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Childhood Sleep Problems and Young Adult Health Outcomes

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Childhood Sleep Problems and Young Adult Health Outcomes

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An abstract of
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Abstract

Childhood Sleep Problems and Young Adult Health Outcomes

By Brooke L. Reidy

Objective: Despite a growing body of literature demonstrating associations between sleep problems and diminished physical health, few studies have explored the prospective effects of childhood sleep problems on adult health. In particular, little is known about the impact of childhood sleep on health relative to established risk factors, such as youth depression and environmental stress. The current study investigated the prospective association between childhood sleep problems and young adult health outcomes, and looked to see whether this relationship held when accounting for known predictors of adult health problems. **Methods:** The current sample included 710 mother-youth dyads that participated in a study evaluating children's development from birth to age 20. Structural equation modeling (SEM) was employed to explore the potential effects of sleep on young adult health, and then to assess whether such associations remained when measures of youth stress and depression were individually entered into the overall model. **Results:** In the tested model, childhood sleep was a significant predictor of young adult health (β = .40, p <.001). Childhood sleep continued to predict to young adult health over and above the effects of early adversity, child health at age 5, youth and maternal depression through age 15, and chronic social stress at age 15, when these variables were entered into the model. Youth sleep and nonsocial chronic stress at age 15 independently predicted to young adult health. **Conclusions:** Results from the present analyses suggest that sleep problems in childhood and adolescence can have negative health consequences in young adulthood. Moreover, sleep problems in childhood and adolescence are still significant predictors of health outcomes when a variety of alternative predictor variables are controlled. Given this, sleep may be an important intervention target for child populations at risk for health conditions later in life.

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Sleep is as an active biological process that is fundamental to restoring and maintaining homeostatic balance. As such, sleep plays a critical role in health, wellness, and physical development (Okun, 2011). Studies in the animal and adult human literature suggest that sleep problems may serve as a physiological stressor to the body, and demonstrate that sleep deprivation can have acute and potentially long-term health effects (see Meerlo, Sgoifo, & Suchecki, 2008; Okun, 2011; McEwen, 2006). However, few studies have examined the prospective effects of sleep problems in childhood on adult health. Instead, much of the research predicting adult health outcomes has focused on associations with early adversity, chronic environmental stress, and depression, which are in turn associated with concurrent sleep problems (Felitti et al., 1998; Raposa, Hammen, Brennan, O'Callaghan, & Najman, 2013; Kiecolt-Glaser & Glaser, 2002; Sadeh et al., 1996; Hall et al., 2000). Identifying whether a direct association between childhood sleep and health exists beyond the effects of other risk factors could inform health-focused interventions. The current study will investigate the prospective effects of childhood sleep quality on young adult health, and will examine whether this association holds above and beyond other predictors established in the literature, such as early life stress.

Sleep as a Risk Factor for Adult Health Problems

Sleep problems in adolescence and adulthood have been implicated as a risk factor for several chronic health conditions (Zhang, Lama, Li, Li, & Wing, 2012; Altman et al., 2012; Lazaratou, Soldatoub, & Dikeos, 2012). Among adolescents, poor sleep has been associated with lower health-related quality of life, and linked prospectively to non-suicidal self-injury (Roeser, Eichholz, Schwerdtle, Schlarb, & Kübler, 2012; Lundh, Bjärehed, & Wångby-Lundh, 2012). Alterations in adult sleep duration have been

associated with increased risk for obesity, diabetes, hypertension, high blood pressure, and cardiovascular disease (Buxton & Marcelli, 2010; Magee, Kritharides, Attia, McElduff, & Banks, 2011; Altman et al., 2012). Animal research complements these findings, demonstrating that sleep restriction is associated with a variety of metabolic and immune changes, including alterations in glucose tolerance and metabolism, reduced leptin levels, and increased inflammatory marker levels (Banks & Dinges, 2007; Broussard, Ehrmann, Van Cauter, Tasali, & Brady, 2012; Knutson, Spiegel, Peneva, & Van Cauter, 2007; Van Cauter, Spiegel, Tasali, & Leproult, 2008; Okun et al., 2011; McEwen, 2006).

Few studies have explored the potential long-term health risk of sleep problems that begin early in childhood. One prospective study showed that sleep problems among children ages 3-5 years old predicted substance abuse in early adolescence (Wong, Brower, Fitzgerald, & Zucker, 2004). Sleep difficulties among children are also associated with increased risk for both childhood and adult obesity (Landhuis, Poulton, Welch & Hancox, 2008; Cappuccio, Taggart, Kandala, & Currie, 2008). A randomized-control melatonin treatment study found that among children with chronic sleep-onset insomnia, those that received melatonin treatment had concurrent improvements in sleep onset and general health status, suggesting that sleep may play a causal role in child health (Smits et al., 2003). These findings collectively demonstrate that across development, sleep problems may negatively impact individual's health, and suggest that sleep-related interventions can improve an individual's current and long-term health status. However, there is still little known about the long-term health consequences of sleep problems that occur in childhood.

The Effects of Environmental Stress and Depression on Adult Health

While the literature investigating sleep as a predictor of health outcomes is just emerging, a large body of research demonstrates that childhood adversity and chronic environmental stress can have profound and potentially long-term effects on human health. Adverse childhood experiences, such as low socioeconomic status (SES), family dysfunction, and various forms of maltreatment (e.g., physical, emotional, or sexual abuse, neglect), have been associated with increased risk for a variety of health problems in adulthood, including poor cardiovascular health, obesity, liver disease, substance abuse and suicide (Anda, Butchart, Felitti, & Brown, 2010; Felitti et al., 1998; Dube et al., 2001; Gunstad et al., 2006; Dong et al., 2004). Early adversity has also been associated with more frequent emergency room and physician visits, poorer health outcomes based on patient ratings, and increased risk for pain disorders, medical disability, and autoimmune disease-related hospitalizations (Chartier, Walker, & Naimark, 2007; Dube et al., 2009).

Findings in the human and animal literature have demonstrated that chronic stress is a risk factor for several health conditions, including obesity, metabolic syndrome, and cancer (De Vriendt, Moreno, & De Henauw, 2009; Chandola, Brunner, & Marmot, 2006; Thaker, Lutgendorf, & Sood, 2007). In fact, a growing body of literature suggests that chronic stress may be a mechanism by which early adversity affects long-term health outcomes (Raposa et al., 2013; Hazel, Hammen, Brennan, & Najman, 2008; Shonkoff, Boyce, & McEwen, 2009). Our research group recently revealed that chronic stress, as well as youth depression, mediated the relationship between early adversity and young adult health. Specifically, our group demonstrated that early adversity negatively

impacted young adult health indirectly through its effects on youth stress and depression at age 15 (Raposa et al., 2013).

Given these findings, it is perhaps not surprising that adult depression is associated with increased risk for a variety of chronic health conditions, including arthritis, diabetes, and coronary heart disease (Moussavi, Chatterji, Verdes, et al., 2007; De Groot, Anderson, Freedland, Clouse, & Lustman, 2001; Carney & Freedland, 2003; Katon & Ciechanowski, 2002). Similarly, youth depression is linked to increased risk for obesity, immune problems, and poorer health quality in adulthood, and has been positively associated with substance abuse and suicide later in life (Pine, Goldstein, Wolk, & Weissman, 2001; Caserta, Wyman, Wang, Moynihan, & O'Connor, 2011; Keenan-Miller, Hammen, & Brennan, 2007; McKowen, Tompson, Brown, & Asarnow, 2013; Weissman et al., 1999).

The Relationship Between Sleep and Environmental Stress

A growing body of animal and human research suggests a potential bidirectional relationship between sleep and stress. Animal studies have repeatedly linked stress and sleep, suggesting that various types of chronic stress (e.g. restraint, foot shock) can induce acute and long-term alterations in sleep quality and circadian rhythms (Papale, Andersen, Antunes, Alvarenga, & Tufik, 2005; Kant et al., 1995; Sanford, Yang, Wellman, Liu, & Tang, 2010; Hegde, Jayakrishnan, Chattarjib, Kuttya, & Laxima, 2011; Dugovic, Maccari, Weibel, Turek, & Van Reeth, 1999). Human studies have similarly shown that daytime and bedtime stress are associated with poorer sleep efficiency and increased slow wave sleep latency (Akerstedt, Kecklund, & Axelsson, 2007; Akerstedt et al., 2012). Likewise, individuals who experience more stressful events over the course of

their lives are at an increased risk for sleep problems; in turn, poor sleep is positively associated with experiences of current stressful events (Koskenvuo, Hublin, Partinen, Paunio, & Koskenvuo, 2010). Individuals with insomnia have also been shown to perceive life events as being more stressful, and report higher levels of arousal prior to falling asleep (Morin, Rodrigue, & Ivers, 2003).

The Relationship Between Sleep and Depression

An abundance of research has also suggested a bidirectional association between sleep problems and depression. Epidemiological studies have demonstrated that these two health conditions are highly co-morbid, with as many as 90% percent of depressed individuals reporting impaired sleep quality (Riemann, Berger, & Voderholzer, 2001; Tsuno, Besset, & Ritchie, 2005). Relative to non-depressed individuals, individuals with depression demonstrated alterations in REM sleep latency and overall sleep duration, as well as reduced slow wave sleep (for a review, see Tsuno, Besset, & Ritchie, 2005). Supporting a bidirectional association, research findings have also shown that individuals with insomnia are at increased risk for depression (Baglioni et al., 2011). However, studies examining youth sleep problems as a predictor of later depression have produced mixed results (Gregory et al., 2005, Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009).

The Current Study

Taken together, the above-mentioned findings implicate sleep as an important contributing factor influencing long-term health. Despite a growing body of research linking sleep to health, to date no published study has prospectively investigated the effects of childhood sleep on adult health or examined the relative contribution of sleep problems to poor health, as compared to other established risk factors like stress and

depression. To address this gap in the literature, the current study will investigate the potential long-term consequences of childhood sleep problems on health, accounting for the influence of early adversity, experiences of chronic environmental stress, and youth risk for depression. The current analyses will use longitudinal data collected from a cohort of over 700 families to test hypotheses that: 1) sleep problems in childhood and adolescence will predict adverse health outcomes in early adulthood, and 2) child and adolescent sleep will continue to predict young adult health problems after accounting for the effects of youth depression and youth exposure to stress.

Methods

Participants

Participants included 710 mother-youth dyads followed prospectively from birth to age 20. This sample was drawn from a larger birth cohort that comprised the Mater-University Study of Pregnancy (MUSP), which enrolled over 7,000 children born between 1981 and 1984 in Queensland, Australia, and followed them through age five (Keeping et al., 1989). A subset of these mother-youth dyads were recruited to participate in a follow-up study focused on the intergenerational transmission of depression when youth were ages 15 and 20. Recruitment for this "high-risk" youth sample was informed by maternal self-reported histories of depression measured from birth to youth age five, in that selected mothers represented a wide range of depressive symptom chronicity and severity (see Hammen & Brennan, 2001). Of the 991 mother-youth dyads targeted for continued study participation, 815 (82%) participated at age 15, and 710 (72%) participated and completed at least one health-related measure at age 20.

Mother-youth dyads in the current sample were primarily Caucasian (91.4%;

8.6% Asian, Aboriginal, or Pacific Islander) and lower to middle income; 49% of the youth in the age 20 sample were male. Participants in the current study did not differ significantly from the original MUSP cohort with respect to initial family income (t[6747]=.09, p=.93), number of siblings (t(df=6667)=.74, p=.46), and ethnicity (X^2[3, 7018]=7.21, p=.07). Differences in youth gender among the current sample and the MUSP cohort trended on significance, such that there was a greater proportion of females in the current sample (X^2(df=1, N=7223)=3.85, p=.05). Relative to the original MUSP cohort, mothers in the current sample had a significantly higher level of education (t(df=7164)=2.17, p=.03).

Procedures

Mothers were assessed four times during their child's first five years of life: once in pregnancy, once in the days following their child's birth, once 6 months after their child's birth, and once when their child was five years old. During each visit, mothers completed questionnaires related to their health and psychosocial experiences, as well as that of their child. When youths were 15 and 20 years of age, questionnaires and semi-structured interviews were administered to mothers and youths to assess youth psychopathology, physical health, and stress exposure, as well as maternal psychopathology. During all waves of data collection, written informed consent and assent were obtained and participants were compensated for their time. Study protocols for assessments at youth age 15 and 20 were approved by the University of California, Los Angeles (UCLA) Institutional Review Board, the Emory University Institutional Review Board, and the University of Queensland Ethics Committee.

Measures

Sleep. Early childhood sleep problems were measured via maternal report when youths were 5 years old, using a question assessing child sleep irregularity between ages 2-4, as well as an item on the Achenbach Child Behavior Checklist (CBCL) that asked about sleep difficulties in the past 6 months at age 5. Additional measures of sleep were collected at age 15 using mother's responses to four sleep-related items (e.g., trouble sleeping, sleeps less than other kids) on the Child Behavior Checklist (CBCL) for ages 6-18 and youth responses to the same four sleep-related items on the corresponding Youth Self-Report Scale (YSR) for ages 11-18. These checklists are each comprised of over 100 items that assess the presence of behavioral and emotional problems. Across ages, the CBCL and YSR forms demonstrate high reliability and internal consistency and have been widely used to assess internalizing and externalizing behavior problems in children and adolescents (Achenbach & Rescorla, 2001). In this sample the age 15 mother sleep scale had an alpha of .64 and the youth sleep scale had an alpha of .65. For all sleep measures, higher scores indicate higher levels of sleep disturbance.

Early Adversity. Consistent with our previous studies (Hazel et al., 2008; Raposa et al., 2013), early adversity was assessed using an aggregate measure of various life stressors that occurred during youth's first five years of life. Specifically, information about youth exposure to five indicators of early adversity - parental separation, relationship discord, maternal stress, family income, and maternal depressive symptoms - was collected via maternal report at four time points in early childhood. Total adversity severity scores were compiled using a count system, where each type of adversity endorsed was given a point. In order to create this cumulative score, continuous measures

of adversity were recoded as being present or absent using the 33rd percentile as a cut-off. However, due to the fact that few youths experienced all five types of adversity in early childhood, individuals that experienced four or more adversities were combined, so that the overall distribution of early adversity severity scores ranged from 0-4.

Youth Stress. Composite measures of chronic social and non-social stress exposure were created using severity scores coded from semi-structured interviews with youths at age 15. To assess chronic stress, the UCLA Life Stress Interview (Hammen, 1991) was used to probe and rate the severity of adolescents' ongoing exposure to stress in each of several social (e.g. romantic relationships, family relationships) and non-social (e.g. academic performance) domains of functioning over the past 6 months. Exposure severity was rated by the interviewer from 1 (exceptionally good conditions) to 5 (extreme adversity) using behaviorally-specific anchor points (Raposa et al., 2013). Intraclass correlations were computed for both social and non-social domains, and ranged from .55 (romantic relationship) to .94 (academic performance; Raposa et al., 2013). Chronic severity scores for social and non-social domains were averaged to generate composite social and non-social stress variables.

Youth Health. Early childhood measures of health were collected via maternal report when youths were five years old. Specifically, mothers responded to a single item related to youth health quality by rating their child's health from 1 (*Excellent Health*) to 4 (*Poor Health*). Health outcomes at age 20 were assessed using youth responses to health-related questions on the semi-structured UCLA Life Stress Interview. Youths were asked about their general health over the past 6 months and probed for information related to specific indicators of physical illness (e.g., being overweight, receiving medical

treatment). Responses to these questions were used to rate youths' overall quality of health on a 5-point scale, where higher scores indicated poorer health quality (interrater reliability = .77; see Keenan-Miller et al., 2007).

Health outcomes in young adulthood were also measured using both a general health item and the physical functioning subscale of the SF-36 Health Survey (Ware, Snow, & Kosinski, 2000). For the general health item, youth participants were asked to rate their current overall health quality from 1 (Excellent) to 5 (Poor). Likewise, for the physical functioning scale, participants were asked to complete 10 items regarding the extent to which individual's current health limited their ability to perform specific daily activities. Responses on this scale ranged from 1 (yes, limited a lot) to 3 (no, not limited at all). This scale is a widely used and well-validated measure of general health and health-related quality of life, and demonstrates predictive validity with respect to chronic medical illness severity (McHorney, Ware, & Raczek, 1993).

Youth and Maternal Mental Health. Presence or absence of lifetime youth depression was also measured at youth age 15 using the Kiddie Schedule for Affective Disorders and Schizophrenia, Epidemiological Version (KSADS-E; Orvaschel, 1995). Clinician interviews were conducted separately with mothers and youths regarding youths' current and lifetime history of mental health disorders, and independent ratings were then combined. Depression was coded as being present if youth met DSM-IV criteria for major depression, dysthymia, or depression not otherwise specified. A second clinician who was blind to youth diagnoses rated seventy-five randomly selected interviews to establish inter-rater reliability; weighted kappas for all ratings were greater than .70. Likewise, given that maternal depression has been positively associated with

youth depression and health problems (Hammen & Brennan, 2001; Turney, 2011), mother's lifetime diagnosis of major depressive disorder or dysthymia was also assessed at youth age 15 using the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1995). Reliability was assessed using data from 52 women in the study; for independent ratings of current and past major depressive disorder and dysthymia, all weighted kappas were above .80.

Data Analytic Plan

Several study variables of interest, namely those related to childhood sleep and young adult health, as well as early adversity, child health at age five, and chronic stress at age 15, showed significant skew and were log transformed for subsequent analyses.

Structural equation modeling (SEM) was used to assess childhood sleep in association with young adult health. Due to significant intercorrelations among dependent measures (see Table 1), a latent factor was used to measure youth health at age 20, the primary outcome in the study. Likewise, a latent factor for youth sleep in childhood was created using three significantly intercorrelated measures of sleep in early childhood and adolescence (see Table 2). All competing factors tested in relation to Hypothesis 2 were significantly associated with the independent and dependent variables using models demonstrating adequate fit, and were entered individually as separate predictor variables in the model.

SEM analyses were computed using AMOS 20.0 software. Model fit was evaluated using a combination of absolute and incremental fit indices, including the likelihood-ratio chi-square (χ^2) index, the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA). In the current analyses, a non-significant

χ^2 indicated non-discrepant fit between the population covariance and the covariance predicted by the tested model (Kline, 2011). However, this statistic can be overly sensitive to sample size at large values of *N* (Kline, 2011). Given the large size of the current sample, a significant χ^2 index did not preclude adequate model fit. The CFI assesses the improved fit of the tested model relative to an independence (null) model, and ranges in value from 0 – 1. Values over .90 indicated adequate model fit and over .95 indicated good fit (Hu & Bentler, 1999). Likewise, for the RMSEA, which measures the relative 'poorness' of tested model fit, values under .06 indicated good fit (Hu & Bentler, 1999; Kline, 2011).

Results

Sleep and Young Adult Health

Preschool parent questionnaire items, maternal CBCL and YSR sleep item scores from youth age 15 were all significantly correlated; thus, a latent construct for childhood sleep problems comprised of these indices was entered as the primary predictor in the overall SEM model (see Figure 1). The subsequent model tested had a significant demonstrated χ^2 statistic, but otherwise demonstrated good fit (χ^2 [8, 710] = 18.10, p=.021; CFI =.96; RMSEA = .04, 90% CI: .02-.07). Supporting our first hypothesis, childhood sleep problems significantly predicted health problems at youth age 20 (β = .40, p <.001).

Stress and Depression

Youth experiences of early adversity and chronic stress, youth health at age 5, lifetime presence of youth depression at age 15, and maternal lifetime presence of depression at youth age 15, were each entered separately into the overall SEM model

examining the association between childhood sleep and adult health. Resulting models all demonstrated adequate fit (see Table 3). Supporting hypothesis two, childhood sleep problems continued to significantly predict to young adult health problems ($p<.001$), over and above the effects of early adversity, child health at age 5, youth and maternal depression through age 15 as well as youth chronic social stress at 15. Of note, only chronic non-social stress at age 15 independently predicted adult health when childhood sleep was included in the model (see Table 4).

Discussion

In one of the first studies to prospectively investigate the relationship between childhood sleep and long-term health, SEM analyses revealed that sleep problems in childhood and adolescence significantly predict negative health outcomes in young adulthood. These results build upon a growing body of literature demonstrating a relationship between sleep and physical health, and suggest that higher levels of reported sleep problems throughout childhood are associated with poorer overall health and increased levels of health-related functional impairment at age 20.

Furthermore, the present analyses demonstrated that when the effects of several factors known to influence adult health, including youth depression, early adversity and chronic adolescent stress were included in the overall model, childhood sleep remained a significant predictor of young adult health outcomes. Even when chronic non-social stress was also found to significantly predict to young adult health, the standardized beta weights for chronic non-social stress and childhood sleep were comparable (both approximately .32). These findings indicate that the health risk associated with poor sleep in childhood cannot be attributed solely to the effects of stress or depression. Given the

limitations related to sleep measurement in the current study – namely that sleep measures were derived from a limited set of parent/self-report items, and that youth sleep was measured at two time points – these findings are quite robust, and suggest that sleep problems in childhood should be given more consideration as an independent health risk factor.

This study extends recent investigations of the association between sleep and later health problems, particularly through the use of structural equation modeling. SEM allowed for a multi-measure assessment approach by utilizing latent factors to measure the predictor and outcome variables. Although this was an observational study, the use of SEM and the temporal relationships of the included constructs enabled the testing of predictions about the effects of childhood sleep on health outcomes.

The present results also complement a growing body of research investigating sleep's impact on human cognition and development. Several studies suggest that sleep problems and/or deprivation in childhood and adolescence can negatively impact individual's attention, memory, and general neurobehavioral functioning (O'Callahan et al., 2010; Kopasz et al., 2010; Sadeh, Gruber & Raviv, 2002). Recent work has also linked sleep problems with increased risk for dementia and Alzheimer's disease (see Camargos, Louzada, & Nobrega, 2013). Taken together with the current findings, this work suggests that sleep may not only have widespread health effects, but may also have lasting impacts on human brain maturation and aging. Given this, sleep may be a useful, if not critical, intervention target for children at risk for both physical health problems and neurocognitive deficits.

The current study did not address potential mechanisms underlying the association between childhood sleep problems and adult health outcomes. However, several biological factors may be involved in this relationship. For example, sleep problems may affect individual health through its modulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis (Van Cauter et al., 2008; Buckley & Schatzburg, 2005). Sleep deprivation has been shown to lead to increased basal cortisol levels and increased stress reactivity (Grandner, Patel, Gehrman, Perlis, & Pack, 2010), and when chronic, can have cumulative effects that can alter individuals' neurobiological stress response (Meerlo et al., 2008). Likewise, sleep problems may influence health through its effects on inflammation, as sleep restriction has been related to variety of metabolic and immune changes, including alterations in glucose tolerance and metabolism, reduced leptin levels, and increased inflammatory marker levels (Banks & Dinges, 2007; Broussard et al., 2012; Knutson et al., 2007; Van Cauter et al., 2008; Okun, 2011). Future studies should explore the potential mediating role of such factors in explorations of childhood sleep and health.

As indicated above, this study has several limitations. First, data for the current analyses were collected from a sample of youth at high genetic risk for developing depression. The increased vulnerability of the youth in this sample to depression and early life stress provides strong evidence that sleep can influence long-term health independently of these known risk factors. However, the generalizability of the present findings is somewhat limited. Future replications of this study should examine the relationship between childhood sleep and long-term health in unselected populations of children.

Additionally, all measures for sleep and health were collected via maternal and/or self-report. Moderate associations between self-report measures and objective (i.e., polysomnography, actigraphy) measures of sleep have been documented (Carskadon et al., 1976; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; Wolfson et al., 2003; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008); however, studies have shown that individuals tend to overestimate sleep latency and underestimate sleep disturbance relative to objective measures (Carskadon et al., 1976; Baker, Maloney, & Driver, 1999). Thus, objective measures of sleep latency, duration and efficiency should be included in future studies to confirm these findings and identify specific aspects of sleep that influence health outcomes.

Self-report measures of health and biological indicators of health status have also been shown to be significantly positively correlated (McAdams, Dam, & Hu, 2007; Okura et al., 2004). In fact, some studies have shown that self-report measures of health are predictive of future health problems (Idler & Angel, 1990; Ferraro & Su, 2000). However, more objective measures of health status (e.g., assessment of exercise capacity (peak VO2), cholesterol, body mass index, blood pressure, etc.) would facilitate a more comprehensive investigation of sleep's effects on health, as well as potential underlying mechanisms.

Finally, while this was one of the first studies to assess sleep longitudinally, sleep disturbance was only assessed at two time points (preschool period and youth age 15) in childhood. Future projects should incorporate concurrent measures of adult sleep and health risk behaviors associated with chronic stress and/or sleep problems to examine their potential roles in the relationship between childhood sleep problems and adult health.

Additionally, future prospective studies should have more frequent and consistent measures of sleep across childhood and adolescence, as a means of investigating the developmental effects of sleep problems on health.

While not included in the present analyses, sleep measures were collected during infancy via maternal report, where mothers were asked to report their child's level of "sleeplessness" at six months of age. Whereas our early childhood and adolescent sleep measures were significantly correlated with one another (suggesting persistent sleep problems across development), our infant sleep measure was not correlated with later maternal reports of youth sleep problems. This discrepancy suggests that stable sleep problems may not materialize until the preschool period, but also supports the notion that pervasive, chronic sleep problems can arise in early childhood. Specifically, these findings implicate the preschool years as a potential time-point for early sleep intervention, and more broadly, highlight the importance of measuring sleep at various time points across development as a means of understanding when and how childhood sleep problems manifest, are maintained, and affect health. Given this study's potential health implications, future studies of sleep and its prospective health effects should incorporate measures of sleep and health at several points in early and middle childhood, as well as adolescence and early adulthood, to investigate specific patterns and developmental trajectories of sleep problems associated with various adverse adult health outcomes.

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Appendix

Table 1.
Correlation Matrix of Health Variables, Youth Age 20

	1	*2*	*3*
UCLA Chronic Stress Health	-		
2. SF-36 Gen. Health	.332**	-	
3. SF-36 Physical Functioning	.227**	.224**	-

** p < .01. *Notes. All variables were log transformed. Given that responses for the SF-36 Physical Functioning subscale were in the opposite direction of other health outcome measures, this variable was recoded for consistency in direction prior to running SEM models.*

Table 2.
Correlation Matrix of Youth Sleep Variables

	1	*2*	*3*
1. Irregular Sleep 2-4 yrs.	-		
2. YSR Sleep Questions, 15 yrs.	.126**	-	
3. MCBCL Sleep Questions, 15 yrs	.222**	.242**	-

** p < .01. *Notes. All variables were log transformed.*

Table 3.
SEM Model Fit Statistics: Overall Model with Individually Entered Competing Factors

Competing Factor	*Chi Square*	*CFI*	*RMSEA*
Early Adversity	X^2 (d.f=12, N=710) = 27.972, p=.006	.94	.043
Child Health, Age 5	X^2 (d.f=12, N=710) = 27.489, p=.007	.94	.043
Maternal Depression	X^2 (d.f=12, N=710) = 18.835, p=.093	.97	.028
Youth Depression, Age 15	X^2 (d.f=12, N=710) = 24.930, p=.015	.95	.038
Youth Social Stress, Age 15	X^2 (d.f=12, N=710) = 19.217, p=.083	.97	.029
Youth Non-Social Stress, Age 15	X^2 (d.f=12, N=710) = 24.448, p=.018	1.00	.038

Table 4.
Influence of Competing Factors on Young Adult Health in Tested Model

Competing Factor	β (*SE*)	*p*
Early Adversity	0.06	0.303
Child Health, Age 5	0.09	0.118
Maternal Depression	0.03	0.600
Youth Depression, Age 15	0.04	0.468
Youth Social Stress, Age 15	0.05	0.452
Youth Non-Social Stress, Age 15	**0.32**	< **.001**

Notes. In addition to the latent factor of childhood sleep, the overall model included the above variables, tested individually, as separate predictors of youth health at age 20. Childhood sleep remained a significant predictor of young adult health in all tests (β: .32 -.40, *p*<.001).

Figure 1: The Effects of Childhood Sleep on Young Adult Health