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Prospective associations between chronic youth sleep problems and young adult health

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ABSTRACT

Objectives: The current study investigated prospective associations between youth sleep problems across childhood and adolescence, as well as the relationship between chronic youth sleep problems and young adult health. Exploratory analyses investigated this sleep-health relationship in the context of several established risk factors, including youth depression and environmental stress.
Design: This project is an extension of the Mater-University Study of Pregnancy, a longitudinal study that followed more than 7000 children across early development.
Setting: Brisbane, Australia.
Participants: Seven hundred ten mother-child dyads assessed from birth to age 20.
Measurements: We used maternal report measures to assess the persistence of youth sleep problems. We used structural equation modeling to explore the relationship between chronic maternal-reported youth sleep problems and subjective reports of young adult health quality and to assess whether associations remained when other potential health risks were included in the model.
Results: Path analyses revealed that sleep problems in early childhood predicted sleep problems in middle adolescence, which predicted sleep problems at age 20. Structural equation models showed that chronic youth sleep problems predicted youth health quality at age 20 ($\beta = .263$, $P < .001$) over and above the effects of early adversity, chronic childhood illness, maternal depression, lifetime youth depression, and chronic youth stress.
Conclusions: Chronic sleep problems can emerge in childhood and may contribute to negative health outcomes in young adulthood. Chronic youth sleep problems remain a significant predictor of poor health when tested against other known health risk factors, suggesting that sleep may be an important health intervention target.

Sleep and health are undeniably linked. An extensive body of literature has demonstrated that sleep is critically involved in promoting physical growth, tissue restoration, and homeostatic balance.[1] In addition, patterns of disrupted sleep and patterns of short or long sleep duration have been associated with the presence of several chronic health conditions, including chronic pain,[2] obesity,[3] and cardiovascular disease,[3,4] as well as lower self-reported health-related quality of life[5,6] and greater health-related functional impairment.[7] Despite this, few studies have examined the longitudinal relationships of sleep disturbance over time or the prospective associations between sleep disturbance and health across childhood and adolescence. Given the extensive cognitive, emotional, and physical development that occurs in childhood and adolescence, examining these relationships may provide information about the role of sleep in health that could inform future health-related prevention programs.

A growing body of research has revealed that sleep patterns change drastically across childhood and adolescence and are generally marked by a decrease in overall sleep duration, napping, and night waking.[8,9] Although studies have shown that youth sleep varies considerably between individuals, research indicates that many children show stability in their sleep duration trends from infancy through age 10 years.[10] Investigations of the relative continuity of sleep problems in clinical pediatric populations have shown that although the

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majority of children do not have continued sleep difficulties, between 5% and 20% of children have persistent parent-reported sleep difficulties over the course of several (ie, 6-12) months.[11,12] Collectively, this work suggests that for a subset of individuals, sleep problems emerging in childhood may become persistent, or chronic, in nature; however, to date, no studies have investigated the chronicity of sleep problems from childhood into adolescence.

Prospective studies examining sleep disturbance in childhood and its association with later health outcomes are also rare. Building on cross-sectional studies linking sleep duration and body mass index, one recent study demonstrated that short sleep duration among children is associated with increased risk for adult obesity.[13] 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 youth health.[14] Taken together, these findings demonstrate the potential long-term impact of sleep disruption across development. However, researchers must extend the current literature to fully understand the relationship between sleep problems, particularly those that persist throughout childhood and adolescence, and later health outcomes.

The current study attempts to fill these gaps in the literature by examining the chronicity of sleep problems in childhood and adolescence, as well as the relationship between chronic youth sleep problems and health quality in young adulthood. Given that the longitudinal study from which the data were derived was not designed to examine sleep as a primary focus, our sleep problem measures consist only of subjective maternal reports. As such, this study represents a preliminary investigation of the sleep-health relationship in childhood and adolescence that will potentially serve as a platform for future investigations in this line of research.

The current study uses longitudinal data collected from a cohort of more than 700 families over 2 decades to test hypotheses that: (1) youth sleep problems in the preschool period will be associated with youth sleep problems in middle adolescence, which in turn will be associated with sleep problems in early adulthood; and (2) chronic youth sleep problems will predict subjective reports of health quality in early adulthood. Exploratory analyses will also investigate whether the relationship between chronic youth sleep problems and young adult health remains after accounting for the effects of psychosocial factors that have been shown to significantly contribute to adult health outcomes, including early adversity,[15] experiences of chronic environmental stress,[16,17] and youth depression.[18]

Participants and methods

Seven hundred ten mother-youth dyads participated in the current prospective study from birth to youth age 20. This sample was part of a larger birth cohort that comprised the Mater-University Study of Pregnancy (MUSP), which enrolled more than 7000 children born between 1981 and 1984 in Queensland, Australia, and followed them through age 5.[19] We recruited this subset of mother-youth dyads to participate in a follow-up study focused on the intergenerational transmission of depression when youths were ages 15 and 20. Specifically, we identified this at-risk youth sample using maternal self-reported histories of depression measured from birth to youth age 5 and selected mothers who represented a wide range of depressive symptom chronicity and severity. Of the 991 mother-youth dyads targeted for continued study participation, 815 (82.2%) participated at age 15, and 710 (71.6%) participated and completed at least 1 health-related measure at age 20.

Mother-youth dyads in the current sample were primarily white (91.5%; 8.5% Asian, Aboriginal, or Pacific Islander) and lower to middle income; 48.5% of the youths 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 ($t[6747] = .089$, $P = .93$), number of siblings ($t[df = 6667] = .741$, $P = .46$), and ethnicity ($\chi^2[3, 7018] = 7.211$, $P = .07$). Differences in youth sex among the current sample and the MUSP cohort approached significance, such that there was a greater proportion of females in the current sample ($\chi^2[df = 1, N = 7223] = 3.848$, $P = .05$). Relative to the original MUSP cohort, mothers in the current sample had a significantly higher level of education ($t[df = 7164] = 2.171$, $P = .03$).

Procedures

We assessed mothers 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 5 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, we administered questionnaires and semi-structured interviews assessing mental and physical health to each dyad. During all waves of data collection, participants provided written informed consent and assent and were compensated for their time. The University of Queensland Ethics Committee approved the study protocols for all waves of data collection; the University of California, Los Angeles (UCLA) and Emory University Institutional Review Boards additionally approved assessments at youth age 15 and 20.

Measures

Sleep

Mothers reported on their child's sleep problems at youth age 5, 15, and 20. When youths were 5 years old, mothers reported the extent to which their child's sleep was irregular between ages 2 and 4 years. Responses ranged from 1 (never) to 4 (often). At ages 15 and 20, mothers responded to 4 sleep-related items (eg, does your child have nightmares, trouble sleeping, sleep less than other children) on the Child Behavior Checklist (CBCL), used for ages 6-18, and 2 sleep-related items (ie, does your child feel tired without good reason, have trouble sleeping) on the Adult Behavior Checklist (ABCL), used for ages 18-59. Item responses ranged from 0 (not true) to 2 (very/often true), where higher scores indicated higher levels of sleep disturbance. As such, combined item responses on the CBCL ranged from 0 to 8; responses ranged from 0 to 4 on the ABCL. Combined item scores for both the CBCL and ABCL were highly skewed and were log transformed for subsequent analyses (see "Data analytic plan"). Across ages, the CBCL and ABCL demonstrate high reliability and good internal consistency.[20,21] In the current sample, maternal-reported sleep problems were significantly associated with youth-reported sleep problems on CBCL ($r = .257$, $P < .001$) and ABCL ($r = .325$, $P < .001$).

Youth health

Mothers reported the number of chronic illnesses their child had at youth age 5. Responses ranged from 0 to 4. At age 20, youths responded to health-related questions on the semi-structured UCLA Life Stress Interview. Specifically, interviewers asked youths about their general health over the past 6 months and probed for information related to specific indicators of physical illness (eg, being overweight, receiving medical treatment). We used these responses to rate youths' overall quality of health on a 5-point scale, where higher scores indicated poorer health quality. This measure demonstrated good interrater reliability (intraclass correlation coefficient = .77) and was moderately correlated ($r = .41$, $P < .001$) with youth-reported health at age 20.

Youths also completed the Short Form–36 (SF-36) Health Survey[22] at age 20. A general health item reflected youths' rating of their current overall health quality from 1 (excellent) to 5 (poor). The physical functioning scale reflected youths' rating of the extent to which their current health limited their ability to perform 10 daily activities (eg, lifting or carrying groceries, climbing a flight of stairs, walking several blocks, bathing, and dressing). Scores ranged from 1 (yes, limited a lot) to 3 (not limited at all); lower scores indicated more health-related functional disability. The SF-36 is a widely used and well-validated measure and demonstrates predictive validity with respect to chronic medical illness severity.[23]

Early adversity

Consistent with our previous studies,[24,25] we assessed early adversity using an aggregate measure of various life stressors that occurred during youths' first 5 years of life (ie, relationship discord, parental separation, maternal stress, and family income). Mothers reported on these early life stressors at one or more time points in pregnancy, at birth, 6 months postpartum, and at youth age 5. We compiled total adversity severity scores for each subject by summing the number of adversities endorsed. To ensure that this measure captured sufficient variance in levels of adversity, we recoded continuous measures of each type of adversity as being present or absent using the 33rd percentile as a cutoff.

Youth stress

Researchers interviewed youths at age 20 using the UCLA Life Stress Interview[26] as a means of probing and rating the severity of youths' ongoing exposure to stress in each of several social (ie, close friendships, romantic relationships, family relationships, social life) and nonsocial (ie, academic performance, work life, finances) domains of functioning over the past 6 months. Independent teams rated exposure severity from 1 (exceptionally good conditions) to 5 (extreme adversity) using behavioral anchor points.[25] Interrater reliabilities across chronic stress domains ranged from .76 to .82 for age 20 team ratings. We used mean ratings of youth social and nonsocial stress to create composite variables for these measures.

Youth and maternal depression

Using the Structured Clinical Interview for DSM-IV[27], we assessed for the presence or absence of lifetime youth depression at youth age 20. We also assessed mothers, lifetime diagnosis of major depressive disorder or dysthymia through youth age 15 using the Structured Clinical Interview for DSM-IV.

Data analytic plan

We used structural equation modeling (SEM) to test study hypotheses using AMOS 20.0 software. SEM is a statistical modeling technique that facilitates hypothesis testing at the construct level. In SEM, constructs are referred to as latent variables and are derived from multiple measured variables by extracting the common variance among them.[28] In the current study, we planned to include latent factors for chronic youth sleep problems and youth health quality at age 20 in the overall SEM model of interest.

We determined model fit using a combination of absolute and incremental fit indices, including the likelihood-ratio χ^2 index, the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA). In the current analyses, a nonsignificant χ^2 indicated nondiscrepant fit between the population covariance and the covariance predicted by the tested model.[28] However, this statistic can be overly sensitive to sample size at large values of N.[28] 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 to 1. Values greater than .90 indicated adequate model fit, and those greater than .95 indicated good fit.[29] Likewise, for the RMSEA, which measures the relative "poorness" of tested model fit, values less than .08 indicated adequate model fit, and those less than .06 indicated good fit.[29]

Maternal reports of early adversity, youth health at age 5, and youth sleep in childhood, adolescence, and early adulthood showed significant skew, as did youth reports of health at age 20. Therefore, we log transformed these variables before entering them in our tested models. In addition, because the physical functioning scale of the SF-36 Health Survey was scored in the opposite direction of other age 20 health measures, we recoded this variable for consistency in direction before running our SEM models.

Results

Table 1 lists the descriptive statistics for youth sleep and young adult health measures included in the present analyses. Intercorrelations among most variables are reported below.

Demographics factors including youth sex, youth ethnicity, parity, family income at study entry, parent education, and maternal age at child's birth were assessed as potential confounds in subsequent SEM models. Child sex was the only potential confound significantly associated with both latent variables included in the tested SEM model (ie, chronic youth sleep problems, young adult health quality; Table 2); sex was, therefore, controlled for in subsequent SEM analyses.

Associations among childhood and adolescent sleep problems

Sleep items collected at youth age 5 were significantly correlated with sleep measures at youth age 15 ($r = .222$) and 20 ($r = .146$). Maternal-reported youth sleep at age 15 and age 20 was also positively correlated ($r = .435$). In addition, in support of our first hypothesis, path analyses demonstrated that sleep problems in the preschool period significantly predicted sleep problems at age 15 ($\beta = .224$, $P < .001$), which in turn significantly predicted sleep problems at age 20 ($\beta = .434$, $P < .001$).

Chronic youth sleep problems and young adult health

Given the noted prospective associations among sleep in childhood and adolescence, we derived a latent factor for chronic youth sleep using measures of sleep in early childhood, adolescence, and young adulthood. To confirm that this latent factor reflected chronic sleep problems, we created a count of the number of assessment periods that the subject scored higher than the median on sleep problems and assessed the relationship of this count variable to sleep latent factor scores. A 1-way analysis of variance revealed that individuals' latent sleep scores varied significantly as a function of their sleep problem count scores ($F_{3,706} = 551.619$, $P < .001$). Post-hoc Duncan analyses demonstrated that each successive increase in the number of assessment periods where sleep problems were present was associated with a significant increase in the mean score on the sleep latent factor. Furthermore, we confirmed that the effect size for these calculations was greater than that of 1-way analyses of variance computed for each of our measured sleep variables, suggesting that our latent factor of chronic sleep problems was in fact more reflective of differences in participants' sleep problems count scores than individual measures of sleep disturbance.

Youth age 20 measures of health on the SF-36 scale were significantly correlated with one another ($r = .224$); these were also significantly intercorrelated with youth responses to health-related questions on the UCLA Chronic Stress Interview ($r = .332$ [SF-36

General Health], $r = .227$ [SF-36 Physical Functioning]). Therefore, we created a latent factor of subjective youth health quality as the primary outcome measure in the overall model of interest.

The resulting predictive model, which included both latent measures (Fig. 1), had good fit (χ^2 [12, 710] = 21.139, $P = .05$; CFI = .971; RMSEA = .033; 90% confidence interval [CI]: .003-.055) and, in support of our second hypothesis, revealed that chronic youth sleep problems significantly predicted health problems at youth age 20 ($\beta = .263$, $P < .001$). Inspection of modification indices indicated that independent measures of child sleep at specific periods did not add significant variance above and beyond chronic sleep problems in the prediction of young adult health problems (all χ^2 values < 1.50). Supporting our third hypothesis, youth sleep problems continued to significantly predict young adult health problems over and above the effects of all individually entered competing factors of interest (Table 3). Resulting models all demonstrated adequate fit (Table 4). Of note, youth depression, chronic social stress, and chronic nonsocial stress at age 20 independently predicted young adult health quality when chronic youth sleep problems were included in the tested model.

Discussion

In one of the first studies to prospectively investigate associations between sleep and health across childhood and adolescence, maternal-reported youth sleep problems in early childhood predicted sleep problems in middle adolescence, which in turn predicted sleep problems in young adulthood. These findings build on previous research demonstrating the continuity of sleep duration patterns throughout childhood and suggest that the persistence of sleep problems may emerge as early as the preschool period. Future research should expand upon this initial work to identify risk factors for developmental trajectories marked by chronic or persistent sleep difficulties.

Furthermore, in the current study, chronic youth sleep problems predicted poorer subjective health quality in young adulthood, even when controlling for effects of established psychosocial risk factors for poor health, such as early adversity, chronic stress, and depression. These results build upon a growing body of adult literature demonstrating associations between sleep disturbance and impaired health and, importantly, suggest that youth sleep problems should be given more consideration as an independent health risk factor. These findings also indicate that early interventions for children with persistent sleep problems could have broad public health implications.[14,30]

The current study serves as an important first step in understanding the prospective associations between sleep problems and health in childhood and adolescence. However, this study is not without its limitations, and future projects should extend this work in several potential directions. First, data for the current analyses were collected from a sample of youths at increased risk for developing depression. Although the increased vulnerability of the youths in this sample to depression and early life stress provides strong evidence that sleep can influence long-term health above and beyond these known risk factors, the current findings may be somewhat limited in terms of their generalizability. In our sample, maternal depression status did not moderate the relationship between youth sleep and young adult health; however, future studies should also explore associations between youth sleep and health in nonclinical, non–high-risk, community samples. The current study was also limited in terms of its measures of sleep and health, as both were assessed solely through interviews and/or subjective report. In regard to our sleep measures, the use of maternal reports was advantageous because it decreased shared method variance (ie, youths were not reporting on both their sleep and health problems). Maternal report measures were also significantly associated with youth-reported sleep problems at age 15 and 20, and both maternal- and youth-reported sleep problems predicted health quality at age 20. Recent studies have supported the use of subjective health measures, showing that they are significantly related to biological indicators of health status[31,32] and are predictive of future health problems.[33] One study also demonstrated that maternal reported child sleep duration was significantly associated with actigraphy measures of total sleep time.[34] Despite this, more objective measures of sleep latency, duration, and efficiency should be included in future studies to confirm the current findings and to identify specific aspects of sleep that influence health. Likewise, future studies should incorporate biological assays of health status (eg, exercise capacity [peak Vo_2], cholesterol, body mass index, blood pressure, proinflammatory markers) to improve validity and explore potential mechanisms explaining sleep-health associations.

Despite revealing statistical associations between chronic youth sleep problems and health quality, the current study did not test a theoretical model examining mechanisms underlying the potential

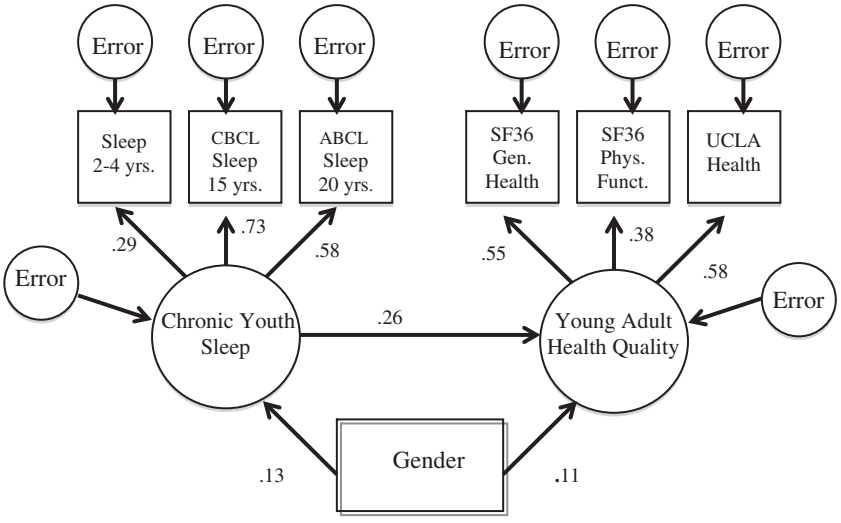

Fig. 1. Overall estimated SEM model[a] investigating prospective relationships among chronic youth sleep and young adult health quality. [a]Latent variables are depicted using circles, whereas measured variables are depicted using square or rectangular boxes.

cumulative health effects of chronic sleep problems in childhood and adolescence. Several biological factors are implicated as potential mediators in the relationship between sleep and health. For example, sleep disturbance may impact health through its modulation of the hypothalamic-pituitary-adrenal axis. Sleep deprivation has been shown to lead to increased basal cortisol levels and increased stress reactivity[35] and, when chronic, can have cumulative effects that can alter individuals' neurobiological stress response.[36] Likewise, sleep disturbance is associated with a variety of metabolic and immune changes and may influence health through its effects on inflammation.[37] Future studies should build upon our initial findings by investigating the mediating role of these factors in the relationship between youth sleep and health status.

In addition, future longitudinal studies should assess bidirectional relationships among sleep, depression, stress, and health. The existing literature supports a bidirectional relationship between sleep disruption and risk factors like stress[38,39] and depression.[40] Repeated assessments of each of these constructs across development would allow researchers to use cross-lagged analyses to identify the best intervention targets and time points in this developmental risk process, which is likely highly transactional in nature. Inclusion of more frequent and evenly spaced measures of sleep disturbance across childhood and adolescence would also facilitate analysis of sleep trajectories across development and their relative association with health status later in life.

Conclusions

Sleep is as an active biological process fundamental to the promotion of health, wellness, and physical development. Although its importance may often be overshadowed by other risk factors, the current study not only demonstrates the potential for youth sleep problems to persist through childhood but also highlights the potential long-term health effects of chronic youth sleep problems. If replicated, these findings suggest that pediatric providers should actively address sleep problems when treating youths.

Disclosure

The National Health and Medical Research Council; the Mater Misericordiae Mother's Hospital in Queensland, Australia; and the National Institute of Mental Health (MH52239). One author is also supported by a NARSAD Young Investigator Award from the Brain and Behavioral Research Fund. No authors have conflicts of interest to report.

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Appendix

Table 1
Descriptive statistics for sleep and health variables of interest.

Variable	Mean	SD
Irregular sleep 2-4 y	2.11	1.04
CBCL Sleep Questions, 15 y	.820	1.23
ABCL Sleep Questions, 20 y	1.04	1.03
UCLA Chronic Stress Health Score	2.31	.57
SF-36 General Health Item	2.59	.90
SF-36 Physical Functioning Scale	28.00	3.62

Table 2
SEM fit statistics, results: sex predicting chronic sleep problems, youth adult health.

Latent factor	χ^2	CFI	RMSEA (90% CI)	β	P
Youth sleep	χ^2 ($df = 2$, N = 710) = 1.883, $P = .39$	1.00	.00 (.00-.073)	.121	.02
Adult health	χ^2 ($df = 2$, N = 710) = 7.971, $P = .02$	.954	.065 (.023-.115)	.157	.004

Table 3
Influence of competing factors on young adult health in tested model, controlling for sex.[a]

Competing factor	β	P
Early adversity	0.095	.08
Chronic childhood illness	0.002	.35
Maternal depression	0.051	.34
Youth depression, age 20	**0.246**	<**.001**
Youth social stress, age 20	**0.361**	<**.001**
Youth nonsocial stress, age 20	**0.522**	<**.001**

[a] In addition to the latent factor of childhood sleep, the overall model included the above factors, tested individually as separate predictors of youth health at age 20, as well as youth sex, which was entered as a covariate. Childhood sleep remained a significant predictor of young adult health in all tests ($\beta = .088$-.266, $P = .012$-<.001). Sex also remained a significant predictor of youth sleep and young adult health for models including early adversity, chronic childhood illness, maternal depression, and youth chronic nonsocial stress.

Table 4
SEM model fit statistics: overall model with individually entered competing factors, controlling for sex.

Competing factor	χ^2	CFI	RMSEA (90% CI)
Early adversity	χ^2 ($df = 17$, N = 710) = 32.074, $P = .02$	.955	.035 (.015-.054)
Chronic childhood illness	χ^2 ($df = 17$, N = 710) = 23.917, $P = .12$	.978	.024 (0.00-.045)
Maternal depression	χ^2 ($df = 17$, N = 710) = 23.957, $P = .12$	.979	.024 (0.00-.045)
Youth depression, age 20	χ^2 ($df = 17$, N = 710) = 53.819, $P < .001$	.907	.055 (.039-.072)
Youth chronic social stress, age 20	χ^2 ($df = 17$, N = 710) = 35.949, $P = .005$	.952	.040 (.021-.058)
Youth chronic nonsocial stress, age 20	χ^2 ($df = 17$, N = 710) = 26.698, $P = .06$	.979	.028 (0.00-.048)

References

1. Okun ML. Biological consequences of disturbed sleep: important mediators of health? *Jpn Psychol Res*. 2011;53(2):163–176.
2. Smith MT, Haythornthwaite JA. How do sleep disturbance and chronic pain inter-relate? Insights from the longitudinal and cognitive-behavioral clinical trials literature. *Sleep Med Rev*. 2004;8(2):119–132.
3. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Soc Sci Med Elsevier Ltd*. 2010;71(5):1027–1036.
4. Grandner M, Sands-Lincoln M. Sleep duration, cardiovascular disease, and proinflammatory biomarkers. *Nat Sci*. 2013:93–107.
5. Roeser K, Eichholz R, Schwerdtle B, Schlarb AA, Kubler A. Relationship of sleep quality and health-related quality of life in adolescents according to self- and proxy ratings: a questionnaire survey. *Front Psychiatry*. 2012;3:76.
6. Strine TW, Chapman DP. Associations of frequent sleep insufficiency with health-related quality of life and health behaviors. *Sleep Med*. 2005;6(1):23–27.
7. Roberts RE, Roberts CR, Duong HT. Chronic insomnia and its negative consequences for health and functioning of adolescents: a 12-month prospective study. *J Adolesc Health*. 2008;42(3):294–302.
8. Iglowstein I, Jenni OG, Molinari L, Largo RH. Sleep duration from infancy to adolescence: reference values and generational trends. *Pediatrics*. 2003;111(2):302–307.
9. Sadeh A, Mindell JA, Luedtke K, Wiegand B. Sleep and sleep ecology in the first 3 years: a web-based study. *J Sleep Res*. 2009;18(1):60–73.
10. Jenni OG, Molinari L, Caflisch JA, Largo RH. Sleep duration from ages 1 to 10 years: variability and stability in comparison with growth. *Pediatrics*. 2007;120(4):e769–e776.

11. Goodlin-Jones B, Schwichtenberg AJ, Iosif A-M, Tang K, Liu J, Anders TF. Six-month persistence of sleep problems in young children with autism, developmental delay, and typical development. *J Am Acad Child Adolesc Psychiatry*. 2009;48(8):847–854.
12. Lycett K, Mensah FK, Hiscock H, Sciberras E. A prospective study of sleep problems in children with ADHD. *Sleep Med*. 2014;15(11):1354–1361.
13. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study. *Pediatrics*. 2008;122(5):955–960.
14. Smits MG, van Stel H, Van Der Heijden K, Meijer A, Coenen AML, Kerkhof GA. Melatonin improves health status and sleep in children with idiopathic chronic sleep-onset insomnia: a randomized placebo-controlled trial. *J Am Acad Child Adolesc Psychiatry*. 2003;42(11):1286–1293.
15. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) study. *Am J Prev Med*. 1998;14(4):245–258.
16. De Vriendt T, Moreno LA, De Henauw S. Chronic stress and obesity in adolescents: scientific evidence and methodological issues for epidemiological research. *Nutr Metab Cardiovasc Dis*. 2009;19(7):511–519.
17. Chandola T, Brunner E, Marmot M. Chronic stress at work and the metabolic syndrome: prospective study. *BMJ*. 2006;332(7540):521–525.
18. Keenan-Miller D, Hammen CL, Brennan PA. Health outcomes related to early adolescent depression. *J Adolesc Health*. 2007;41(3):256–262.
19. Keeping JD, Najman JM, Morrison J, Western JS, Andersen MJ, Williams GM. A prospective longitudinal study of social, psychological and obstetric factors in pregnancy: response rates and demographic characteristics of the 8556 respondents. *BJOG*. 1989;96(3):289–297.
20. Achenbach TM, Rescorla LA. Manual for the ASEBA School-Age Forms & Profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families; 2001.
21. Achenbach TM, Rescorla LA. Manual for the ASEBA Adult Forms & Profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families; 2003.
22. Ware JE, Kosinski M, Dewey JE, Gandek B. SF-36 Health Survey: Manual and Interpretation Guide. Quality Metric Inc.; 2000
23. McHorney CA, Ware Jr J, Raczeck A. The MOS 36-Item Short-Form Health Survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. *Med Care*. 1993;31(3):247–263.
24. Hazel NA, Hammen C, Brennan PA, Najman J. Early childhood adversity and adolescent depression: the mediating role of continued stress. *Psychol Med*. 2008;38(4):581–589.
25. Raposa EB, Hammen CL, Brennan PA, O'Callaghan F, Najman JM. Early adversity and health outcomes in young adulthood: the role of ongoing stress. *Health Psychol*. 2014;33(5):410–418.
26. Hammen C. Generation of stress in the course of unipolar depression. *J Abnorm Psychol*. 1991;100(4):555–561.
27. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured clinical interview for DSM-IV axis 1 disorders. Washington, DC: American Psychiatric Press; 1995.
28. Kline RB. Principles and Practice of Structural Equation Modeling. Guilford Press; 2011.
29. Hu LT, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct Equ Model A Multidiscip J*. 1999;6(1):1–55.
30. Quach J, Hiscock H, Ukoumunne OC, Wake M. A brief sleep intervention improves outcomes in the school entry year: a randomized controlled trial. *Pediatrics*. 2011;128(4):692–701.
31. Okura Y, Urban LH, Mahoney DW, Jacobsen SJ, Rodeheffer RJ. Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. *J Clin Epidemiol*. 2004;57(10):1096–1103.
32. McAdams MA, Dam RM, Hu FB. Comparison of self-reported and measured BMI as correlates of disease markers in US adults. *Obesity*. 2007;15(1):188–196.
33. Ferraro KF, Su Y-P. Physician-evaluated and self-reported morbidity for predicting disability. *Am J Public Health*. 2000;90(1):103.
34. Iwasaki M, Iwata S, Iemura A, et al. Utility of subjective sleep assessment tools for healthy preschool children: a comparative study between sleep logs, questionnaires, and actigraphy. *J Epidemiol*. 2010;20(2):143–149.
35. Grandner MA, Patel NP, Gehrman PR, Perlis ML, Pack AI. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies. *Sleep Med Rev Elsevier Ltd*. 2010;14(4):239–247.
36. Meerlo P, Sgoifo A, Suchecki D. Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Med Rev*. 2008;12(3):197–210.
37. Knutson K, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. *Sleep Med Rev*. 2007;11(3):163–178.
38. Koskenvuo K, Hublin C, Partinen M, Paunio T, Koskenvuo M. Childhood adversities and quality of sleep in adulthood: a population-based study of 26,000 Finns. *Sleep Med*. 2010;11(1):17–22.
39. Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. *Psychosom Med*. 2003;65(2):259–267.
40. Tsuno N, Besset A, Ritchie K. Sleep and depression. *J Clin Psychiatry*. 2005:1255–1270.