sleep and risk-taking on genetic, neurobiological, behavioral and cultural levels. Not only might the suggested research provide insights about the particular association of behavior across time, but also about larger issues about stress and coping in developing systems.

The possibility that the association between sleep and risk-taking behavior might result from common genetic influences or the interaction between genes and environment should be a goal of future research. Genes have been identified that are important to setting circadian rhythms (Ebisawa, 2007; von Schantz & Archer, 2003) and understanding individual differences in sleep need (Raizen, Mason & Pack, 2006) and reactions to sleep loss (Goel, Banks, Mignot & Dinges, 2010). Likewise, risk-taking has been associated with a number of genetic variations for everything from risky decision making (Juhsaz et al, 2010), to sexual risk-taking and delinquency (Verweij, Zietsch, Bailey & Martin, 2009), and substance use (Connor, Hellermann, Gerhard, Ritchie & Noble, 2010).

Of particular interest, some overlap exists in the research on genetics of sleep and risk-taking. For one, variations in the serotonin transporter gene (5HTT), which regulates serotonin function and uptake, has been linked to sleep quality under certain conditions (Brummett, et al, 2007) and risk-taking propensity (Kuhnen & Chiao, 2009). The cumulative evidence from a number of observational and experimental studies in humans and non-human primates looking at serotonin allele polymorphisms has generated a hypothesis that this gene modifies an organism’s sensitivity to stress in the environment (Caspi, Hariri, Holmes, Uher & Moffitt, 2010). Under this hypothesis, poor sleep and risk-taking,

particularly substance use, could be understood as different behavioral reactions to stress, so a gene that alters sensitivity to environmental stressors might be particularly relevant to describing their association. Based on the previous research and current understanding of the role of serotonin transporter genes, the 5HTT gene would be an excellent focus for future research. Prior work also suggests that any research into the genetic basis for an association between sleep and risk-taking would be well advised to consider gene X environment interactions.

Other neurobiological links between sleep and risk-taking may also be worth investigating, including the role of cortisol. Cortisol, a glucocorticoid released in response to stress, is known to also be important for the regulation of sleep and wake cycles (Randler & Schaal, 2010), having acquired the nickname of the “anti-sleep” hormone. At the same time, researchers have shown that cortisol is related to risk-taking through effects on sensation seeking (Freeman & Beer, 2010) and decision-making (van den Bos, Harteveldt & Stoop, 2009) and that differences in stress reactivity (as measured by cortisol response to a number of stressors) are related to actual risk-taking behavior (Halpern, Campbell, Agnew, Thompson & Udry, 2002). Base levels of cortisol and fluctuating cortisol responses to stress, therefore, might be a common influence on sleep and risk-taking that explain their association. Risk-taking during the day might lead to increased levels of cortisol, which would inhibit sleep onset in the evening, lead to less restorative sleep and greater propensity for risk-taking during the following day. Further, it is possible that reciprocal influences of sleep and risk-taking behaviors on cortisol responses not only operate on a day-to-day basis but also may lead to cortisol dysregulation across time.

This dissertation revealed that impulse control may be an important individual characteristic that links sleep to risk-taking, however a number of other temperamental or personality traits might underlie the association between sleep and risk-taking. Self-regulation, for example, may lie at the heart of the association between sleep and risk-taking and may also explain why youth with other problems with self-regulation, such as ADHD, also have been found to have higher than normal rates of sleep problems (Alfano & Gamble, 2009). Certainly, research has found a substantial link between self-regulatory abilities and risk-taking behavior already (Magar, Phillips & Hosie, 2008) and a link between sleep and self-regulation during infancy has been studied (Feldman, Weller, Sirota & Eidelman, 2002), but little is known about how self-regulation effects sleep of adolescents. This line of inquiry may be particularly fruitful in explaining the relationship between sleep and risk-taking, and more broadly, about what sleep may tell us about an individual's regulatory abilities.

In this dissertation, parental monitoring was included as a potential common influence on sleep and risk-taking, however, future research may also consider other external forces such as peer groups. Peers are known to be important predictors of risk-taking behavior (Gardner & Steinberg, 2005), but almost nothing is currently known about peer influences on sleep habits. However, one might reasonably guess that sleep habits, like other traits, demonstrate homophily and peer groups would, consequently, be likely to share sleep behaviors. The peer effect on sleep may be all the more potent in today's world when youth can be technologically connected with each other, influencing each other's sleep, even while in the privacy of their own homes.

Cumulative risk models suggest an additional approach to understanding the association between sleep and risk-taking in which one considers the accumulation of risk

instead of the influence of individual risk variables separately. Potentially, this approach may provide additional information, especially as it pertains to the emergence and maintenance of problem behaviors (Moffitt & Caspi, 2001). Indeed, recent research using the SECCYD dataset (Roisman, Monahan, Campbell, Steinberg & Cauffman, 2010) has found that youth with persistent anti-social behavior problems through 15 years were more likely to have experienced a number of disadvantages from infancy, including being raised in single-parent homes, low levels of maternal sensitivity, poorer cognitive functioning, poorer health and lower socioeconomic status. Undoubtedly much could be gained if similar analyses were conducted looking at the emergence of sleep changes and the maintenance of those changes through adolescence as the outcome. That information might be useful in uncovering common precursors of sleep and risk-taking behaviors. More importantly, future research that considers cumulative risk, sleep and risk-taking behavior may provide information about the regulation of behavior and aspects that are particularly sensitive to the negative effect of stressors.

Having discussed a number of potential factors on multiple levels that future researchers exploring the link between sleep and risk-taking during adolescence should consider, it should be said that full application of a developmental systems approach to this question will also require methods that can capture potential reciprocal influences over time. Current thinking on the changes in sleep and risk-taking during adolescence suggests that intrinsic and extrinsic forces work together. Unfortunately, current research of those intrinsic and extrinsic forces remains isolated. Much could be gained by quasi-experimental studies, like the research of Forbes and Dahl (2010) that exploit naturally occurring variations in pubertal development to better understand how biological and social levels interact.

Timing in terms of development, but also in terms of research design, are critical to gaining a better understanding of the relationships between sleep and risk-taking. If we don't look closely enough at interactions that occur on a daily basis, we risk missing important information about coping mechanisms that work to alter the effects that sleep and risk-taking may have on each other. If we look too closely, we will likely overlook the importance of factors that take time to unfold. For these reasons, research that seeks to shed light on the feedback loop between daytime behaviors (including risk-taking) and nighttime sleep should be done on both micro and macro time scales.

In this dissertation, I have attempted to integrate information about biological maturation (pubertal development), cognitive functioning (executive functioning), social forces (parental monitoring), and behavioral tendencies (impulse control) that influence and potentially explain the link between sleep and risk-taking. It is interesting to note that the focus of this study was on the association between sleep, a relative unknown, and risk-taking, a very well explored topic of adolescent development. As a result, much of the variables that were considered as either moderators or mediators were chosen primarily from what is known about their relation to risk-taking. If more research is done to understand the predictors and consequences of poor sleep and sleep changes during adolescence, then the factors that may be investigated in answering the question of the association between sleep and risk-taking will likely look different and may produce additional significant findings. Going forward, I believe that this dissertation shows that there is great utility in applying the developmental systems framework to research on the reciprocal influences between sleep and daytime behavior. It is hoped that future work will allow the field to gain a better understanding of the function of sleep, from genes to culture.



Table 1- Descriptive characteristics of NICHD Study of Early Child Care sample (n=1077)

<u>Variable</u>	
<i>Baseline Characteristics(Sixth Grade)</i>	
Child gender	
Female	51%
Male	49%
Ethnicity	
White	78%
African American	11%
Latino	6%
Other	5%
Parent education	
HS or less	39%
Some college/College degree	49%
Post-graduate training	12%
Income to Needs Ratio	$M=4.54, SD=4.15$
Child Depressive Symptoms	$M=1.41, SD=2.15$
Pubertal Development	Range (1 to 5)
	male $M=1.60, SD=.71$
	female $M=3.00, SD=.77$
Parental Monitoring	Range (1 to 4)
	$M= 2.51, SD=.47$

Table 2- Study variables

Construct	Instrument/Task	6 <sup>th</sup> grade Collection	15 year Collection
Executive Function (mediator)	Stroop task		Child
	Tower of London		Child
Depressive symptoms (co-variates)	Child Depression Inventory	Child	Child
Impulsivity (mediator)	Weinberger Adjustment Inventory		Child
Parental Monitoring (third variable)	Parental Supervision and Monitoring	Child	Child
Pubertal Timing (third variable)	Health and Physical Development Assessment (Tanner Stage)	Clinician	
Risk-taking, real-world (dependent variable)	Risky Behavior Questionnaire	Child	Child
Risk-taking, in lab (dependent variable)	Balloon Analogue Risk Task		Child
Sleep Habits -sleep duration -sleep schedule variability -sleep problems  (independent variables)	My Sleep	Child	Child

Table 3- Intercorrelations, means and standard deviations for key variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	
1. Sleep duration (6 <sup>th</sup> )		1.00											
2. Sleep variability (6 <sup>th</sup> )	-.27**	1.00											
3. Sleep problems (6 <sup>th</sup> )	.01	.13**	1.00										
4. Sleep duration (15yr)	.34**	-.12**	.03	1.00									
5. Sleep variability (15 yr)	-.08*	.23**	.04	-.04	1.00								
6. Sleep problems (15yr)	-.06	.13**	.37**	-.05	.07*	1.00							
7. Overall risk-taking (6 <sup>th</sup> )	-.18**	.24**	.26**	-.09**	.16**	.17**	1.00						
8. Substance use (6 <sup>th</sup> )		-.11**	.09**	.10**	-.04	.02	.06	.47**	1.00				
9. Overall risk-taking (15yr)	-.15**	.16**	.12**	-.19**	.20**	.18**	.53**	.14**	1.00				
10. Substance use (15yr)	-.08*	.12**	.07*	-.09**	.16**	.17**	.28**	.11**	.58**	1.00			
11. Sexual risk-taking (15yr)	-.10**	.06	.05	-.13**	.11**	.12**	.27**	.14**	.45**	.44**	1.00		
12. B.A.R.T. (15yr)	-.06	.01	-.02	.02	.03	-.03	.03	-.01	.02	.02	-.02	1.00	
<i>M</i>		9.48	1.41	19.55	8.27	1.73	24.01	.12	.04	.39	.65	.11	36.33
<i>SD</i>		.79	1.10	5.16	1.01	1.25	5.59	.09	.28	.19	1.34	.26	12.19

Note. \* $p < .05$ , \*\* $p < .01$

Table 4- Change over time for key study variables

Variable	<u>6<sup>th</sup> Grade</u>		<u>15 yrs</u>		<i>t(df)</i>	<i>p</i>	<u>95% CI</u>		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			<i>LL</i>	<i>UL</i>	
Sleep duration	9.48	.79	8.28	1.00	33.31(868)	.000	1.12	1.26	1.33
Sleep variability	1.39	1.07	1.69	1.26	-6.10(826)	.000	-.041	-.21	-.26
Sleep problems	19.53	5.09	24.04	5.55	-23.23(946)	.000	-4.90	-4.13	-.85
All risk-taking	2.26	1.91	6.14	5.67	23.95(932)	.000	3.57	4.20	-.92
Substance use	.04	.25	.64	1.33	-13.74(932)	.000	-.68	-.51	-.63

*Note.* CI= confidence interval; *LL*=lower limit; *UL*=upper limit.

Table 5- Sleep variables predicting risk-taking

<u>General risk-taking</u>						
<u>Model 2</u>						
Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.355(.017)**			.580(.082)**		
Collection site	.001(.002)			.001(.002)		
Gender	.078(.013)**			.073(.014)**		
Income to needs	-.007(.002)**			-.007(.002)**		
Child depression	.013(.003)**	.079	17.026**	.013(.003)**		
Sleep duration (6 <sup>th</sup> gr)				-.024(.008)**	.088	15.291**

Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.333(.018)**			.295(.021)**		
Collection site	.002(.002)			.003(.002)		
Gender	.080(.014)**			.079(.014)**		
Income to needs	-.007(.002)**			-.006(.002)**		
Child depression	.015(.003)**	.094	18.192**	.015(.003)**		
Sleep variability (6 <sup>th</sup> gr)				.023(.007)**	.12	17.22**

Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.355(.017)**			.287(.031)**		
Collection site	.001(.002)			.001(.002)		
Gender	.078(.013)**			.078(.013)**		
Income to needs	-.007(.002)**			.007(.002)**		
Child depression	.013(.003)**	.079	17.03**	.010(.003)**		
Sleep problems (6 <sup>th</sup> gr)				.004(.001)*	.086	15.06**

Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.604(.086)**			.747(.091)**		
Collection site	.000(.002)			.001(.002)		
Gender	.077(.014)**			.075(.014)**		
Income to needs	-.007(.002)**			-.007(.002)**		
Child depression	.013(.003)**			.013(.003)**		
Sleep duration (6 <sup>th</sup> gr)	-.027(.009)**	.096	15.58**	-.013(.009)		
Sleep duration (15 year)				-.033(.008)**	.12	16.51**

Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.301(.021)**			.279(.021)**		
Collection site	.003(.002)			.002(.002)		
Gender	.079(.014)**			.077(.014)**		
Income to needs	-.006(.002)**			-.006(.002)**		
Child depression	.015(.003)**			.014(.003)**		
Sleep variability (6 <sup>th</sup> gr)	.022(.007)**	.113	1.63**	.017(.007)*		
Sleep variability (15 year)				.020(.005)**	.13	17.12**

Variable	Model 1 <i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>B(SE)</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Constant	.291(.031)**			.180(.039)**		
Collection site	.001(.002)			.001(.002)		
Gender	.079(.013)**			.091(.013)**		
Income to needs	-.008(.002)**			-.007(.002)**		
Child depression	.010(.003)**			.009(.003)**		
Sleep problems (6 <sup>th</sup> gr)	.004(.001)*	.090	15.70**	.001(.001)		
Sleep problems (15 year)				.006(.001)**	.12	17.17**

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*Note.* *N*= 897, \**p*<.01, \*\**p*<.001



Table 6- Analysis of impulse control as a mediator

		<i>a</i> path effects	<i>b</i> path effects	Mediated effect	Sobel test statistic
		( <i>X</i> → <i>M</i> )	( <i>M</i> → <i>Y</i> )	( <i>c</i> ')	
Sleep duration					
75	→general risk-taking	.073(.037)*	-.161(.014)**	-.041(.015)**	-1.95*
Sleep variability					
	→general risk-taking	-.104(.028)**	-.148(.014)**	.019(.011)	3.50**
Sleep problems					
	→general risk-taking	-.030(.001)**	-.163(.014)**	.001(.003)	10.85**

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