Sleep Health 5 (2019) 187–192

Contents lists available at ScienceDirect

Sleep Health

Journal of the National Sleep Foundation

journal homepage: sleephealthjournal.org

Sleep quality and perceived health in college undergraduates with adverse childhood experiences

Darlynn M. Rojo-Wissar, MPH [a,*], Ryan D. Davidson, PhD [b], Connie J. Beck, PhD [b], Ume S. Kobayashi, BS [c], Angela C. VanBlargan, BS, BA [c], Patricia L. Haynes, PhD [c]

[a] Johns Hopkins Bloomberg School of Public Health, Department of Mental Health
[b] University of Arizona College of Science, Psychology Department
[c] University of Arizona Mel and Enid Zuckerman College of Public Health, Department of Health Promotion Sciences

ARTICLE INFO

Article history:
Received 14 June 2018
Received in revised form 18 October 2018
Accepted 16 November 2018

Keywords:
Adverse childhood experiences
Sleep quality
Depression anxiety

ABSTRACT

Background: Research demonstrates that poor sleep quality is a predictor of chronic mental and physical health problems. The link between adverse childhood experiences (ACEs) and poor health outcomes is also well established; however, few studies have examined the relationships between ACEs, sleep quality, and physical and mental health.
Methods: The current study used structural equation modeling to assess the direct and indirect relationships between ACEs, sleep quality, symptoms of depression and anxiety, and general health perception in a sample of college undergraduates (N = 399), a group of individuals whose age is notable for only recently transitioning out of childhood.
Results: Indirect (ie, mediation) effects indicated with 95% confidence that sleep quality mediated the relationship between ACEs and general health perception, depressive symptoms, and symptoms of anxiety. Sleep quality did not account for the entire relationship between ACE score and these health outcomes, indicating partial mediation. When reversing the mediator and outcome variables, depression and anxiety fully mediated the relationship between ACE score and sleep quality.
Conclusion: The results of this study suggest that sleep quality may be an important intermediary mechanism by which ACEs might contribute to poor health outcomes and especially poor general health perception. Prospective longitudinal research is needed to examine the directionality of the relationships between ACEs, sleep quality, and physical and mental health outcomes over time.

Introduction

Adverse childhood experiences (ACEs) are stressful or traumatic events that occur before the age of 18. Examples of ACEs include physical, verbal, and sexual abuse; neglect; witnessing domestic violence; parental divorce; household substance abuse; mental illness; and incarceration.[1] Exposure to ACEs is prevalent throughout the US population, with 61.55% of individuals reporting experiencing at least 1 ACE and 24.64% of individuals reporting experiencing 3 or more ACEs.[2] The high prevalence of ACEs is cause for concern given the dose-response relationship associating ACEs with chronic mental and physical health problems later in life.[1,3]

The original ACE study, which primarily included white adults older than 35 years, found associations between cumulative ACE exposure and risk for alcoholism, drug abuse, depression, suicide attempts, smoking, self-rated poor health, obesity, heart disease, cancer, lung disease, skeletal fractures, and liver disease.[1] Subsequent ACE studies found further support for the link between ACEs and chronic diseases, risky health behaviors, and mental health problems across a variety of age groups and settings.[4–7] Even in emerging adulthood, ACEs are associated with an increased risk for mental health conditions such as depression and anxiety,[8] and overall greater self-reported health concerns and medical care utilization.[9] Much of the research in this area, however, has focused on older adult samples. To expand the ACE literature, it is important to study these relationships in younger samples, such as college students, as it provides insight into potential mediators involved in the ACE-health associations in the earlier years that could be targeted for intervention.

Preliminary research supports that sleep may operate as one such mechanism. A growing body of literature has found associations between ACEs and adult sleep disorders.[10] For example, a large survey of Canadian adults found a relationship between early life adversity and current troubled sleep.[11] Other studies show that family conflict

* Corresponding author. Tel.: +1 410 955 3908.
E-mail address: drojowi1@jhu.edu. (D.M. Rojo-Wissar).

https://doi.org/10.1016/j.sleh.2018.11.007
2352-7218/

and abuse during childhood significantly predicted adult insomnia.[12,13] A few studies suggest the relationship between sleep and early life adversity may actually begin in childhood, closer to the time when the adversities occurred. In one study, children in families with higher parental depressive symptoms and conflict had shorter actigraphy-measured and self-reported sleep duration than children in families with less conflict and parental depressive symptoms.[14] Additionally, adopted children with a history of maltreatment were found to have significant sleep disruption, which was associated with increased externalizing behaviors.[15] Taken together, these studies indicate that exposure to ACEs is associated with sleep problems for both children and adults. It has been proposed that ACEs may promote vulnerability to sleep problems through alteration of the hypothalamic-pituitary-adrenal axis resulting from epigenetic changes that occur after continued activation of stress response systems.[16,17] These biological changes are thought to increase vulnerability to stress-related hyperarousal and insomnia.

Similar to ACEs, sleep disturbances are shown to be associated with chronic diseases in later life.[18] Some of these chronic diseases include obesity, diabetes, and cardiovascular disease.[18,19] As with ACEs, sleep disturbances are also found to be associated with mental health disorders like anxiety and depression.[20] Despite what we know about the relationships between ACEs and health and sleep quality and health, only a few studies have examined both sleep and health outcomes in the context of ACEs. One study found that poor sleep quality mediated the association between cumulative childhood abuse and risk of a metabolic syndrome diagnosis in adult men and women.[21] Another reported similar findings, showing that childhood abuse was associated with sleep disturbances, inflammation, and subsequently hypertension in middle-aged adults.[22]

To date, there are no known studies that have examined sleep quality as a potential mediator between cumulative exposures to the breadth of ACEs assessed in the ACE questionnaire and symptoms of depression and anxiety, and general health perception. The current study is the first study to our knowledge to investigate these associations. It is a first step in understanding the relationship between *cumulative* exposure to ACEs, sleep quality, and health, with the goal of providing a greater foundation for future longitudinal studies. Consistent with previous findings of sleep acting as a mediator between specific types of early life adversity and health, we examined sleep quality as a potential mediator between ACEs and each health variables independently (Fig. 1). Because this is a cross-sectional study designed to inform future prospective research, we also examined reverse mediational processes, that is, whether health problems mediate the relationship between ACEs and sleep quality.

Methods

Participants

Subjects were recruited through a mass survey given to undergraduates enrolled in introductory psychology courses during the 2014 fall semester at the University of Arizona. From the mass survey, a total of 399 students between the ages of 18 and 30 selected to participate in the study and received course credit for their participation. To eliminate external factors that could influence sleep, exclusion criteria included engagement in shift work; positive screening for sleep apnea; diagnosis of narcolepsy, sleep apnea, schizophrenia, or bipolar disorder; and use of medications to treat hallucinations or delusions.

Procedure

This study was approved by the University of Arizona's Human Subject Protections Program. Undergraduates enrolled in introductory psychology courses during fall semester of 2014 were given a mass survey containing screening questions from multiple investigators at the university for selection of potential participants into various studies. Following completion of the mass survey, students were able to access an online Web site where they chose which research studies to participate in for course credit. Select ACE items were administered in the in-person mass survey on paper. Students who elected to participate in the current study provided informed consent electronically and were electronically instructed to complete a self-report survey that included demographic data, measures related to perceived states of mental and physical health, subjective sleep quality, and several additional ACE items relevant to younger adults.

Measures

Adverse childhood experiences

Adversity was measured using the original ACE survey.[1] In addition, 4 adversity items previously suggested as being relevant for younger cohorts were included.[23] Each ACE item was coded as a dichotomous variable (0 = not experienced; 1 = experienced). Participant's reported parental income was used as a proxy for socioeconomic status (SES). The lowest 10% of reported parental income for the sample was coded as having low SES (scored as 1).

General health perception

General health perception (GH) was measured using the 5-item subscale from the 36-item Short Form Health Survey[24] which has been validated for use in the general population,[25] and the internal consistency of the 5 items in the current study was within acceptable limits ($\alpha = .79$). The items were summed using a 5-point Likert scale rating how true or false statements were about their perceived health status (1 = definitely true, 2 = mostly true, 3 = about halfway between mostly true and mostly false, 4 = mostly false, and 5 = definitely false).

Depression

The Patient Health Questionnaire- 9 (PHQ-9), a validated measure of depression in young adults, was used to measure symptoms of depression.[26,27] The PHQ-9 is a 9-item questionnaire concerning depressive symptoms experienced over the past 2 weeks. Each item

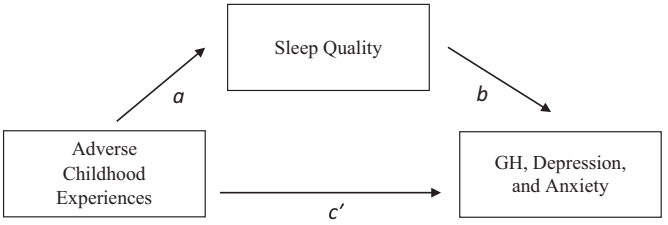

Fig. 1. Hypothesized mediational model. The above figure demonstrates the hypothesized mediational model, with *a* indicating the effect of ACEs on sleep quality and *b* indicating the effect of sleep quality on each of the included outcome measures (GH, depression, and anxiety). The direct effect of ACE score on each outcome is noted as *c*′.

was scored on a 4-point Likert scale (0 = not at all; 1= several days; 2=more than half the days; 3 = nearly every day). A summary score was then calculated, and composite score interpretations were no depression (0-4), mild (5-9), moderate (10-14), moderately severe (15-19), and severe (20-27). Consistent with convention in sleep literature, the sleep-related item was removed to minimize conceptual overlap between sleep and depression[28]; together, the 8 items had good internal consistency ($\alpha = .86$).

Anxiety

The Generalized Anxiety Disorder-7 (GAD-7) was used to measure anxiety.[29] This is a 7-item measure, which assesses the severity of symptoms of generalized anxiety over the previous 2 weeks. Participants rated the severity of each item on a 4-point Likert scale (0 = not at all; 1 = several days; 2 = more than half the days; 3 = nearly every day). The internal consistency of the 7 items in the current study ($\alpha = .91$) was excellent. Summary score interpretations included cutoffs of 5, 10, and 15 for mild, moderate, and severe anxiety, respectively.

Sleep quality

The Pittsburgh Sleep Quality Index (PSQI) assessed sleep quality and disturbances over the past month. Nineteen items generate 7 component scores of subjective sleep quality, latency, duration, efficiency, disturbances, use of medication, and daytime dysfunction. The summation of these component scores yields 1 global score, which was used for this study. A cutoff score of greater than 5 is indicative of serious sleep problems, with 89.6% sensitivity and 86.5% specificity.[30] The internal consistency of the items in the current study was acceptable ($\alpha = .73$).

Data analysis

To examine if sleep quality mediated the effects of ACE score on health (depression, anxiety, and GH), 3 mediation models were tested using a structural equation modeling, nonparametric bootstrap method with bias-corrected confidence estimates.[31] In testing for a mediation, several effects are estimated: the effect of ACE score (X) on sleep quality (M) (a in Fig. 1); sleep quality (M) on health (Y) (b); the ACE score (X) on health (Y) through sleep quality (M), which is called the *indirect effect* (a^*b); ACE score (X) on health (Y) keeping sleep quality constant (M), which is called the *direct effect* (c'); and the total effect (c), which is the combination of the indirect and direct effects (or the effect of ACE score [X] on health [Y]). Bootstrapping does not require a^*b to be normally distributed and so is the preferred method to test if the indirect effect is different from zero.[31] If the indirect effect is significant, then mediation is present.[32] If the direct effect is nonsignificant in the presence of a significant indirect effect, the effect of X on Y exists completely through M, and if the direct effect is significant, M accounts for a portion of the relationship between X and Y.[32] Model A examined GH as the health outcome of interest, Model B examined depression as the outcome, and Model C examined anxiety as the outcome. Because these data are cross-sectional, 3 additional mediation models were run reversing the mediator and outcome variable paths to test whether each health measure separately mediated the relationship between ACE score and sleep quality. Model D examined perceived general health as the mediator, model E examined depression as the mediator, and Model F examined anxiety as the mediator. Participant race and sex were adjusted for in all hypothesized and path reversal models. Since low SES was used as an ACE item, parental income was not included as a covariate in the models.

To account for missing data, multiple imputation was used for each model. Included in the imputation models were the main variables of interest and auxiliary variables. Auxiliary variables were associated with the main variables of interest and missingness to improve the model and increase the likelihood of the assumption that the data were missing at random. Multiple imputation and mediation analyses were completed using the bmem package in R, which imputes missing data and runs the structural equation modeling mediation model in each bootstrap sample.[33] After obtaining the mediation effect estimates, confidence intervals of the model parameters and mediation effects are constructed. All analyses were performed in R version 3.5.1.

Results

The mean age of the participants was 18.71 (SD = 1.09) years, and 30% were male. Participants self-reported their race and identified as 58% White, 22% Hispanic, 12% Asian, and 8% other. Descriptive data on each variable of interest for complete cases and full data are provided (Table 1). The average ACE score was 1.65 (SD = 1.89) for males and 1.85 (SD = 2.08) for females. By race/ethnicity, the average ACE score for participants identifying as whites was 1.54 (SD = 1.72), Hispanics 2.31 (SD = 2.64), Asians 1.27 (SD = 1.39), and others 3.14 (SD = 2.51). Bivariate correlations were examined for all variables of interest using complete cases (Table 2).

Approximately 21% of participants were absent on the first day when select ACE items were administered during the mass survey (n = 85) and thus had incomplete ACE data. Data for the PSQI (n = 56), GH (n =19), PHQ-9 (n = 40), and GAD-7 (n =16) were missing for 14%, 4.7%, 10%, and 4% of participants, respectively. The missingness of ACE, PSQI, PHQ-9, and GAD-7 scores were associated with at least 1 variable and are therefore considered missing at random. Sixty-six percent of participants had complete data on ACE score, PSQI, and PHQ-9; 72% had complete data on ACE score, PSQI, and GAD-7; and 73% had complete data on ACE score, PSQI, and GH. To be conservative and consistent across models, the combination of our main variables of interest with the highest amount of missing data was used to determine the number of imputations to perform, which was 34% missing data. We used 80 imputations and 1000 bootstraps, which perform well with 40% missing data.[33]

The hypothesized bootstrap mediation models in the imputed data as well as in the complete cases are provided in Table 3. Higher ACE scores were associated with poorer sleep quality (a). In turn, poorer sleep quality was associated with poorer GH (model A, b), greater depressive symptoms (model B, b), and greater symptoms of anxiety (model C, b). The indirect effects (a^*b) of ACE score on GH (Model A), depression (Model B), and anxiety (Model C) individually, through sleep quality, were all different than zero with 95% confidence. Moreover, higher ACE scores remained associated with GH, depression, and anxiety even after taking into account the indirect effect of ACE score through sleep quality (c').

The path reversal bootstrap mediation models in the imputed data and combined cases are provided in Table 4. Higher ACE scores were associated with poorer GH (Model D, a), greater depressive symptoms (Model E, a), and more symptoms of anxiety (Model D, a). In turn, poorer GH (model D, b), greater depressive symptoms (Model E, b), and more symptoms of anxiety (Model F, b) were

Table 1
Complete case and full data descriptive statistics

	Complete cases			Full data
	M (SD)	n	M (SD)	n
ACEs	1.79 (2.04)	225	1.75 (1.99)	315
PSQI	6.25 (3.11)	225	6.00 (3.12)	366
PHQ-9	5.54 (4.78)	225	5.77 (4.97)	359
GAD-7	6.29 (5.21)	225	6.02 (5.15)	383
GH	18.98 (3.89)	225	19.14 (3.98)	380

Table 2
Bivariate correlation matrix between variables of interest in complete case data*, **

	ACEs	PSQI	GAD-7	PHQ-9	GH
ACEs	–				
PSQI	.28***	–			
GAD-7	.38***	.42***	–		
PHQ-9	.38***	.44***	.75***	–	
GH	−.24***	−.29***	−.26***	−.32***	–

* $P < .05$.
** $P < .01$.
*** $P < .001$.

each individually associated with poorer sleep quality. The indirect effects (a^*b) of ACE score on sleep quality through GH (Model D), depression (Model E), and anxiety (Model F) were all different than zero with 95% confidence. When considering the indirect effect of ACE score on sleep quality through depression and anxiety, ACE score was no longer directly associated with sleep quality (c'), indicating full mediation. Higher ACE scores still remained associated with sleep quality when considering the indirect effect of ACE score on sleep quality through GH (c').

Discussion

The results of this study suggest that sleep quality may be an important intermediary mechanism by which ACEs might contribute to poor health outcomes. In particular, sleep quality seems to have the strongest mediational relationship between ACEs and GH. When comparing reverse models, the effect was slightly larger when sleep quality mediated the relationship between ACEs and GH vs GH mediating the relationship between ACEs and sleep quality. This is consistent with previous research showing that sleep quality mediates the relationship between cumulative exposure to abuse (emotional, physical, and sexual) and diagnosis of chronic medical conditions later in life.[21,22] These findings along with previous research indicate that sleep quality may be an important pathway or mechanism by which exposure to early life adversity impacts long-term physical health.

When investigating the relationship between ACEs and depression and anxiety, a different pattern emerged. Sleep quality partially mediated the relationship between ACEs and depression and anxiety, whereas depression and anxiety fully mediated the relationship between ACEs and sleep quality. Further research is needed to investigate the temporal nature of these relationships, but these findings indicate that depression and anxiety may be a potential pathway affecting sleep quality in young adults who have been exposed to early life adversity and abuse.

Taken together, findings from the GH, depression, and anxiety models suggest that the relationship between ACEs, sleep, and general and mental health may work within a feedback loop, with each symptom (sleep quality, depression, anxiety, and GH) affecting the others. This is consistent with previous research showing bidirectional and cyclical relationships between sleep and both mental and physical health.[34]

Limitations

There are several limitations to consider when interpreting results of the current study. The cross-sectional design limits the ability to assess the directionality of the associations between sleep and health. Despite the limitations of cross-sectional mediation analyses,[35] these findings are consistent with previous studies showing sleep disturbance as a mediator between other categories of early life adversity or stress and health.[21,22] Further, the current study used self-report assessments of sleep and health and did not include objective measures. As previously noted, there can be differences in subjective and objective measures of sleep quality.[36] Generalizability of the current study is limited to college students, with the participants in the current study being primarily White or Hispanic. These young adults may be distinctly different from those young adults who do not attend college given that maltreatment histories are associated with impairments in academic achievement.[37] In addition, previous research indicates that there may be different pathways by which depression is associated with early life adversity.[38] Although we controlled for race/ethnicity in the current analyses, there may be racial/ethnic or sex differences in the frequency of exposure to ACEs as well as for the relationships between ACEs and physical and mental health. Lastly, there was a significant portion of missing data. Multiple imputation with 80 imputations (recommended number for approximately 30% missingness) and addition of auxiliary variables were used to handle missing data.

Although there were several study limitations, there were also several strengths, including being the first study to show a relationship between sleep quality and health variables in college students with cumulative exposure to a wide array of ACEs; use of a sample of young adults who could be targeted for early intervention; use of a sophisticated missing data handling technique (multiple imputation)

Table 3
Bootstrap mediation effects of hypothesized models of ACEs on health as mediated through sleep quality

		Imputed data				Complete cases			
Model	Effect	Estimate	SE	LL 95% CI	UL 95% CI	Estimate	SE	LL 95% CI	UL 95% CI
A	*a*	0.43	0.09	0.25	0.62	0.33	0.11	0.13	0.56
	b	−0.28	0.07	−0.42	−0.15	−0.18	0.08	−0.33	−0.03
	c'	−0.36	0.13	−0.64	−0.12	−0.34	0.15	−0.66	−0.08
	a × *b*	−0.12	0.04	−0.21	−0.07	−0.06	0.03	−0.14	−0.01
	c	−0.48	0.12	−0.74	−0.25	−0.40	0.14	−0.69	−0.15
B	*a*	0.42	0.09	0.25	0.61	0.33	0.10	0.13	0.53
	b	0.58	0.08	0.43	0.74	0.44	0.09	0.26	0.59
	c'	0.60	0.16	0.28	0.88	0.65	0.16	0.37	0.96
	a × *b*	0.24	0.06	0.14	0.37	0.14	0.05	0.06	0.27
	c	0.84	0.16	0.52	1.13	0.80	0.16	0.50	1.12
C	*a*	0.41	0.09	0.21	0.58	0.33	0.10	0.13	0.54
	b	0.56	0.08	0.41	0.74	0.46	0.10	0.27	0.65
	c'	0.71	0.16	0.38	0.99	0.89	0.17	0.56	1.20
	a × *b*	0.23	0.06	0.12	0.35	0.15	0.06	0.06	0.28
	c	0.94	0.16	0.59	1.23	1.04	0.17	0.71	1.36

Model A includes GH as the outcome, Model B includes depression as the outcome, and Model C includes anxiety as the outcome. Results are based on 1000 bootstrap samples, and 80 imputations were generated for the imputed data; all Models are adjusted for race and sex. 95% CI, bias-corrected confidence interval; LL, lower limit; SE, bootstrap standard error; UL, upper limit.

Table 4
Bootstrap mediation effects of path reversal models of ACEs on sleep quality as mediated through health

		Imputed data				Complete cases			
Model	Effect	Estimate	SE	LL 95% CI	UL 95% CI	Estimate	SE	LL 95% CI	UL 95% CI
D	*a*	−0.46	0.12	−0.70	−0.25	−0.40	0.14	−0.68	−0.13
	b	−0.18	0.04	−0.26	−0.11	−0.11	0.05	−0.22	−0.01
	c'	0.32	0.09	0.12	0.50	0.28	0.11	0.06	0.50
	a x b	0.08	0.03	0.04	0.15	0.05	0.03	0.01	0.11
	c	0.41	0.09	0.23	0.58	0.33	0.10	0.14	0.54
E	*a*	0.85	0.16	0.53	1.15	0.80	0.16	0.48	1.09
	b	0.24	0.03	0.18	0.31	0.21	0.05	0.13	0.33
	c'	0.21	0.10	0.03	0.41	0.16	0.11	−0.04	0.38
	a x b	0.20	0.05	0.11	0.30	0.17	0.06	0.09	0.32
	c	0.42	0.09	0.24	0.59	0.33	0.10	0.13	0.53
F	*a*	0.95	0.16	0.63	1.25	1.04	0.17	0.75	1.38
	b	0.22	0.03	0.16	0.28	0.20	0.04	0.12	0.29
	c'	0.21	0.10	0.02	0.39	0.12	0.11	−0.08	0.35
	a x b	0.21	0.05	0.13	0.32	0.21	0.05	0.12	0.34
	c	0.42	0.09	0.24	0.58	0.33	0.10	0.14	0.54

Model D includes GH as the mediator, Model E includes depression as the mediator, and Model F includes anxiety as the mediator. Results are based on 1000 bootstrap samples, and 80 imputations were generated for the imputed data; all models are adjusted for race and sex.

and statistical technique (bootstrap structural equation modeling approach to mediation); a relatively large sample size; and well-validated measures of subjective sleep and health.

Implications and future directions

The current findings have several important implications. Results provide evidence that negative health correlates of ACEs can be observed closer to the time of the adversities, in emerging adulthood. These findings suggest that early intervention could be integral to preventing long-term negative health outcomes for individuals with ACEs and that sleep may be an effective target. College campuses could target sleep health through college campus community health promotion programs and student health services. Administering a population-relevant ACE questionnaire[23,39] to students coming into the student health clinic with somatic complaints or symptoms of depression or anxiety can alert providers to assess for and treat existing sleep problems. Targeting sleep as an intervention for mental and physical health outcomes may be effective, regardless of directionality. Previous studies show that treatment of insomnia with cognitive behavioral therapy for insomnia can improve mental and physical health symptoms.[40] In college students, sleep may be a more amenable behavioral intervention than direct mental health intervention given the negative stigma associated with seeking behavioral health assistance.

Future research examining sleep quality as a mediator between ACEs and health should address limitations of the current study by using a longitudinal study design, objective measures of sleep quality (actigraphy) and health (health records, heart rate variability, heart rate, blood pressure), and diverse samples. Future studies could then examine whether the relationships between ACEs, sleep quality, and health vary by sex and/or race/ethnicity, as previous research indicates that this may be the case. Findings from these studies will inform the design of interventions targeting sleep quality and health in young adults with ACEs. As the field begins to better understand how ACEs, sleep quality, and mental and physical health outcomes are linked, interventions can be even more tailored to a specific young adult population to potentially prevent some of the negative long-term health consequences of ACEs. Advancements in this area could have a cascade effect on the well-being of individuals exposed to ACEs as well as the economic consequences associated with ACEs.

Conclusions

The current findings are consistent with previous findings linking greater exposure to ACEs with poorer sleep quality,[11,13] perceived health,[9] depression, and anxiety symptoms.[8] However, this is the first study to show a relationship between sleep quality and health variables in college students with cumulative exposure to a wide array of ACEs. Most research to date has examined relationships between ACEs and physical and mental health disorders in later life.[1,4,23] The current findings suggest that sleep quality may interact in a bidirectional manner with physical and mental health outcomes following exposure to ACEs. This study also suggests that sleep is an important factor to assess in the context of ACEs and an important outcome to screen for and address in college campus health services.

Role of funding source

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Darlynn M. Rojo-Wissar is supported by the National Institute of Mental Health's Psychiatric Epidemiology Training Program (5T32MH014592-39; PI: Zandi, Peter).

Disclosure

The authors have no conflicts of interest to disclose.

Acknowledgments

Ms. Rojo-Wissar would like to thank the late Dr. Dick Bootzin for his support and guidance in the development of this study alongside Dr. Connie Beck and Dr. Tricia Haynes.

References

1. 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. https://doi.org/10.1016/S0749-3797(98)00017-8.
2. Merrick MT, Ford DC, Ports KA, Guinn AS. Prevalence of adverse childhood experiences from the 2011-2014 Behavioral Risk Factor Surveillance System in 23 states. *JAMA Pediatr*. 2018. https://doi.org/10.1001/jamapediatrics.2018.2537.
3. Dube SR, Felitti VJ, Dong M, Giles WH, Anda RF. The impact of adverse childhood experiences on health problems: evidence from four birth cohorts dating back to

1900. *Prev Med*. 2003;37(3):268–277. https://doi.org/10.1016/S0091-7435(03)00123-3.
4. Brown MJ, Thacker LR, Cohen SA. Association between adverse childhood experiences and diagnosis of cancer. *PLoS One*. 2013;8(6):1–6. https://doi.org/10.1371/journal.pone.0065524.
5. Dong M, Giles WH, Felitti VJ, et al. Insights into causal pathways for ischemic heart disease: Adverse childhood experiences study. *Circulation*. 2004;110(13):1761–1766. https://doi.org/10.1161/01.CIR.0000143074.54995.7F.
6. Forster M, Grigsby TJ, Rogers CJ, Benjamin SM. The relationship between family-based adverse childhood experiences and substance use behaviors among a diverse sample of college students. *Addict Behav*. 2018;76(September 2017):298–304. https://doi.org/10.1016/j.addbeh.2017.08.037.
7. Rehkopf DH, Headen I, Hubbard A, et al. Adverse childhood experiences and later life adult obesity and smoking in the United States. *Ann Epidemiol*. 2016;26(7):488–492.e5. https://doi.org/10.1016/j.annepidem.2016.06.003.
8. Li M, D'Arcy C, Meng X. Maltreatment in childhood substantially increases the risk of adult depression and anxiety in prospective cohort studies: systematic review, meta-analysis, and proportional attributable fractions. *Psychol Med*. 2016;46(04):717–730. https://doi.org/10.1017/S0033291715002743.
9. Thompson R, Flaherty EG, English DJ, et al. Trajectories of adverse childhood experiences and self-reported health at age 18. *Acad Pediatr*. 2015;15(5):503–509. https://doi.org/10.1016/j.acap.2014.09.010.
10. Kajeepeta S, Gelaye B, Jackson CL, Williams MA. Adverse childhood experiences are associated with adult sleep disorders: a systematic review. *Sleep Med*. 2015;16(3):320–330. https://doi.org/10.1016/j.sleep.2014.12.013.
11. Baiden P, Fallon B, den Dunnen W, Boateng GO. The enduring effects of early-childhood adversities and troubled sleep among Canadian adults: a population-based study. *Sleep Med*. 2015;16(6):760–767. https://doi.org/10.1016/j.sleep.2015.02.527.
12. Gregory AM, Caspi A, Moffitt TE, Poulton R. Family conflict in childhood: a predictor of later insomnia. *Sleep*. 2006;29(8):1063–1067 [http://www.scopus.com/inward/record.url?partnerID=yv4JPVwI&eid=2-s2.0-33747455341&md5=adc04f5dc4416ed0bb55b1bb41cc3a2f].
13. Greenfield EA, Lee C, Friedman EL, Springer KW. Childhood abuse as a risk factor for sleep problems in adulthood: evidence from a U.S. national study. *Ann Behav Med*. 2011;42(2):245–256. https://doi.org/10.1007/s12160-011-9285-x.
14. El-Sheikh M, Kelly RJ, Bagley EJ, Wetter EK. Parental depressive symptoms and children's sleep: The role of family conflict. *J Child Psychol Psychiatry Allied Discip*. 2012;53(7):806–814. https://doi.org/10.1111/j.1469-7610.2012.02530.x.
15. Cuddihy C, Dorris L, Minnis H, Kocovska E. Sleep disturbance in adopted children with a history of maltreatment. *Adopt Foster*. 2013;37(4):404–411. https://doi.org/10.1177/0308575913508715.
16. Palagini L, Drake CL, Gehrman P, Meerlo P, Riemann D. Early-life origin of adult insomnia: does prenatal-early-life stress play a role? *Sleep Med*. 2015;16(4):446–456. https://doi.org/10.1016/j.sleep.2014.10.013.
17. Davidson RD, O'Hara KL, Beck CJA. Psychological and biological processes in children associated with high conflict parental divorce. *Juv Fam Court J*. 2014;65(1):29–44. https://doi.org/10.1111/jfcj.12015.
18. Gilbert LK, Breiding MJ, Merrick MT, et al. Childhood adversity and adult chronic disease: an update from ten states and the District of Columbia, 2010. *Am J Pract Nurs*. 2015;48(3):345–349. https://doi.org/10.1016/j.amepre.2014.09.006.
19. Wu Y, Zhai L, Zhang D. Sleep duration and obesity among adults: a meta-analysis of prospective studies. *Sleep Med*. 2014;15(12):1456–1462. https://doi.org/10.1016/j.sleep.2014.07.018.
20. Alvaro PK, Roberts RM, Harris JK. A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. *Sleep*. 2013;36(7):1059–1068. https://doi.org/10.5665/sleep.2810.
21. Lee C, Tsenkova V, Carr D. Childhood trauma and metabolic syndrome in men and women. *Soc Sci Med*. 2014;105:122–130. https://doi.org/10.1016/j.socscimed.2014.01.017.
22. Petrov ME, Davis MC, Belyea MJ, Zautra AJ. Linking childhood abuse and hypertension: sleep disturbance and inflammation as mediators. *J Behav Med*. 2016;39(4):716–726. https://doi.org/10.1007/s10865-016-9742-x.
23. Finkelhor D, Shattuck A, Turner H, Hamby S. A revised inventory of Adverse Childhood Experiences. *Child Abuse Negl*. 2015;48:13–21. https://doi.org/10.1016/J.CHIABU.2015.07.011.
24. Ware Jr JE, Sherbourne CD. The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. *Med Care*. 1992;30(6):473–483. https://doi.org/10.2307/3765916.
25. Brazier JE, Harper R, Jones NM, et al. Validating the SF-36 health survey questionnaire: new outcome measure for primary care. *BMJ*. 1992;305(6846):160–164. https://doi.org/10.1136/bmj.305.6846.160.
26. Keum BT, Miller MJ, Inkelas KK. Testing the factor structure and measurement invariance of the PHQ-9 across racially diverse U.S. college students. *Psychol Assess*. 2018. https://doi.org/10.1037/pas0000550.
27. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9. *J Gen Intern Med*. 2001;16(9):606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x.
28. Buysse DJ, Hall ML, Strollo PJ, et al. Relationships between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and clinical/polysomnographic measures in a community sample. *J Clin Sleep Med*. 2008;4(6):563–571. https://doi.org/10.1017/S174413741200015X.
29. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder. *Arch Intern Med*. 2006;166(10):1092. https://doi.org/10.1001/archinte.166.10.1092.
30. Buysse DJ, Reynolds III CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1988;28(2):193–213. https://doi.org/10.1016/0165-1781(89)90047-4.
31. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behav Res Methods*. 2008;40(3):879–891. https://doi.org/10.3758/BRM.40.3.879.
32. MacKinnon DP, Fairchild AJ, Fritz MS. Mediation analysis. *Annu Rev Psychol*. 2007;58(1):593–614. https://doi.org/10.1146/annurev.psych.58.110405.085542.
33. Zhang Z, Wang L. Methods for mediation analysis with missing data. *Psychometrika*. 2013;78(1):154–184. https://doi.org/10.1007/s11336-012-9301-5.
34. Zee PC, Turek FW. Sleep and health: everywhere and in both directions. *Arch Intern Med*. 2006;166(16):1686–1688. https://doi.org/10.1001/archinte.166.16.1686.
35. Maxwell SE, Cole DA, Mitchell MA. Bias in cross-sectional analyses of longitudinal mediation: partial and complete mediation under an autoregressive model. *Multivar Behav Res*. 2011;46(5):816–841. https://doi.org/10.1080/00273171.2011.606716.
36. Buysse DJ, Hall ML, Patrick, et al. Epworth Sleepiness Scale (ESS), and Clinical/Polysomnographic Measures in a Community Sample. Vol 4. http://jcsm.aasm.org/Articles/040608.pdf; 2008, Accessed date: 3 October 2018.
37. Romano E, Babchishin L, Marquis R, Fréchette S. Childhood maltreatment and educational outcomes. *Trauma Violence Abuse*. 2014;16(June):418–437. https://doi.org/10.1177/1524838014537908.
38. Turner HA, Butler MJ. Direct and indirect effects of childhood adversity on depressive symptoms in young adults. *J Youth Adolesc*. 2003;32(2):89–103. https://doi.org/10.1023/A:1021853600645.
39. Davidson RD, Rojo-Wissar DM, Sullivan AW, Beck CJ. Assessing health outcomes and youth-related items for the adverse childhood experiences questionnaire. *Emerg Adulthood*. 2018. https://doi.org/10.1177/2167696818756402.
40. Smith MT, Huang MI, Manber R. Cognitive behavior therapy for chronic insomnia occurring within the context of medical and psychiatric disorders. *Clin Psychol Rev*. 2005;25(5):559–592. https://doi.org/10.1016/j.cpr.2005.04.004.