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Sleep Problems Across Development: A Pathway to Adolescent Risk Taking Through Working Memory

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Abstract Problematic sleep can be detrimental to the development of important cognitive functions, such as working memory, and may have the potential for negative behavioral consequences, such as risk-taking. In this way, sleep problems may be particularly harmful for youth—whose cognitive abilities are still developing and who are more susceptible to risky behavior. Using data from a large, national, longitudinal study, continuity and change in sleep problems were examined from 2 to 15 years of age and associated with deficits in working memory at age 15 and risk taking behaviors at age 18. Participants ($N = 1,364$ children; 48.3 % female) were assessed for sleep problems (parent-report), working memory (behavioral task), and risk taking behavior (youth self-report). The sample was predominantly White (80.4 %); additional races represented in the sample included Black/African American (12.9 %), Asian/Pacific Islander (1.6 %), American Indian/Eskimo/Aleut (.4 %), and Other (4.7 %). The findings suggest that sleep problems are likely to cascade across development, with sleep problems demonstrating continuity from infancy to early childhood, early childhood to middle childhood, and

middle childhood to adolescence. Although sleep problems in infancy, early childhood, and middle childhood were not directly related to adolescent working memory, sleep problems during adolescence were associated with poorer adolescent working memory. In turn, these deficits in working memory were related to greater risk taking in late adolescence. In summary, the present results suggest that sleep problems in earlier periods are indicative of risk for sleep problems later in development, but that sleep problems in adolescence contribute uniquely to deficits in working memory that, in turn, lead to risky behavior during late adolescence.

Keywords Sleep problems · Sleep disturbances · Working memory · Risk taking · Adolescence

Introduction

It has been estimated that 25–40 % of children suffer from some type of sleep problem (Owens 2005). Moreover, the occurrence of sleep problems in childhood does not appear to be transitory: developmental continuity in sleep problems has been documented from infancy to childhood, such that 41 % of infants identified as having sleep problems at 8 months experienced continuing difficulties at 3 years (Zuckerman et al. 1987). Continuity has also been observed across childhood, such that children who experienced sleep problems before age 2 years were more likely to experience continuing sleep issues between 4 and 12 years of age (Stein et al. 2001). Some evidence suggests that this continuity also remains into adolescence for more general sleep problems, but not for more specific sleep problems, such as nightmares, bedwetting, or talking/walking in sleep (Friedman et al. 2009).

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Adolescence is a time marked by dramatic changes in sleep patterns (Colrain and Baker 2011). During puberty, adolescents experience significant reductions in slow wave sleep (Carskadon 2002), as well as changes in their circadian rhythm known as a “delayed phase preference,” which involves a desire to stay up later in the evening and sleep in later in the morning (Hagenauer et al. 2009). Essentially, adolescents’ sleep patterns reflect a change from being an “early bird” to a “night owl.” School start times have largely been incompatible with adolescents’ natural preference for going to bed late and sleeping in, leading many adolescents to experience an increase in daytime sleepiness (Carskadon et al. 1998; Carskadon 2011) and report a greater occurrence of insufficient sleep (Colrain and Baker 2011). Although poor sleep quality or quantity may be detrimental to cognitive functioning at any age, it may be especially harmful during adolescence due to the significant biological and psychosocial changes that occur at this stage (Dahl and Lewin 2002).

One aspect of cognitive functioning that seems to be particularly influenced by poor sleep is working memory, a component of executive functioning. In fact, poor sleep quality has been associated with decreased working memory performance in children and adolescents (Steenari et al. 2003). Working memory is the capacity for storing and manipulating information in one’s mind (Baddeley 2010) and research suggests that it may play a role in adolescent risk taking through its influence on impulse control (Romer et al. 2011). Working memory serves as a cognitive filter, allowing a person to hold information in one’s mind in order to consider potential consequences of one’s actions. In this way, deficits in working memory may contribute to risky decision-making.

Across development, risky behavior tends to follow an inverted U-shaped developmental trajectory that includes normative increases in risk taking during adolescence, followed by declines during the transition to adulthood (Steinberg 2008). This peak in risk taking that occurs during middle to late adolescence is thought to be the result of an imbalance between low impulse control and high reward-seeking tendencies (Steinberg 2010). As youth transition to adolescence, their sleep patterns also undergo important changes that may exacerbate this cognitive–affective imbalance and contribute to risky behavior (Telzer et al. 2013).

Thus, adolescence appears to be a time of greater likelihood of experiencing sleep problems. Yet, for many adolescents, sleep problems do not begin here. Surprisingly, although considerable research has examined how poor sleep at a given time is associated with concurrent or subsequent outcomes, rarely do these studies take into account the pattern of sleep problems across time and how sleep problems at various stages of development may

impact functional outcomes, such as working memory and risk taking behavior. In the present research, we use a longitudinal dataset to examine (a) continuity and change in sleep problems from 2 to 15 years of age, and (b) the association between sleep problems at various stages of development, adolescent working memory, and risky behavior.

Sleep and Working Memory

Working memory, a component of executive functioning, is a multi-faceted ability that involves selective attention, retrieval of past experiences held in long-term memory, and manipulation of information held in the short-term memory (Kane and Engle 2002). Performance on tasks of working memory has been found to improve linearly from age 4 to 15 years (Gathercole et al. 2004). Working memory skills continue to become refined throughout adolescence, as demonstrated by changes in brain activity associated with working memory from childhood through adolescence (see review by Best et al. 2009). With age, prefrontal brain regions appear to become more specialized for working memory. Importantly, brain regions recruited for working memory (such as the prefrontal cortex) seem to be negatively affected by sleep deprivation (Muzur et al. 2002).

Although working memory continues to develop across childhood and into adolescence, it appears that both variability in normative sleep patterns and the incidence of sleep problems has been associated with working memory across different periods of development from infancy to adolescence. For example, in a study of infants and toddlers, greater proportions of time spent sleeping during the night relative to the day at 12 months was associated with better performance on tasks of impulse control and conflict-executive functioning (a factor score comprised of working memory, set shifting, and inhibitory control) at 26 months; higher percentages of time spent sleeping during the night relative to the day at 18 months were associated with concurrent working memory scores at 18 months as well as with impulse control at 26 months (Bernier et al. 2010).

Concurrent sleep problems have been associated with working memory in school-aged children as well. Performance deficits were observed in relationship to variability in normative sleep habits in 6- to 13-year-old children on working memory tasks (Steenari et al. 2003). In particular, the findings indicated that reduced sleep efficiency and duration, as well as increased sleep latency (operationalized as time to transition from wakefulness to sleep), were differentially associated with working memory deficits. Likewise, in a study of 174 high school students, sleep duration was found to be positively associated with

working memory. Specifically, those students who slept less than 8 h per night performed worse on two types of complex working memory tasks than those who slept between 8 and 9 h per night (Gradisar et al. 2008).

The relationship between sleep and working memory appears to be present even among adult populations. In a study of on-call and non-call rotation medical residents, sleep variability and reduced sleep duration were more prevalent during on-call rotations and were significantly associated with a reduction in working memory capacity (Gohar et al. 2009). Some findings are mixed, however. One neuroimaging study revealed that, although sleep deprivation resulted in longer response times on both simple and complex tasks of working memory, performance was only reduced on the simple working memory task; performance on the complex task remained intact following sleep deprivation due to greater compensation by the prefrontal and thalamic brain regions (Chee and Choo 2004). Additional work has suggested that various brain regions are differentially activated following sleep deprivation depending upon the complexity of the working memory task (Choo et al. 2005).

Taken together, these findings reveal complex associations between sleep and working memory that vary with the age of the participants and the measures used to assess sleep and working memory ability. Longitudinal work conducted using the same or conceptually similar measures of sleep over time in relation to later measures of working memory is rare; as such, one cannot know for certain whether only concurrent associations exist between sleep and working memory or whether continuity and change in sleep problems over time differentially contributes to the development of working memory dysfunction. One longitudinal study examining associations between sleep and executive functioning suggests that patterns of developmental change in sleep patterns over time may be more predictive of executive functioning problems relative to the presence or absence of early sleep problems. Friedman et al. (2009) examined associations between the presence of at least one sleep problem during childhood and three components of executive functioning (working memory, inhibition, and task shifting) at 17 years of age. Examination of individual differences in the trajectories of sleep problems over time suggests that the presence of early sleep problems on its own does not predict later executive functioning. Rather, the degree of change in sleep problems over time was more predictive of later executive functioning than the initial level of sleep problems measured in childhood. Although additional work is needed to confirm and extend the findings obtained through longitudinal research, data suggest that the chronicity of sleep problems may be a better predictor of executive functioning deficits relative to concurrent sleep habits or

problems. The present study seeks to replicate this finding, specifically with working memory, and extend it to examine the influence on risky behavior in late adolescence.

Working Memory and Risk Taking

The adolescent brain undergoes numerous developmental changes that make adolescents more vulnerable to risk taking than children or adults. During preadolescence, the dopaminergic system experiences a restructuring that involves a reduction in the density of dopamine receptors in the striatum and prefrontal cortex, leading to an increased dopamine response (see review by Sisk and Zehr 2005). The neurotransmitter, dopamine, is an integral component of the brain's reward circuitry; therefore, the increased dopamine response that occurs following dopaminergic reorganization leads adolescents to experience increased reward-responsivity and sensation-seeking tendencies. Neurobiological models indicate that impulse control develops linearly across development and is not complete in adolescence (Casey et al. 2011). Thus, dopaminergic activity increases prior to complete development of cognitive control systems in the prefrontal cortex, leading to a period of high impulsivity and reward-seeking behavior during mid-adolescence, making this a vulnerable time for risk-taking (Steinberg 2008).

Through its role in impulse control, working memory deficits may leave adolescents at an increased susceptibility to risk-taking. Some researchers have postulated that working memory may be enmeshed with other executive functioning skills, such that engagement of working memory abilities may necessarily involve recruitment of other executive skills, such as inhibition (Bell et al. 2007). It is perhaps because of this overlap that working memory has been associated with impulse control and self-regulation (Whitney et al. 2004). Working memory has been negatively correlated with delay discounting (a critical feature of impulsivity that involves the devaluation of larger long-term rewards for smaller short-term gains; Bobova et al. 2009). In a 3-year longitudinal study of youth, Romer et al. (2011) found that working memory was associated with two types of impulsivity: sensation seeking tendencies (which was associated with better working memory) and acting without thinking (which was associated with poorer working memory). Although both forms of impulsivity were correlated with subsequent risk taking, the relationship was stronger for acting without thinking (Romer et al. 2011). Similarly, acting without thinking was found to mediate the association between weak working memory and early sexual initiation among adolescents (Khurana et al. 2012). Together, these findings suggest that working memory may be necessary for impulse control and

therefore, weak working memory may place adolescents at risk for engagement in a variety of risky behaviors.

Study Aims and Research Questions

The present study seeks to examine the role of sleep problems across development on working memory deficits and risky behavior. Prior research suggests continuity in sleep problems over time, such that those who experience sleep problems in infancy are more likely to experience sleep problems as children, and so on. However, relatively few studies have examined sleep longitudinally across development. Notably, the present study documents the developmental patterns of sleep problems from infancy through adolescence within a single study. In doing so, we test how sleep problems in one developmental period forecast sleep problems in the subsequent developmental period. Note that when we refer to cascade models, we are eluding to a progressive phenomenon that occurs across time in which variables build upon one another (e.g., variable A predicts variable B which predicts variable C). Specifically, we test the continuity of sleep problems across development and separately test how sleep problems in each developmental period are associated with problems with working memory and risk taking. Existing research suggests the prevalence of sleep problems may differ between racial groups (Durrenne and Lichstein 2006), as well as by gender and socioeconomic status (Arber et al. 2009). Further, there may be racial and socioeconomic differences in the relation between sleep and cognitive functioning (Buckholt et al. 2007). Therefore, the present study will use these factors as covariates in the analyses.

The present study will address the following research questions. (1) What is the developmental pattern of problem sleep from infancy through adolescence? Based on prior literature that has shown continuity in early sleep problems throughout childhood and early adolescence (Stein et al. 2001), we hypothesize that there will be continuity in sleep problems over time, such that those who are poor sleepers at one developmental period will be more likely to remain poor sleepers at subsequent developmental periods. Likewise, we hypothesize that those who are classified as good sleepers at one developmental period will be more likely to remain good sleepers in subsequent periods of development. (2) Are sleep problems across development related to working memory? Existing research suggests that sleep problems are associated with concurrent working memory deficits (Steenari et al. 2003); therefore, we seek to extend this finding by examining how sleep across various stages of development will be associated with adolescent working memory. We hypothesize that sleep problems in each developmental period will be

related to working memory in adolescence, but that the concurrent effect of adolescent sleep problems on adolescent working memory will be the strongest. (3) Are sleep problems across development related to risk taking? Several studies have documented an association between poor sleep and risk taking behavior (Catrett and Gaultney 2009; Clinkinbeard et al. 2011; O'Brien and Mindell 2005). Thus, we hypothesize that sleep problems in each developmental stage will be negatively associated with risk taking. (4) Do the effects of sleep on working memory cascade onto late adolescent risk taking? Based on existing research that suggests poor sleep may exacerbate cognitive difficulties related to risk taking (Telzer et al. 2013), we hypothesize that the effects of sleep problems will be associated with working memory deficits in adolescence, which in turn will be associated with risk taking in late adolescence.

Method

Participants

Participants were selected from a sample of children and mothers enrolled in the National Institute of Child Health and Human Development Study of Early Childcare and Youth Development (for a full description of the study see The NICHD Early Child Care Research Network 2005). Beginning in 1991, participants for this study were recruited from selected hospitals at 10 data collection sites across the United States. Families were recruited using conditional random sampling to ensure an appropriate distribution of families from various backgrounds and childcare experiences. Eligibility criteria maintained that enrolled newborns were born healthy and full-term, mothers spoke English, families planned to live in the catchment area for at least a year and did not reside in neighborhoods that were considered too dangerous for visitation (according to police), among other factors. Additional details about the recruitment and exclusionary criteria used to select participants are available at the study website (<http://www.nichd.nih.gov/research/supported/secyd/overview.cfm>). All participants gave their informed consent prior to their inclusion in the study (parental consent was obtained for youth participants).

Youth participants remained in the study from birth through age 18 years and were assessed intermittently across this time. The study involved five waves of data collection: Phase I (birth through age 3 years), Phase II (54 months through 1st grade), Phase III (2nd through 6th grades), Phase IV (7th through 9th grades), and Phase V (age 18 years). The Phase I sample consisted of 1,364 youth and their families (out of the potential 8,986 families that were screened for eligibility). The overall retention

rate was 57.3 %, with 782 of the original 1,364 children participating in the final wave (Phase V) of data collection.

Youth participants were approximately equally distributed in gender (51.7 % male) and were predominately White (80.4 %). Additional races that were represented in our sample included Black/African American (12.9 %), Asian/Pacific Islander (1.6 %), American Indian/Eskimo/Aleut (.4 %), and Other (4.7 %).

Measures

Sleep Problems

The maternal-reported Child Behavior Checklist (CBCL; Achenbach 1991, 1992) and youth-reported Youth Self-Report (YSR; Achenbach 1991) were selected for use as the measure of sleep for this study, as these offered a consistent measure of sleep across all time points. The data from the CBCL were collected at 24, 36, 54 months, kindergarten, 1st grade, 3rd grade, 4th grade, 5th grade, and 6th grade. The data from the YSR were only used at age 15, as this was the first time point that this test was administered to youth participants. The CBCL consists of 99 items describing behavioral and emotional problems, plus an open-ended item for reporting additional problems. The developmentally appropriate CBCL scales were used at each age (i.e., the CBCL/2–3 was used at the 24 and 36 month assessments and the CBCL/4–18 year assessment was used for the 54 month through 6th grade assessments). The YSR consists of 119 items, with 89 items paralleling those found in the CBCL/4–18. This test is administered in a similar format as the CBCL and is appropriate for obtaining self-reports from youth between the ages of 11 and 18.

In order to map onto developmental outcomes of behavior across age, the assessment items in the CBCL change slightly across time. Out of nine potential sleep items that were asked at various developmental time points, only five were asked at every time point. The five consistent items evaluated: (1) trouble sleeping, (2) occurrence of nightmares, (3) whether child appeared overtired, (4) whether child slept less than others their own age, and (5) whether child “talks or cries out in sleep” (early childhood assessment) or “talks or walks in sleep” (age 4–18 assessment). Note the wording of the final item varied slightly from infancy to childhood to follow developmental appropriateness, with the change first appearing in the 54-month assessment. Comparable items are found in both the CBCL and YSR with one exception. The YSR does not ask youth to report on talking or walking in their sleep, while the CBCL does ask parents to report on this behavior. For these analyses, we opted to include all five items regardless of the slight variation in wording on the

fifth item to help promote variability in a measure of sleep problems. Each item was rated on a three-point scale ranging from 0 (not true of the child) to 2 (very true of the child). Higher scores indicate greater sleep problems.

As the CBCL does not have an established subscale for measuring sleep problems, we conducted analyses to test if the five items could be condensed into a single scale. Exploratory factor analysis (EFA) was used to examine whether a single or multiple latent factors best described these five items. In structural equation modeling, EFA results are determined by comparing model fit when different numbers of subscales are estimated (i.e., 1 scale, 2 scales, 3 scales, and so on). At each time point, we conducted an EFA on the sleep items from the CBCL. Across all time points, results indicated that a two-factor model provided better fit to the data than a one-factor model. The two-factor model had excellent fit, as indicated by CFI values greater than 0.95, RMSEA values less than 0.05, and non-significant Chi square values. Table 1 describes the fit indices and rotation models suggested in the EFAs at each time point, comparing the 2 subscale solution to the 1 subscale solution. Given that the age 15 time point had one fewer item than the other time points, we did not conduct an EFA, but rather, tested if the reliability estimates suggested that a similar scale could be created.

In general, consistent factor solutions were derived at each time point. The two factors that emerged appear to reflect two different types of sleep problems, namely sleep disturbances and sleep difficulties. The factor pertaining to sleep disturbances included items pertaining to having nightmares and talking/crying or talking/walking in sleep, whereas the factor associated with sleep difficulties included items regarding trouble sleeping, being overtired, and sleeping less than others. This pattern emerged at six of the ten time points (24, 36 months, kindergarten, 3rd grade, 4th grade, and 6th grade).

We next tested if the loadings for both of the subscales could be constrained to be equal without diminishing model fit. If model fit is not diminished by constraining the parameters to be equal, taking the mean of the items in each scale is appropriate, rather than using factor loadings. At each time point, factor analysis indicated that the loadings could be constrained to be equal with adequate model fit, suggesting that taking the average of items was appropriate instead of using factor loadings. Moreover, at the time points where the solution was more ambiguous, constraining parameters to match the solution derived at other time points did not significantly reduce model fit, suggesting that the same two subscale solution of sleep disturbances and sleep difficulties could reliably be used at each time point. Consequently, we calculated two age-invariant scales of sleep that reflect sleep disturbances (i.e., problems that disrupt sleep: nightmares, walking/talking in

Table 1 Fit indices and variable loadings for a two factor EFA model at each time point on maternal report CBCL items

Time point	Fit indices	EFA factor loadings		Item number ^a	1-Factor loadings	2-Factor loadings	
		1	2			1	2
24 Months	CFI	.806	1.000	1	.466	.012	.650
	RMSEA	.141	.000	2	.631	.754	-.005
	Chi Sq. Sign.	.000	.703	3	.325	.090	.296
	N	1,190		4	.327	-.116	.590
	Missing	64		5	.601	.570	.053
36 Months	CFI	.077	.998	1	.331	.013	.608
	RMSEA	.162	.034	2	.669	.796	.005
	Chi Sq. Sign.	.000	.123	3	.321	.123	.332
	N	1,176		4	.219	-.118	.590
	Missing	78		5	.726	.603	.118
54 Months	CFI	.877	.996	1	.623	.021	.566
	RMSEA	.091	.037	2	.365	2.308	.000
	Chi Sq. Sign.	.000	.117	3	.349	.005	.341
	N	1,057		4	.522	-.050	.626
	Missing	197		5	.297	.067	.238
Kindergarten	CFI	.793	.994	1	.647	.262	.396
	RMSEA	.120	.047	2	.402	.801	-.003
	Chi Sq. Sign.	.000	.070	3	.270	.150	.182
	N	1,058		4	.501	-.002	.839
	Missing	196		5	.273	.351	.052
1st grade	CFI	.808	1.000	1	.748	1.349	.000
	RMSEA	.120	.008	2	.397	.007	.606
	Chi Sq. Sign.	.000	.303	3	.292	.077	.237
	N	1,028		4	.451	.241	.101
	Missing	226		5	.227	-.087	.502
3rd grade	CFI	.783	1.000	1	.629	.286	.407
	RMSEA	.128	.000	2	.403	.702	.000
	Chi Sq. Sign.	.000	.679	3	.372	.081	.379
	N	1,026		4	.466	-.007	.691
	Missing	228		5	.298	.441	-.003
4th grade	CFI	.829	1.000	1	.814	.195	.643
	RMSEA	.117	.000	2	.351	.734	-.002
	Chi Sq. Sign.	.000	.611	3	.217	.143	.154
	N	1,022		4	.501	-.041	.629
	Missing	232		5	.221	.394	.006
5th grade	CFI	.898	1.000	1	.676	.457	.223
	RMSEA	.089	.000	2	.455	.651	-.015
	Chi Sq. Sign.	.000	.687	3	.323	.242	.153
	N	1,020		4	.481	.000	1.134
	Missing	234		5	.316	.352	.037
6th grade	CFI	.879	1.000	1	.753	.296	.536
	RMSEA	.103	.000	2	.499	.873	-.002
	Chi Sq. Sign.	.000	.445	3	.302	.098	.280
	N	1,023		4	.419	-.022	.588
	Missing	231		5	.306	.314	.089

Bolded numbers indicated which latent factor is the best fit for an item. Items with boxes around them represent items where it is unclear which latent factor they should load onto

^a Item 1: Trouble sleeping. Item 2: Nightmares. Item 3: Overtired. Item 4: Sleeps less than most kids. Item 5: Talks/cries or talks/walks in sleep

sleep) and sleep difficulties (i.e., difficulties falling asleep or obtaining adequate sleep quality or quantity: trouble sleeping, overtired, sleeping less than most kids). The scales were calculated by taking the average of the items at each age. Because the YSR had one fewer sleep item than the CBCL, sleep difficulties at age 15 is made up of the single item that loaded onto this scale at the earlier time points. Mother report at age 15 on sleep problems was highly correlated with youth report (sleep difficulties $r = .54$ between youth and mother report; sleep disturbances item $r = .20$ between youth and mother report). It is not surprising that the correlation is more modest on the nightmare item as youth would be better reporters of this internal conscious state than parents and parents would only know a nightmare occurred if a youth disclosed this information. Fifty percent of the data were required in order to receive a value on the subscale. Reliability estimates are biased towards number of items, and on scales with small numbers of items the alpha is a poor indicator of measure fit (Cortina 1993). As such, we rely on the factor analysis solution for support for our solution.

In subsequent analyses (see the results section) these sleep subscales were used to derive latent classes within four developmental periods: infancy (age 2 assessment), early childhood (ages 3, 4½, and kindergarten assessments), middle childhood (1st through 6th grade assessments), and adolescence (age 15 assessment).

Risky Behavior

Self-reported risk taking behavior was measured using the Risky Behavior Protocol when participants were 18 years old. This measure was developed for the SECCYD and was based on constructs included in many studies of adolescent behavior (Conger and Elder 1994; Johnston et al. 2003; Resnick et al. 1997; SAMHSA 2002). This measure addresses several types of risky behavior, including sexual risk taking, safety and violence-related risk behaviors, as well as drug and alcohol-related risk taking. Participants were asked to report how many times in the past year they had engaged in each of the 53 risky behaviors, such as doing something dangerous on a dare or attacking someone with the idea of seriously hurting them. Participants rated their answers on a scale ranging from 0 (never) to 2 (more than twice). Scores were recoded after data collection (0 = never and 1 = at least once) and were summed to create a measure of risk taking. Missing data were imputed with proportional weighting for participants who completed at least 36 items (this would be about 2/3 of the inventory). Possible scores range from 0 to 53, with higher values indicating more risk taking. This scale had good reliability ($\alpha = .88$).

Working Memory

Working memory was assessed as an aspect of executive functioning that is highly related to both sleep issues (Steenari et al. 2003; Gradisar et al. 2008) and risk taking (Romer et al. 2011). In the present study, youth completed the Operation Span (OSPAN) task was used to assess participants' working memory (Turner and Engle 1989) at age 15 in the laboratory setting. Participants were asked to evaluate whether a given answer to a series of math problems was true or false. After completing each math equation, participants were presented with a letter to memorize. After a series of equation/letter pairs (i.e., a set), participants were asked to recall the letters in the order they were presented by checking a series of 12 boxes listed on a recall screen. This entire process represented a single trial. Five different set lengths were used, ranging from 3 to 7 equation/letter pairs per trial. After practicing these activities, participants completed a series of trials. Participants were given feedback regarding their success in answering equations and recalling letters correctly at the end of each trial. Participants were instructed to try to work quickly and to aim for a math accuracy score of 85 % or greater. Once participants had finished 3 trials in each of the 5 different set sizes, for a total of 75 sets in 15 trials, the task was considered complete. For the purposes of this study, the total score was used to represent working memory. This score was obtained using the sum of the number of letters identified correctly across all sets within a trial. The OSPAN task has demonstrated convergent validity by showing consistency with other measures of working memory capacity (Engle et al. 1999).

Covariates

Sex, race (dummy coded for White and not-White), and socioeconomic status were used as control variables throughout the analyses. Socioeconomic status was assessed at 24 months, the earliest time point in the current analyses, based on the US Census Bureau tables as the ratio of family income to the poverty threshold for each household size. The assessment of income to needs at 24 months was highly correlated with the assessment of income to needs at 1 month ($r = .50$) and at age 15 ($r = .49$). In statistical models, these three variables were used as covariates for working memory at age 15 and risk taking at age 18.

Plan of Analysis

In a series of structural equation models, we (a) derived latent classes of sleep disturbances and difficulties to identify different types of sleepers at each age, (b) the

transitional probabilities of moving between latent classes of sleep disturbances and difficulties between each developmental period to determine the developmental patterning of sleep problems across age, (c) the association between sleep problems at each age and working memory at age 15, (d) the association between sleep problems at each developmental period and risk taking behavior at age 18, and (e) whether any documented association between latent classes of sleep problems and risk taking behavior was explained by the association between latent classes of sleep problems and working memory at age 15. We discuss the specific details of these analyses in turn.

Latent transition analysis was utilized to identify patterns of sleep disturbances and sleep difficulties at each developmental age period: infancy (24 months), early childhood (36, 54 months, and kindergarten), middle childhood (1st grade, 3rd grade, 5th grade, and 6th grade), and adolescence (age 15). Within each developmental time period, we used latent profile analyses (a person-centered mixture modeling approach), using MPlus version 6.0 (Muthén and Muthén 1998–2010) to identify groups of individuals who exhibited similar patterns of sleep difficulties and disturbances. To select the best latent class solution, we utilized the Lo–Mendell–Rubin Adjusted Likelihood Ratio Test (Yungtai et al. 2001), which assesses whether the model with k classes provides significantly better fit than the model with $k - 1$ classes. If the LMR-LRT was not significant, the model with $k - 1$ fewer latent classes was selected. In addition to using the LMR-LRT, we examined model entropy and posterior probabilities of latent class assignment as indicators of model fit. Values for entropy and posterior probabilities of latent class membership range from 0 to 1, with higher values indicating more accurate classification of individuals. In general, values over .70 are considered acceptable and values over .90 are considered excellent in terms of fit (Clark and Muthén 2009). Simulation studies indicate that when entropy is high (greater than .80), classifying individuals into latent classes is appropriate (i.e., assigning individuals to membership in their most likely latent class and utilizing that variable as an independent or dependent variable). However, using this method can lead to decreased standard errors of auxiliary variables; consequently, interpreting trend effects is not recommended and researchers should rely on $p < .05$ as a minimum standard (Clark and Muthén 2009).

After the appropriate number of latent classes was decided upon in each developmental period, we conducted a latent transition analysis (Nyland et al. 2007). Latent transition analysis was used to estimate the transitional probabilities of class membership in each developmental period based on the latent class membership in the previous developmental period (i.e., transitional probabilities of

changing sleep classes from infancy to early childhood, from early childhood to middle childhood, and from middle childhood to adolescence). Transitional probabilities indicated the likelihood that individuals remained in the same latent class (continuity) or transitioned to a different latent class (discontinuity) across development.

Because of the complexity of the model, to investigate the longitudinal effects of sleep problems on working memory and risk taking, we used posterior probabilities to assign individuals to their most likely latent class of sleep problems in each developmental period. Subsequently, we tested a series of structural equation models to determine if there were direct effects of sleep problems within each time period on (a) working memory at age 15 and (b) risk taking at age 18. In these models, we compared a series of nested models where we compare model fit to determine if including direct paths from sleep problems at each developmental time period to working memory or risk taking improves the overall structure of model fit. When paths did not improve model fit, a more parsimonious model (i.e., more restricted model where the path is constrained to zero) was used. All of these models included the covariates of sex, race (White or not), and socioeconomic status on the distal outcomes (working memory and risk taking at age 18). Finally, after determining the association between sleep problems and working memory and risk taking, we tested a final statistical model that examined if the effects of sleep problems on working memory cascaded to effect risk taking at age 18. Thus, the results of this final model indicate how sleep problems from infancy to adolescence are related to working memory, and if this, in turn, is related to risk taking in late adolescence.

Approaches to missing data varied by the stage of the analyses. For latent class analyses, values on all sleep scales in a given developmental period were required to be assigned membership in a latent class. Individuals had to have valid membership in at least one latent class to be included in the latent transition analyses. This is standard for mixture modeling and resulted in 110 cases not being included in the latent class analysis. These 110 cases did not differ on any key demographics from the analytic sample. Across the remaining analyses, full information maximum likelihood estimation was used to make use of all participants. Thus, even if a child did not have valid information about latent classes, he or she could provide data on the associations between other variables (e.g., working memory and risk taking, race and risk taking, sex and working memory, and so on). Individuals had to provide data on at least one of the endogenous variables (working memory or risk taking) to be included in these models, but they did not have to include information about sleep at each stage. This is because these participants could provide valid data on other associations within the

Table 2 Latent transition analysis of sleep problems from infancy to adolescence

	Latent status	
	Good sleeper	Poor sleeper
<i>Average probability of membership</i>		
Infancy	0.99	0.97
Early childhood	0.99	0.93
Middle childhood	0.99	0.95
Adolescence	1.00	0.99
<i>Probability of transitioning to...</i>	<i>... Early childhood latent status</i>	
<i>Conditional on...</i>		
<i>... Infancy latent status</i>		
Good sleeper	0.74	0.26
Poor sleeper	0.31	0.69
<i>Probability of transitioning to...</i>	<i>... Middle childhood latent status</i>	
<i>Conditional on...</i>		
<i>... Early childhood latent status</i>		
Good sleeper	0.91	0.09
Poor sleeper	0.74	0.26
<i>Probability of transitioning to...</i>	<i>... Adolescence latent status</i>	
<i>Conditional on...</i>		
<i>... Middle childhood latent status</i>		
Good sleeper	0.69	0.31
Poor sleeper	0.71	0.29

structural equation model (i.e., the association between working memory and risk taking) but not other associations (i.e., the stability of sleep problems). In the final SEM model that estimates the associations between sleep problems over time, working memory, and risk taking, 44 individuals from the sample were not included due to missing data.

Results

What is the Developmental Pattern of Problem Sleep over Time?

In infancy, middle childhood, and adolescence, the LMR-LRT suggested that a 2-class solution provided the best fit to the data (i.e., adding a third class did not improve model fit: infancy (LMR-LRT = 112, $p = .25$), middle childhood (LMR-LRT = 1,214.441, $p = .10$), and adolescence (LMR-LRT = 10.129, $p = .28$). Although results suggested that a 3-class solution did provide better fit to the data in early childhood than a 2 class solution (LMR-LRT = 544.613, $p = 0.2$), one latent class consisted of less than 4 % of the sample. As such, the 2-class solution

was selected as more parsimonious and consistent with the other developmental periods. The results of the latent transition analysis (LTA) are presented in Table 2. The table presents (1) the number of individuals in each latent class at each developmental period and (2) the conditional probability of moving from one status to another across developmental time. Two classes were identified at each time point. One class was marked by low levels of sleep difficulties and disturbances. We call this class the “Good” sleepers. The second class was marked by high levels of sleep problems and disturbances. We refer to this class as the “Poor” sleepers. Figure 1 presents the means on sleep disturbances and sleep difficulties by age period. The number of individuals classified in each of these classes was relatively consistent from infancy to middle childhood, but there were fewer good sleepers in adolescence: 77 % of participants were classified as good sleepers in infancy; 89 % of participants were classified as good sleepers in early childhood; 85 % of participants were classified as good sleepers in middle childhood; 64 % of participants were classified as good sleepers in adolescence. At each time point, posterior probabilities were greater than 0.93 for each latent class, suggesting that individuals were well classified.

Seventy-four percent of individuals who were classified as good sleepers in infancy remained so in early childhood, whereas 69 % of those who were classified as poor sleepers in infancy remained so during early childhood, suggesting high continuity in sleep problems from 2 years of age to kindergarten. From early childhood to middle childhood, individuals who were classified as good sleepers in early childhood were likely to remain so in middle childhood (91 %), but only 26 % of individuals who were poor sleepers in early childhood remained so in middle childhood. Instead, individuals who were classified as poor sleepers in early childhood were most likely to transition to being good sleepers in middle childhood (74 %). Few individuals moved from being a good sleeper in early childhood to being a poor sleeper in middle childhood. Finally, most individuals who were good sleepers in middle childhood remained so in adolescence (69 %), however, there was a 31 % chance that individuals who were good sleepers in middle childhood would move into the poor sleeper class in adolescence. Of those who were poor sleepers in middle childhood, they were most likely to transition to the good sleeper class during adolescence (71 %), although 29 % remained in the poor sleeper class from middle childhood to adolescence. Across all time points, 478 were good sleepers at every developmental stage, 427 youth were good sleepers at all but 1 developmental stage, 211 youth were good sleepers at 2 developmental stages, 84 youth were good sleepers at only 1 developmental stage, and 11 youth were poor sleepers

Fig. 1 Latent classes of sleep groups

across development. Taken as a whole, the pattern suggests relative stability of good sleepers over time, but also that patterns of sleeping change dramatically across development, with many youth moving in and out of good sleep habits.

Across the model of sleep problems from infancy to adolescence a few general patterns emerge. First, at each developmental period, the majority of children are relatively good sleepers, exhibiting few sleep difficulties or disturbances. Second, there is generally strong stability in remaining a relatively good sleeper between developmental periods, but the continuity for being a poor sleeper across developmental periods was weaker. Indeed, across any developmental period, no more than 30 % of the sample exhibited continuity in poor sleeping, with most youth transitioning into better sleep patterns at the subsequent patterns. This suggests that for most children, periods of being a poor sleeper are short-lived and tied to a single developmental period. Third, examination of the mean levels of sleep disturbances and sleep problems among poor sleepers (see Fig. 1) is markedly higher during adolescence than in earlier developmental periods. While this may reflect differences in measurement (youth self-report are used to generate latent classes at age 15 while parent report is used in earlier periods), it may also reflect increased challenges with sleep during adolescence.

Because posterior probabilities of latent class memberships were excellent and including additional variables in statistical models that derive latent classes can derive different solutions, we classified individuals to their most likely latent class at each developmental period and used this variable as a categorical variable in subsequent models to test the association between sleep problems, working memory, and risk taking.

Fig. 2 Conceptual model of determining the association between sleep problems and outcome measures

Are Sleep Problems Across Development Related to Working Memory?

Having established the developmental pattern of sleep problems from infancy to adolescence, we next turned to an examination of how these sleep problems were related to working memory during adolescence (age 15). Figure 2 presents the basic conceptual model of these analyses. We tested a series of nested structural equation models to determine if there were direct paths from sleep problems at each developmental period (infancy, early childhood, middle childhood, and adolescence) to adolescent working memory. Thus, these models indicate the extent to which

problems with sleep in each developmental period forecast poor working memory during adolescence, above and beyond sex, race, and socioeconomic status.

Specifically, we compare a model where paths from sleep problems in each developmental period are associated with adolescent working memory, allowing for continuity in sleep problems across time and control variables (unrestricted model). In a series of restricted models, we constrain the paths from sleep problems at each developmental period to zero and evaluate the relative fit of the structural equation model. If the model fit does not significantly change when a parameter is constrained to zero, we reject the unrestricted model for the more parsimonious model where the pathways is constrained to zero. In the figure, we first tested if the path from infant sleep problems to working memory improved model fit (path A), then the path from early childhood sleep problems to working memory (path B), then the path from middle childhood sleep problems to working memory (path C), and the path from adolescent sleep problems to working memory (path D). The *difftest* option was used in Mplus to compare differences in model fit across the unrestricted and restricted models. The results indicated that the association between sleep problems in infancy [$\chi^2(1) = .85, p = .36$] and early childhood [$\chi^2(1) = 1.20, p = .27$] on adolescent working memory did not improve model fit and consequently, these pathways were excluded from the final model. In contrast, inclusions of the paths from sleep problems in middle childhood [$\chi^2(1) = 3.80, p = .05$] and adolescence [$\chi^2(1) = .85, p = .36$] to adolescent working memory did significantly improve model fit and were included in the final model. Notably, while the pathway from middle childhood to risk taking is not significant (see subsequent section), inclusion of the pathway estimation did improve the fit to the structure of the data. As such, it is included in the final model. Consequently, in a final model, we estimated cascade paths of sleep problems at each developmental period to sleep problems in the subsequent developmental period, and included direct effects from sleep problems in middle childhood and adolescence to adolescent working memory.

Race and sex were unrelated to differences in working memory at age 15. Lower socioeconomic status was associated with lower working memory at age 15. As found in other models, sleep problems in one developmental period were significantly related to sleep problems at the subsequent developmental period. While the path from middle childhood to working memory at age 15 was not significant, it did improve the overall model fit. Moreover, sleep problems in adolescence were significantly and negatively related to working memory at age 15, suggesting that individuals with sleep problems during adolescence display poorer working memory at age 15. The R^2 of

the model suggested that 3 % of the variance in working memory was explained by sleep problems.

Are Sleep Problems Across Development Related to Risk Taking?

Using the same approach to structural equation modeling established when testing the effects of sleep problems on working memory (see Fig. 2 and previous section on the association between sleep problems across development and working memory), we next tested how sleep problems in each developmental period were related to risk taking in late adolescence (age 18). We compared the model fit of an unrestricted model where late adolescent risk taking was regressed on infant, early childhood, middle childhood, and adolescent sleep problems to a series of restricted models that constrained each of these paths to zero (e.g., the path from infant sleep problems to late adolescent risk taking was constrained to zero; the path from early childhood sleep problems to late adolescent risk taking was constrained to zero; and so on) to test if inclusion of the pathway improved overall model fit. These models accounted for the continuity of sleep problems across developmental period and late adolescent risk taking was regressed on three covariates: sex, race, and socioeconomic status. At no developmental period did the inclusion of paths from sleep problems to late adolescence risk taking increase model fit: infancy [$\chi^2(1) = .03, p = .87$], early childhood [$\chi^2(1) = 1.02, p = .31$], middle childhood [$\chi^2(1) = 1.01, p = .31$], and adolescence [$\chi^2(1) = 1.65, p = .19$]. This suggests that there are no direct associations between sleep problems and late adolescent risk taking. However, we still find evidence of cascading effects of sleep problems across developmental time periods (e.g., sleep problems in one developmental stage are associated with greater chance of sleep problems at the subsequent developmental period). This finding is consistent with the previous model on working memory. Race was unrelated to risk taking at age 18. Males and youth from lower socioeconomic status were more likely to engage in risk taking. Seven percent of the variance in late adolescent risk taking was explained by these covariates.

Do the Effects of Sleep on Working Memory Cascade onto Late Adolescent Risk Taking?

Finally, we combined the previously derived measures of sleep problems on working memory and sleep problems on risk taking, adding an additional pathway between working memory problems and sleep problems. Specifically, we estimated the previously identified model of the associations between sleep and working memory (regressing working memory onto middle childhood sleep problems

Fig. 3 Association between sleep problems and working memory

and adolescent sleep problems) and sleep problems and risk taking (no regression paths were included between sleep problems at each developmental stage and risk taking). Risk taking at age 18 and working memory were regressed on race, sex, and socioeconomic status. Of key interest was a pathway from working memory at age 15 to risk taking at age 18. Comparing a model with this direct path from working memory to risk taking to a model without this path, we determined that including this parameter improved model fit [$\chi^2(1) = 3.61$, $p = .05$].

Figure 2 presents the standardized SEM paths between variables. In this final model, sleep problems in infancy were related to sleep problems in early childhood, early childhood sleep problems were related to middle childhood sleep problems, and middle childhood sleep problems were related to adolescent sleep problems. Youth with lower socioeconomic status performed worse on the working memory task. Males and youth from lower socioeconomic status reported higher risk taking at age 18. Middle childhood sleep problems were not significantly associated with adolescent working memory. Note that in the previous model, inclusion of this path improved model fit; however, the pathway was not significant. In contrast, adolescent sleep problems were associated with worse performance on working memory. In turn, these problems in working memory were related to higher risk taking 3 years later in late adolescence. Using the Wald test, we found that the pathway from working memory to risk taking at age 18 was significantly different than zero (Wald test: $df = 1$; Wald = 3.604, $p = .05$), indicating that poorer working memory at age 15 is associated with greater risk taking

3 years later. Approximately 8 % of the variance in late adolescent risk taking was explained (Fig. 3).

Discussion

It has long been observed anecdotally, as well as empirically, that sleep problems are associated with troublesome behavior among youth, as witnessed by many parents whose youngsters have missed a nap, had bad dreams, or stayed up too late and paid the consequences afterward through irritability, mood swings, acting-out behaviors, or rule-breaking. Prior research has demonstrated associations between sleep duration and delinquency (Clinkinbeard et al. 2011), insufficient sleep and greater incidence of a variety of health risk behaviors (McKnight-Eily et al. 2011), and sleep problems and risky behavior (O'Brien and Mindell 2005). Yet, it is less understood *how* sleep might be connected to risk. Our results suggest that sleep may indirectly influence risk through its effects on working memory. That is, the sleep-risk association is explained through a cascade model in which poor sleep during adolescence predicts working memory deficits, which in turn predict risk taking behavior 3 years later (Fig. 4).

Interestingly, we found a pattern of continuity in sleep problems across development. It is important to note, however, that at each developmental period the majority of children were relatively good sleepers, exhibiting few sleep difficulties. Basically, there was strong stability in remaining a relatively good sleeper between developmental periods. However, the continuity for being a poor sleeper was weaker although still present, suggesting that many youth experience temporary periods when he or she experiences sleep problems. Simply put, our findings show that good sleep (fewer sleep problems) tends to lead to more good sleep, but that some periods of sleep problems from childhood to adolescence are somewhat normative. Thus, if one is considered a good sleeper during infancy, it is likely that one will remain a good sleeper in middle childhood, and vice versa, which is consistent with previous research (Stein et al. 2001). The developmental cascade pattern of sleep problems that was found in the present study supports literature suggesting that the best predictor of sleep problems in late childhood is sleep problems beginning prior to age two (Stein et al. 2001). Notably, the least continuity in sleep problems across time is found during adolescence, when more individuals experience sleep issues. This is notable due to the numerous developmental changes in sleep patterns that occur during adolescence (Hagenauer et al. 2009).

In general, adolescence appears to be a time of vulnerability to sleep problems, marked by higher mean levels of sleep problems, greater frequency of transitions from being

Fig. 4 Association between sleep problems and risk taking

Model fit: $\chi^2 (7) = 28.82$ CFI = 0.91 TLI = 0.87 RMSEA = 0.05
 Path coefficients in bold significant at $p < .05$.

a good sleeper to a poor sleeper, and vulnerability to the consequences of sleep problems (as evinced by greater working memory deficits). Indeed, good sleep during adolescence may be especially crucial, as this was the only developmental stage where sleep was associated with poor working memory. In turn, these working memory problems are associated with greater risk taking 3 years later. As such, avoidance of sleep problems during adolescence should be a top priority for those who are concerned with behaviors associated with working memory, such as school performance, and ultimately the prevention of risk behavior. Moreover, results suggest that the effects of sleep on working memory may be one mechanism for how sleep is related to risk taking during adolescence (Fig. 5).

Surprisingly, the present analyses did not find a direct path from sleep problems at any age to adolescent risk taking. This is contradictory to work that has demonstrated the association between adolescent sleep problems and risk behavior (Catrett and Gaultney 2009; Clinkinbeard et al. 2011; O'Brien and Mindell 2005). However, prior work has focused largely on low sleep duration—rather than sleep problems—in predicting risk behavior (Clinkinbeard et al. 2011; McKnight-Eily et al. 2011). It is quite possible that low sleep duration is something qualitatively different than true sleep problems and this may explain the

difference in our findings. Given that we used a normative, non-clinical sample, our incidence of both sleep problems and risky behavior were fairly low; this may also have contributed to our non-significant finding.

One of the many strengths of this study was the use of a consistent measure of sleep problems across the entire range of development explored in this study, which allowed for longitudinal analyses. Although many studies have investigated sleep problems cross-sectionally or during specific developmental periods, such as infancy or early childhood, very few have examined sleep longitudinally across the comprehensive range of child development (from infancy to late adolescence) that was covered in the present study. Thus, this study is uniquely positioned to describe the patterns of sleep problems over time. Additionally, the large and geographically diverse sample used in this study enhances the generalizability of the study.

Nevertheless, the present study is limited in several respects. First, the greatest limitation in the present study is the measure of sleep problems. Sleep problems were assessed using items from the Child Behavior Checklist (CBCL), an assessment that does not have a standardized sleep scale. However, some research suggests the CBCL sleep items tend to correspond with other measures of sleep, such as sleep diary, actigraphy, and polysomnography; this

Model fit: $\chi^2(18) = 37.06$ CFI = 0.91 TLI = 0.85 RMSEA = 0.03
 Paths in bold significant at $p < .05$.

Fig. 5 Association between sleep problems, working memory, and risk taking behavior

was particularly true for the item “trouble sleeping” (Gregory et al. 2011). Although the sleep items used in the present study are not an established sleep scale, several studies have assessed sleep in a similar manner (Coulombe et al. 2010; Gregory and O’Connor 2002).

A second problem with the sleep assessment was its reliance on maternal- and self-report measures of sleep problems rather than physiological monitoring of sleep quality. Reporter methods have not tended to correlate strongly with biological measures of sleep quality. A study by Sadeh (1996) found that, although parents tended to be accurate in their reports of their children’s sleep schedules (e.g., onset of sleep and duration) as measured by significant and high correlations (ranging from $r = 0.74$ to $r = 0.88$) with actigraphy data, parents were less accurate at reporting their children’s sleep quality. Interestingly, Sadeh’s results showed that parents tend to underreport problems in their children’s sleep (e.g., night-wakings), as opposed to exaggerating such sleep difficulties. Although this research found some discrepancies between the subjective and objective measures, the data suggest that when parents *did* notice problems they tended to be supported by the actigraphy data. Therefore, in the present study, we feel confident that the sleep problems reported by parents are indeed occurring. Furthermore, as stated by Scher and

Asher (2004, p. 298) “definitions of sleep *problems* based on parental reports and sleep *quality*, measured objectively, target different constructs and should not be considered as merely different operational definitions.” Thus, both subjective and objective measures of children’s sleep tell distinct and critical parts of the story, so it is unnecessary for the two measures to correlate highly. In fact, it should be expected that they would diverge because they are used to measure different things. It will be important for future studies to address this issue of sleep problems versus sleep quality by using a combination of self or parent reporting techniques and physiological measures of sleep.

Another important limitation in the study is the lack of a consistent reporter of sleep problems across all ages. The study uses maternal report of children’s sleep problems from age 24 months through the sixth grade assessment, whereas youth report was used at the adolescent assessment when participants were 15 years of age. The present findings revealed inconsistency between maternal reports and youth self-reports of participants’ sleep problems. Therefore, we cannot say with certainty that the transition from “good sleeper” to “bad sleeper” status (and vice versa) that occurred at the age 15 assessment are due to actual changes in sleep problems or due to the change in reporter. Unfortunately, it is difficult to tease apart the cause of these

sleep status transitions during adolescence because it is the first time point in which self-reported sleep data were obtained from the youth. However, adolescents are capable of accurately self-reporting on a range of outcomes including depression (Moretti et al. 1985), physical health (Riley 2004), mental health (Patalay et al. 2014), and delinquent behavior (Thornberry and Krohn 2000). In fact, in a study comparing a variety of sleep assessments (7-day sleep diary, wrist actigraphy, adolescent-report, and parent-report), the findings revealed the importance of using adolescents' estimates of sleep problems during adolescence, as these tended to be more sensitive than parents' reports of youths' sleep problems at this age (Short et al. 2013). Therefore, we feel confident that the sleep problems reported by the youths at age 15 are reflective of true sleep problems and are not artifacts of the change in reporter.

Finally, the present study was correlational rather than experimental and did not perform any manipulations on sleep. However, due to the very nature of sleep problems, it is not possible to perform this study as a true experiment in the sense of assigning individuals to be good or bad sleepers across development. Although it would be interesting for future research to explore the effects of artificial manipulations of sleep (such as periodically waking members of a sleep study to mimic night-wakings), these studies would be inappropriate for addressing the types of long-term, developmental sleep issues focused on in the present study.

By examining the impact of sleep problems across development and exploring the functional consequences of poor sleep, we are better able to understand the full effect of sleep problems on youth development. Additional studies spanning multiple periods of development are needed to fully comprehend the long-term influence of sleep problems on development. Our findings suggest working memory appears to be particularly connected with sleep; therefore, examining additional components of executive functioning appears to be an important avenue for future research.

Conclusion

The present study highlights the importance of good sleep for the development of a strong working memory and the prevention of risk behavior. The present results suggest that sleep problems in earlier periods are indicative of risk for sleep problems later in development, but that sleep problems in adolescence contribute uniquely to deficits in working memory that, in turn, lead to risky behavior during late adolescence. Adolescents, due to normative developmental changes, are prone to impulsivity and reward

seeking, making them more predisposed to risk-taking than their child or adult counterparts (Steinberg 2008). Yet, those adolescents with poor working memory capacity may be especially susceptible to risky behavior. Therefore, the value of intervention to reduce or eliminate sleep problems cannot be understated. Although our transition analyses suggest that sleep problems during infancy are not uncommon, individuals who experience sleep problems during adolescence encounter functional consequences of this deprivation. Although promoting healthy sleep in each stage of development may be important, with respect to promoting positive adolescent outcomes, results indicate that adolescence may be a prime time in which interventions are likely to be most effective.

Our findings support the conclusions drawn by prior research that suggests adolescents are developmentally vulnerable to poor sleep. This vulnerability can be exacerbated by school schedules that are incompatible with youths' delayed phase preference. In practice, delayed school start times would be more conducive to adolescents' natural sleep cycle. Such policy changes would likely result in improved sleep performance among adolescents, as well as reduce the likelihood of working memory deficits and associated risk behavior. Additionally, the prime time for engagement in delinquent and risky behavior is in the hours immediately following school (between 3 and 6 p.m. on school days), as this time is commonly spent unsupervised (Fight Crime: Invest in Kids 2000). Thus, reframing school schedules to be compatible with youths' developmental sleep preferences would have the added benefit of reducing the amount of time youth spend unsupervised by pushing the time of school release later into the evening and closer to the time that parents get off work. Such a policy change has the potential to not only reduce sleep problems among teens, thereby improving working memory and reducing vulnerability to risky decision making, but also reduce opportunities for adolescent risk behavior.

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manuscript and offered expert advice on risk taking and adolescence. All authors read and approved the final manuscript.

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