Journal of Community Health (2024) 49:809–819
https://doi.org/10.1007/s10900-024-01344-7

ORIGINAL PAPER

Adverse Childhood Experiences May Dampen the Protective Role of Sleep Duration on Adolescent Obesity Risk

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Accepted: 13 February 2024 / Published online: 2 April 2024

Abstract

The purpose of this study was to explore Adverse Childhood Experiences (ACEs) as a moderator between sleep duration/irregularity and overweight/obesity in U.S. adolescents. Using the National Survey of Children's Health 2017–2018 cross-sectional dataset, we included adolescents with available sleep and Body Mass Index (BMI) data. In a sample of 24,100 adolescents (mean age = 13.56 years, 49.35% female; 51% White), parents reported adolescent's sleep duration/irregularity, and number of ACEs. Logistic regression estimated the interaction between sleep duration/irregularity and the number of ACEs on overweight/obesity risk (BMI ≥ 85th percentile-for-age) using a stepwise approach and accounting for complex survey design. In the 24,100 adolescents, 33% were overweight/obese, 50% had ≥ 1 ACE, 37% slept < 8–10 h/night, and 14% had irregular sleep. Accounting for covariates and ACEs, every hour increase in sleep duration was associated with 6% decrease in overweight/obesity odds. There was a significant interaction between sleep duration and ACEs; the association between increasing sleep duration and decreasing odds of overweight/obesity was significant only in adolescents without ACEs (OR = 0.87, 95% CI [0.80, 0.95], p < 0.001). Increasing sleep duration is a recognized intervention target to decrease obesity risk, yet in adolescents experiencing ≥ 1 ACE, this protective role may be dampened. Future work may explore mechanisms for overweight/obesity development to inform interventions for adolescents facing adversity.

Keywords Adolescents · Sleep health · Overweight · Obesity · Adverse childhood experiences

Introduction

The World Health Organization classifies childhood obesity as a global epidemic [1]. The prevalence of overweight and obesity has doubled in children and quadrupled in adolescents over the past 30 years, with about one-third of children and adolescents classified as overweight or obese and over 100,000 deaths per year attributed to adiposity-related health problems [2, 3]. Conventional interventions targeting individual health behavior change (e.g., caloric restriction, increased physical activity) and energy balance have not substantially decreased obesity prevalence, potentially due to lack of consideration of complex risk factors [4].

Such contributing risk factors to the obesity epidemic include poor sleep and the presence of Adverse Childhood Experiences (ACEs), with strong associations consistently found between short (< 7 h/night)[5] and irregular sleep (i.e., not going to bed within one hour of the prior night's bedtime on a nightly basis) [6] and the presence of ACEs increasing obesity risk in adolescence and into adulthood [1, 7, 8]. The Centers for Disease Control (CDC) defines ACEs as potentially traumatic events that occur in childhood (0–17 years). They can include experiencing family violence, abuse or neglect; witnessing neighborhood violence; growing up in a household with substance misuse, mental health problems and instability due to parental separation or imprisoned household members [9]. In the United States (U.S.), close to two-thirds of individuals report experiencing at least one ACE, and 25% report experiencing three

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or more ACEs [10]; with Americans from ethnic/minority and low socioeconomic groups reporting greater ACE exposure [11]. Regarding sleep, national data estimates that 58% of U.S. middle school students and 73% of high school students obtain inadequate sleep duration [12, 13], and approximately 50% of U.S. youth have irregular sleep patterns [14]. Obtaining adequate sleep, overweight/obesity prevention and decreasing the prevalence of ACEs in young adults are all Healthy People 2030 goals [15]. Not known is the complexity of the relationships between sleep, ACEs and overweight/obesity, which is necessary to inform intervention development and address these 2030 goals.

Complex and Nuanced Relationships Exist at the Intersection Between Sleep, ACEs and Obesity Among Adolescents

Multiple systematic reviews and meta-analyses have shown a consistent association between short and irregular sleep and increased risk for overweight/obesity, with this risk increasing following exposure to adverse social risks [1, 6, 7, 16]. Adolescence may amplify the sleep-obesity association due to developmental shifts in sleep regulatory processes, including diminishing sleep pressure and circadian phase delay. Such maturational changes are then exacerbated by the external constraint of early school start times which may contribute to shorter sleep duration. Moreover, the "24/7" lifestyle of adolescence [17], makes this developmental stage particularly vulnerable to sleep irregularity [18]. To this end, the mechanisms by which poor sleep confers obesity risk may be more complex in adolescence than in childhood and adulthood [7].

Similarly, decades of research show a direct and dose–response relationship between ACEs and obesity risk into adulthood [15, 16, 19, 20]. More recent work has uncovered the presence of this association as early as adolescence [4, 8, 21]. Disruptions in the family microsystem may contribute to chronic stress activation of the hypothalamic–pituitary–adrenal (HPA) axis and increasing allostatic load (physiological wear and tear of the stress response system), which may interfere with self-regulation, psychosocial and neuroendocrine processes, and subsequent obesity risk [4, 8, 22, 23].

Analogously, potential mechanisms underlying the ACEs-poor sleep association may also be driven by activation of the HPA axis and subsequent neurobiological consequences such as sleep disturbances [24]. Indeed, the association between ACEs and poor sleep has been found in U.S. samples of adolescents, with an increased number of ACEs increasing the risk for irregular and short sleep duration [25, 26]; and this negative association tracking into adulthood [24].

These lifelong effects of childhood adversity and poor sleep on obesity risk underscore the necessity of examining their interrelationships during the sensitive period of adolescence [27]. Adolescence may be a potentially opportune period of the life course for obesity intervention. However, prior to the development of sleep interventions to improve obesity risk, it is necessary to have a more nuanced examination of situations where the influence of sleep and ACEs on obesity risk is amplified or attenuated, so the most vulnerable groups are prioritized in targeted interventions [23].

Theoretical Framework

The Socioecological model guides this work [28]. It considers the influence of family, interpersonal and environmental factors, including interactions and relationships, economic resources, and neighborhood community; all of which may contribute to acquirement of ACEs [21]. Similarly, the degree of safety and dysfunction within the family context is important for sleep initiation and maintenance, which contribute to adequate sleep duration and sleep regularity [29]. Consideration of these family variables within the interpersonal and environmental domains of the socioecological model is necessary when accounting for their interplay with obesity development [12].

Current Study

Short sleep duration/sleep irregularity and the prevalence of ACEs are independent predictors of obesity risk in adolescence; thus, it is possible that the presence of ACEs could serve to intensify the relationship between poor sleep and obesity risk, yet few studies have examined ACEs as a moderator of this relationship. Cross-sectional and longitudinal studies that have examined familial and socioeconomic risk as moderators between sleep and body mass index (BMI) in school-age children found that the association between irregular/short sleep duration and BMI was amplified in children experiencing higher levels of familial and socioeconomic risk [22, 23]. Extending this past work and identifying variance in this association will allow us to identify the most at-risk sub-groups (based on adversity experience) for sleep and obesity-related health disparities in adolescence. Indeed, ACEs exposure could make adolescents more susceptible to the negative effects of poor sleep on overweight/obesity [23]. Not all adolescents are at equal risk for the sleep-obesity relationship [16, 22, 23]. In the absence of adversity, sleep may be protective against obesity risk however, among vulnerable groups, we need to identify what may attenuate this protective effect.

Characterizing the complex nature of the association among ACEs, sleep and obesity risk is essential to inform interventions to prevent childhood obesity among at-risk

populations [16, 30]. Thus, the aim of this study is to explore the interaction between ACEs and sleep duration/sleep irregularity on obesity risk in a national sample of U.S. adolescents. We hypothesized that the association between short sleep duration/sleep irregularity and overweight/obesity will be amplified in adolescents with one or more ACEs.

Methods

Participants and Procedures

The study team obtained cross-sectional data from the National Survey of Children's Health (NSCH) 2017–2018 combined datasets from the Health Resources and Services Administration's Maternal and Child Health Bureau (HRSA MCHB) and the United States Census Bureau [31]. The NSCH is the largest national-level and state-level survey with data on the well-being of children and adolescents aged 0–17 years, as well as the health care needs of their families and communities in the U.S. Participants were derived from a sample of households from the Census Master Address File across 50 states and the District of Columbia. Selection of households were stratified by state, neighborhood poverty, and child presence, with one child randomly selected from multiple children households [31]. Primary caregivers completed questions about the child's mental and physical health, insurance coverage and characteristics of the child's family and neighborhood [31].

Measures

Overweight/Obesity

The outcome variable of overweight/obesity was created through parent report of their adolescent's height and weight. The NSCH calculated BMI as weight (kg)/height $(m)^2$ and used CDC's BMI-for-Age Growth Charts [31] to classify overweight/obesity as BMI ≥ 85th percentile for age, and healthy BMI as 5th percentile to < 85th percentile for age [31].

Sleep Variables

Sleep Duration Parents reported their child's sleep duration through answering the following question: "During the past week, how many hours of sleep did this child get during an average day (counting both nighttime sleep and naps)?" There were seven categories of responses for this question: 1 = *less than 7 h*, 2 = *7 h, 3* = *8 h*, 4 = *9 h, 5* = *10 h, 6* = *11 h*, and 7 = *12 or more hours*. HRSA MCHB then used the American Academy of Sleep Medicine (AASM) guidelines to classify children as getting age-appropriate recommended sleep duration: 1 = *child sleeps the recommended age-appropriate hours on most weeknights*, 2 = *child sleeps less than recommended age-appropriate hours on most weeknights*[31].

Sleep Regularity Parents reported about their adolescent's sleep regularity through responding to the question: "How often does this child go to bed at about the same time on weeknights?": 1 = *always*, 2 = *usually*, 3 = *sometimes*, 4 = *rarely*, 5 = *never*. Sleep irregularity was characterized by whether the child went to bed "sometimes/rarely/never" about the same time each weeknight (vs. always/usually).

Adverse Childhood Experiences

The NSCH did not include all possible ACEs. It only included items that could be validly reported by the adolescents' parent/caregiver. Questions about abuse and neglect were excluded [32]. In the 2017 and 2018 combined dataset, two versions of ACE's measures were present: (a) including all 9-items asked in the survey and (b) excluding the "economic hardship" item. The present study used the ACEs version based on all 9-items [33].

ACEs were based on parent-report of the following question: "Had this child experienced one or more adverse childhood experiences from the list of 9 ACEs?" The nine childhood traumas were: (1) financial hardship; (2) parental divorce/separation; (3) parental death; (4) parental imprisonment; (5) domestic violence; (6) neighborhood violence; (7) living with a mentally ill, suicidal, or severely depressed person; (8) living with someone who has a substance use problem; and (9) experienced unfair treatment because of their race or ethnicity. Cumulative exposure to ACEs was created by summing the total number of adverse experiences and categorized as: 1 = "*No adverse childhood experiences*"; 2 = "*Experienced 1 or more adverse childhood experiences*". This categorization was chosen based on the distribution of the ACEs variable (i.e., approximately 50% of the sample experienced ≥ 1 ACE).

Covariates

The socioecological model [28] and availability of variables in the NSCH dataset guided selection of potential covariates at the individual, social and environmental level.

Individual-level Covariates Adolescents reported their age as a continuous level variable, and sex was assessed categorically: 1 = *male*, 2 = *female*. Adolescent's physical health was captured through parent-report of their child's physical activity level: "During the past week, on how many days did this child exercise, play a sport, or participate in physical activity for at least 60 min?" We categorized physical activity into: 1 = *daily*, 2 = *not daily*. Adolescent's mental health was captured based on parent-report of past or current

diagnosis of depression: "*Has a doctor or other health care provider EVER told you that this child has depression?"* Responses to this question were separated into 3 categories: 1 = *does not have condition*, 2 = *ever told, but do not currently have condition*, 3 = *currently have condition*. We combined category 1 and 2 to show "currently having/not having depression".

Social Covariates Adolescent's race/ethnicity was operationalized as a social construct, self-reported and categorized as: 1 = *Hispanic*, 2 = *White, non-Hispanic*, 3 = *Black, non-Hispanic*, 4 = *Multi-racial* (two or more race categories) or *other* (Asian, Indian/Alaska Native, Native Hawaiian/Other Pacific Islanders), *non-Hispanic*. Family poverty level (FPL) was calculated as a percentage of household income where the child dwelled. We categorized FPL into two categories: 1 = *0–199%*, 2 = ≥ *200%* [34]. Primary caregiver education was based on the highest level of education among reported parents and was categorized as: 1 = *less than high school*, 2 = *high school or GED*, 3 = *some college or technical school*. We combined categories 1 and 2 due to the small number of primary caregivers with less than high school education. Finally, adolescents' neighborhood condition was measured by asking "Does this child live in a neighborhood where there is poorly kept or rundown housing." Responses to this question were based on the following categories: 1 = *good*, 2 = *poorly kept or dilapidated housing.*

Environmental Covariate Exposure to second-hand household smoke was identified through a yes/no question: "Does anyone living in your household use cigarettes, cigars, or pipe tobacco?".

Statistical Analysis

We conducted data analyses using Stata 16.0 (svy package), accounting for sampling weights and design (stratum = state, cluster = household). First, we performed descriptive statistics using weighted means and percentages. Second, using Wald test (continuous variables) and weighted Pearson Chi-square Statistic with Rao-Scott correction (categorical variables), we tested differences in sample characteristics and sleep duration/regularity between adolescents with and without overweight/obesity. Third, we performed logistic regression to obtain odds ratios (ORs) and 95% confidence intervals (CIs) for associations of sleep duration/regularity and ACEs with overweight/obesity (dependent variable), adjusting for age and sex in the basic model. Sleep duration was used as a continuous variable in the adjusted model. We used a hierarchical model-building approach to select additional covariates, including individual, social and environmental factors (see details in covariates). Covariates were selected according to Wald tests for covariates' significance ($p < 0.05$) and Archer Lemeshow goodness-of-fit statistics for model fit ($p > 0.05$). The final models were adjusted for covariates at the individual (age, sex, depression diagnosis, daily physical activity), social (race/ethnicity, family poverty, primary caregiver education and neighborhood conditions) and environmental (household smoke exposure) levels. Fourth, we added interaction terms between ACEs and sleep duration, and, between ACEs and sleep regularity, separately, to test the moderating effect of ACEs. Finally, we tested the significance of interaction terms using the Wald test and compared the model fit using Archer–Lemeshow goodness-of-fit statistics. We further examined and illustrated significant interactions using margins plots command in Stata 16.0. The significance level was set at $\alpha = 0.05$ (two-tailed tests) for all statistical tests.

Results

Sample Characteristics

The final sample included 24,110 adolescents who had complete data on BMI, sleep duration/regularity and ACEs. The weighted mean age of the study sample was 13.56 years old (ranged 10–17 years old). Nearly half of participants were female (49.35%) and White (51%). Thirty-three percent of adolescents (weighted percentage) had overweight/obesity (n = 7,108), and 37% (n = 8,243) slept shorter than AASM's age-appropriate hours. The majority (86%) always/usually (n = 20,784) went to bed at the same time on weeknights. Half of the sample (n = 11,331) experienced one or more ACEs.

Bivariate Associations

Table 1 shows counts and weighted percentages/means of sample characteristics by BMI status. Sleep duration shorter than age-appropriate hours (design-based $F = 21.87$, $p < 0.001$) and having ≥ 1 ACEs (design-based $F = 57.25$, $p < 0.001$) were positively associated with overweight/obesity. The proportion of overweight/obesity significantly differed between sleep regularity groups (design-based $F = 8.94$, $p < 0.001$), and post hoc tests suggested that adolescents usually going to bed at the same time reported the lowest proportion of overweight/obesity (29.71%) compared to those who sometimes/rarely/never (36.86%) went to bed at the same time, or even always (36.93%) went to bed at the same time (p's < 0.05). We found a slightly higher proportion of overweight/obesity in males than females (34.32% vs. 31.66%, design-based $F = 3.53$, $p = 0.06$). Adolescents who exercised for at least 60 min every day (design-based $F = 20.47$, $p < 0.001$) or who did not have a depression diagnosis (design-based $F = 22.93$, $p < 0.001$) or household smoking exposure (design-based $F = 27.90$, $p < 0.001$) had

Table 1 Descriptive statistics[a] of the study sample overall and by overweight/obesity status

	Overall sample (N = 24,110)	Overweight/obesity status, n (%)	
		Not overweight/obese (n = 17,002)	Overweight/ Obese [b] (n = 7108)
Age, M ± SD	13.56 ± 0.03	13.66 ± 0.04	13.36 ± 0.06***
Sex, n (%)			
Male	12,430 (50.48)	8411 (65.87)	4019 (34.13)
Female	11,680 (49.52)	8591 (68.52)	3089 (31.48)
Sleep duration, n (%)			
Healthy	15,867 (62.81)	11,495 (69.75)	4372 (30.25)***
Insufficient	8243 (37.19)	5507 (62.85)	2736 (37.15)
Sleep regularity, n (%)			
Always	5880 (27.71)	3936 (63.07)	1869 (36.93)***
Usually	14,979 (57.79)	10,850 (70.29)	4129 (29.71)
Sometimes/rarely/never	3263 (14.49)	2169 (63.14)	1094 (36.86)
ACE, n (%)			
No ACE	12,779 (49.56)	9585 (72.56)	3194 (27.44)***
Experienced > 1 ACE	11,331 (50.44)	7417 (61.90)	3914 (38.10)
Parent education, n (%)			
> High school	20,152 (69.16)	14,670 (71.80)	5482 (28.20)***
< High school	3958 (30.84)	2332 (56.83)	1626 (43.17)
Family income, n (%)			
0–199% FPL	6391 (32.82)	4016 (59.95)	2375 (40.05)***
> 200% % FPL	17,719 (67.18)	12,986 (72.13)	4733 (27.87)
Race/Ethnicity, n (%)			
White, non-Hispanic	17,185 (50.87)	12,343 (72.37)	4704 (27.63)***
Hispanic	2699 (25.70)	1721 (60.56)	978 (39.44)
Black, non-Hispanic	1561 (13.96)	916 (58.78)	645 (41.22)
Multi-racial/other	2803 (9.47)	2022 (69.67)	781 (30.33)
Neighborhood condition, n (%)			
Good	21,576 (88.32)	15,358 (68.36)	6218 (31.64)***
Poorly kept/rundown	2403 (11.68)	1558 (58.33)	845 (41.67)
Household smoking, n (%)			
No exposure	20,287 (84.54)	14,674 (68.63)	5613 (31.37)***
Having exposure	3717 (15.46)	2258 (59.33)	1459 (40.67)
Daily exercise, n (%)			
No	20,051 (81.11)	13,777 (65.53)	6113 (34.47)***
Yes	4222 (18.89)	3190 (74.22)	984 (25.78)
Current depression, n (%)			
No	22,321 (94.63)	15,928 (67.79)	6393 (32.21)***
Yes	1647 (5.37)	981 (56.29)	666 (43.71)

Healthy BMI: 5th to < 85th percentile-for-age

Wald test (age) and weighted Pearson Chi-square Statistic with Rao-Scott correction (categorical variables) tested group differences

*p < 0.05; **p < 0.01; ***p < 0.001[a]Weighted mean ± linearized standard deviation and weighted percentage

[b]Overweight/obesity: BMI ≥ 85th percentile-for-age

a lower proportion of overweight/obesity compared to their counterparts (p < 0.001).

In terms of social factors, adolescents who were Hispanic or Non-Hispanic Black (vs. White) (design-based F = 21.84, p < 0.001), whose primary caregiver's education ≤ high school (vs. > high school) (design-based F = 74.35, p < 0.001) or family income levels < 200% FPL (vs. ≥ 200% %) (design-based F = 66.65, p < 0.001), lived

in a neighborhood with poorly kept or rundown housing (design-based F = 17.67, p < 0.001) also had a higher percentage of overweight/obesity than their counterparts.

Adjusted Associations Between Sleep and Overweight/Obesity Status

In Table 2, Model 1 (without interaction terms) shows results from a logistic regression of overweight/obesity after controlling for covariates. For sleep duration, every level increase in a nominal scale from 1 to 7 (representing the range from < 6 h to ≥ 11 h) tended to associate with a 6% decrease in the odds of having overweight/obesity (OR = 0.94, 95% confidence interval (CI) [0.88, 1.00]) and this trend reached marginal significance (p = 0.05). Compared with those who sometimes/rarely/never went to bed at the same time, adolescents who had better sleep regularity did not show different odds of overweight/obesity after controlling for covariates (p > 0.05). Adolescents who had experienced at least one ACE had 40% increased odds of overweight/obesity (OR = 1.40, 95% CI [1.21, 1.61], P < 0.001). Social factors including being Hispanic and Black (non-Hispanic) American, family income < 200% FPL, living in neighborhoods with poorly kept houses, having a diagnosis of depression and household smoking exposure remained significantly associated with greater odds of having overweight/obesity compared with their counterparts (p < 0.05). Additionally, females and those having daily exercise had decreased odds of overweight/obesity (p < 0.05).

Interaction Between Sleep and ACEs

Reporting one or more ACEs (OR = 1.13, 95% CI [1.01, 1.31], p = 0.04) significantly interacted with sleep duration on overweight/obesity (Table 2, Model 2). Specifically, increasing sleep duration and decreasing odds of overweight/

Table 2 Survey logistic regression models [a] on overweight/obesity [OR (95% CI)]

	Model 1	Model 2	Model 3
Age	0.92 (0.89, 0.94)***	0.91 (0.89, 0.94)***	0.92 (0.89, 0.94)***
Female	0.86 (0.75, 0.98)*	0.86 (0.75, 0.98)*	0.86 (0.75, 0.98)*
Sleep duration	0.94 (0.88, 1.00)†	0.87 (0.80, 0.95)***	0.94 (0.88, 1.00)†
Sleep regularity (vs. sometimes/rarely/never)			
Always	1.17 (0.94, 1.45)	0.76 (0.65, 0.89)**	1.32 (0.93,1.88)
Usually	0.89 (0.74, 1.08)	0.86 (0.69, 1.07)	1.01 (0.73,1.39)
ACE > 1 (vs. no ACE)	1.40 (1.21, 1.61)***	0.84 (0.51,1.38)	1.65 (1.16,2.35)***
Parent education < high school (vs. > high school)	1.55 (1.30, 1.83)***	1.54 (1.26, 1.88)***	1.55 (1.31, 1.83)***
Family income < 200% FPL (vs. > 200%)	1.19 (1.02, 1.39)*	1.18 (1.30, 1.83)*	1.19 (1.02, 1.39)*
Race/ethnicity (vs. white)			
Hispanic	1.35 (1.12, 1.62)**	1.43 (1.19, 1.72)**	1.43 (1.19, 1.72)**
Black, non-Hispanic	1.48 (1.21, 1.81)***	1.54 (1.26, 1.88)***	1.52 (1.25, 1.86)***
Multi-racial/other	1.03 (0.85, 1.24)	1.05 (0.88, 1.27)	1.05 (0.88, 1.26)
Neighborhood conditions (vs. not poorly kept)			
Poorly kept	1.25 (1.01, 1.55)*	1.25 (1.01, 1.55)*	1.25 (1.01, 1.55)*
Household smoking	1.29 (1.09, 1.54)**	1.25 (1.06, 1.49)**	1.26 (1.06, 1.50)**
Daily exercise	0.59 (0.49, 0.71)***	0.59 (0.49, 0.71)***	0.59 (0.49, 0.71)***
Depression	1.54 (1.23, 1.93)***	1.56 (1.25, 1.95)***	1.53 (1.23, 1.92)***
Interaction terms			
Sleep duration x ACE		1.13 (1.01, 1.27)*	
Sleep regularity x ACE			
Always regular, ACE > 0			0.82 (0.53,1.27)
Usually regular, ACE > 0			0.83 (0.56,1.22)
Archer–Lemeshow test	F = 1.73, p = 0.08	F = 1.59, p = 0.11	F = 1.66, p = 0.10
Wald test		F = 10.44, p < 0.001	F = 6.67, p < 0.001

†p = 0.05; *p < 0.05; **p < 0.01; ***p < 0.001

[a]Model 1: basic model accounting for complex sampling design and weights, and controlling for covariates. Model 2: Model 1 + sleep duration (continuous variable) and ACEs interaction. Model 3: Model 1 + sleep regularity and ACEs interaction

[b]Overweight/obesity: BMI ≥ 85th percentile-for-age; healthy BMI: 5th to < 85th percentile-for-age.Archer–Lemeshow test: p > 0.05 indicates good model fit. Wald test: p < 0.05 indicates adding the interaction term improves model fit

obesity was significant only in adolescents without ACEs (OR = 0.87, 95% CI [0.80, 0.95], p < 0.001). Figure 1 shows a margins plot of the interaction between sleep duration and ACEs on overweight/obesity. As shown in Fig. 1, compared with those having at least one ACE, adolescents without ACEs had a smaller likelihood of overweight/obesity and the group differences in probability of overweight/obesity were intensified with increasing sleep duration (p < 0.05). Adding the interaction terms showed improved model fit (Wald test, p < 0.05) and the model was a good fit for the data (Archer-Lemeshow test, p > 0.05). There was no significant interaction between sleep regularity and ACEs on overweight/obesity (Model 3, p > 0.05).

Discussion

The purpose of this study was to explore the interaction between ACEs and sleep duration/irregularity on obesity risk to inform future childhood obesity interventions among at-risk populations. Results of the moderation analysis showed that the association between increased sleep duration and lower odds of overweight/obesity was only significant in adolescents who experienced no ACEs. There was no significant interaction between sleep regularity and ACEs on obesity risk. Increasing sleep duration is a conventional intervention target to decrease obesity risk, yet our findings imply that in children experiencing ≥ 1 ACEs, this protective role may be dampened.

This is one of the first studies, to the best of our knowledge, to systematically test how sleep health is related to obesity risk in the context of ACEs. As such, our findings extend previous work. Our a priori hypothesis that the association between poor sleep and obesity risk would be amplified in children experiencing ≥ 1 ACEs was not supported by our findings. Instead, we found that the protective role of long sleep duration on decreased obesity risk was only significant in adolescents who experienced no ACEs. Prior work has found that obtaining adequate sleep duration associates with lower risk of obesity development among children who face less familial and socioeconomic risk in early life [22, 23, 30]. Our work extends these findings as ACEs encompasses additional traumas, such as parental death and imprisonment, domestic violence and racial discrimination, which were not collectively captured in prior work. Moreover, our dichotomization of ACEs (≥ 1 vs. 0) is unique, given past studies' exploration of dose–response relationship or categorization of a greater number of ACEs (i.e., 3–4 ACEs) [19, 20, 25]; and makes our findings clinically and nationally relevant, given the high prevalence of children and adolescents experiencing at least one ACE. Moreover, our nationally representative sample increases the generalizability of our findings to U.S. adolescents.

The lack of the sleep-obesity relationship being amplified in adolescents who experienced ACEs may be explained by the multifactorial causes of obesity development in groups who experience trauma. Indeed, trauma is intergenerational, contributing to epigenetic changes that can predispose children to poor physiological regulation and subsequent metabolic effects [35, 36]. As such, improving sleep may not provide enough defense against overweight/obesity development in groups experiencing trauma [36]. Therefore, future obesity interventions need to include trauma informed practices in addition to targeting health behavior changes that are culturally sensitive [4, 16]. On the other hand, adolescents who experience no childhood trauma, and whose prior generations experienced minimal trauma and/or properly addressed their

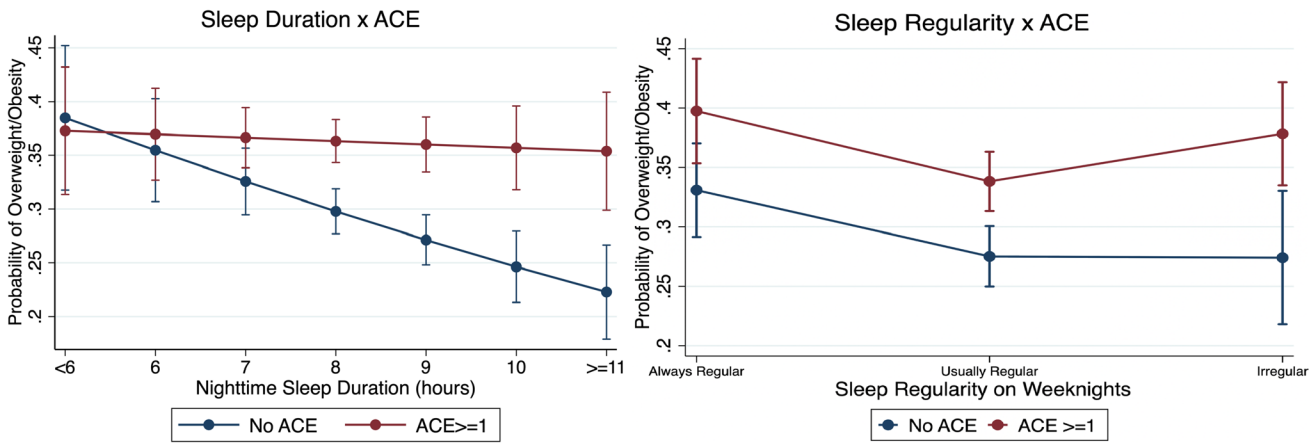

Fig. 1 Interaction between sleep duration/regularity and ACEs on overweight/obesity. We used complex survey design and the Margins command in Stata based on logistic regression to create the margins plots. The plots show when holding sleep duration/regularity (x-axis) constant at different values, what the probability of overweight/obesity is with 95% confidence intervals

trauma, and are obtaining adequate sleep, may have less allostatic load and therefore, are less likely to develop overweight/obesity. This juxtaposition between groups who face and do not face adversity further contributes to the growing disparities in obesity and health.

Approximately one-third of our sample was categorized as overweight/obese which aligns with national estimates of obesity [3]. Only half of our sample reported experiencing ≥ 1 ACE, which is less than the two-thirds prevalence reported nationally [10]. Similarly, a little over one-third of our sample was categorized as sleeping less than the age-recommended sleep duration, which is below the 58–73% reported nationally [12, 13]. Our data's underestimation of short sleep and ACEs may be due to parental report. Indeed, parents often overestimate their child's sleep duration [37] and may be less likely to report family dysfunction or may be unaware of all ACEs experienced by their child [8, 20, 21, 25]. Both parental report of sleep duration and ACEs may be subject to social desirability and/or recall bias. This implies that most likely more than one-third of this nationally representative sample slept less than the recommended age-appropriate hours and more than 50% experienced ≥ 1 ACEs.

Consistent with prior findings, adolescents with social risk factors are at a disproportionate risk for overweight and obesity [38]. There are a variety of explanations for such findings in our sample. Adolescents experiencing ≥ 1 ACEs may be growing up in a risky and stressful family environment, potentially contributing to chronic stress activation of the HPA axis and increasing allostatic load which may interfere with self-regulation of health behaviors, including sleep, and ultimately obesity development [4, 8, 22, 23]. Ethnic and minority families and those with low socioeconomic status are often forced by "redlining" (the historically discriminating housing policy) to live in disadvantaged neighborhoods [39]. Living in poor neighborhood conditions (i.e., rundown housing, lack of cleanliness, vandalism/graffiti) contributes to poor sleep health in adolescents [39]. Moreover, lack of access to green and safe spaces for physical activity or the presence of food desserts (lack of access to affordable and high-quality fresh food) in disadvantage neighborhoods may further contribute to overweight/obesity risk [40].

Our marginally significant finding that greater sleep duration was associated with decreased odds of overweight/obesity is consistent with prior work. Indeed, longitudinal research suggests that children who experience longer sleep duration on average (via parent-report and actigraphy) have slower growth in BMI scores and decreased risk of being overweight/obese in adolescence [30, 41]. Our finding that adolescents with regular sleep schedules did not have decreased odds of overweight/obesity was unexpected given that prior work has found sleep irregularity to be associated with obesity risk [6]. However, more recent work has found sleep duration to have a stronger effect than sleep irregularity on obesity development in adolescents. [42]

Study Limitations

The findings of our study must be interpreted in the context of its design and measurement limitations. The cross-sectional design prohibits the examination of causal relationships. As previously mentioned, the relationships between sleep, ACEs and overweight/obesity risk are nuanced and complex, and therefore require a variety of assessment methods over long periods of time [30]. Similarly, cross-sectional design inhibits the ability to examine intra-individual differences in BMI and sleep duration/irregularity over time [43], and the capture of social jetlag or seasonality differences in both sleep and BMI [26].

Concerning measurement limitations, using a single item to quantify sleep duration and irregularity on weeknights only, limits construct variability and may hinder its accurate measurement. Indeed, classifying sleep irregularity as the standard deviation in actigraphy or sleep diary captured sleep onset time over an extended period of time (including weekends) is more reliable than a one-item sleep regularity measure [6]. Moreover, other metrics of sleep health, such as sleep quality, may have stronger biological underpinnings of metabolic and endocrine systems related to weight, hunger and satiety, which could be affected by sleep and ACEs [12]. The parent-report nature of ACEs is also concerning for the following reasons: (1) parents may be less likely to report family dysfunction (possibly due to mandatory reporting of abuse), (2) parents may not know about all of adolescent's exposure to ACEs, (3) the questionnaire does not ask about timing, duration and severity of ACEs exposure, and (4) the person completing the survey and the relationship to the child may vary from family-to-family and result in inaccuracies [8, 20, 21, 25]. This implies that the 50% prevalence of ACEs in this sample is an underestimation. Further, the measurement of specific types of ACEs was not comprehensive. The NSCH survey did not include measures of emotional, physical or sexual abuse [26], dating violence nor peer-to-peer bullying which may be particularly prevalent in adolescence [20]. Moreover, there was a lack of behavioral data on other factors affecting weight status, such as dietary intake/quality and screen time/screen-based sedentary behaviors.

Provider Implications and Future Research

A recent American Academy of Pediatrics (AAP) Survey of Fellows revealed that few pediatricians ask about ACEs and 89% are unfamiliar with the original ACEs study [8]. Primary care is the ideal setting for ACEs screening as providers can build trust and rapport with families over

the frequent intervals they see and care for them. During this time providers can (1) screen children and families for ACEs, (2) offer empathetic and evidence-based anticipatory guidance, including providing education and interventions around sleep, healthy relationships, nutrition, mindfulness, physical activity and mental health, and (3) provide trauma-informed and family-centered resources and referrals when needed (which may be particularly necessary in families with low socioeconomic status) [4, 27]. Such screening and interventions may attenuate the negative impact of ACEs on health outcomes [26].

Similarly, the primary care setting is an ideal venue to screen for sleep problems and overweight/obesity development. The AAP recommends annual pediatric sleep screening, yet only a fraction of children seen for well-care visits are appropriately screened for sleep problems [44]. Pediatric providers are challenged with screening for a plethora of developmental issues when they have limited time with a growing number of patients [39]. Moreover, there are economic inequities in access to primary care [4]. As such, collaboration between interdisciplinary team members, including psychologists, social workers, school nurses, and educators may lead to more efficient identification of ACEs, unhealthy sleep and weight, particularly among low socioeconomic groups [39].

Once ACEs, sleep and/or weight problems are identified by interdisciplinary providers, culturally sensitive and family-centered interventions are needed. Intervention development would benefit from a clearer depiction of the nuanced associations between these concepts and potential underlying mechanisms. Specifically, future work may aim to understand why the protective effect of adequate sleep on overweight/obesity risk is lessened among adolescents experiencing ≥ 1 ACEs. Additionally, exploring how ACEs may mediate the relationship between sleep and obesity and seeing if intervening to improve ACEs mitigates long-term effects of ACEs and their associated negative impacts on weight management is warranted [26]. Finally, examining whether the effects of ACEs may vary based on the age the first ACE occurred and/or the chronicity of exposures and the co-occurrence of ACEs types across developmental periods may inform ideal intervention timing [20].

Identification of such mechanisms will be strengthened by proper capture of the concepts under exploration. Use of actigraphy (objective measure of sleep and wake time) over parental-report and one-item sleep measures is ideal for capturing sleep duration and regularity among community settings. Additionally, more standardization of the measure of ACEs is needed in healthcare and research venues alike [4]. Finally, although BMI is a standard measure for assessing obesity, especially in large samples, future research may benefit from a more detailed evaluation of central adiposity or percent body fat [22], and expanded measures of physical health, such as blood pressure and blood serum markers of cardiovascular risk (e.g., triglyceride levels, insulin resistance) [12].

Successful interventions may need to focus on the challenges of changing health-promoting behaviors in stressful contexts [25]. Often, ACEs interventions emphasize ensuring child safety, addressing mental health effects of ACEs and implementing trauma-informed practices. In contrast, obesity interventions customarily focus on physical activity, diet quality and other health behaviors; while sleep interventions focus on education and sleep routines. Thus, there is an untapped potential for integration of ACEs-sleep-and-obesity-focused interventions. Given the high prevalence of ACEs and inadequate sleep in adolescents, amending the sleep-obesity relationship may require a trauma-informed approach [4].

Conclusions

Given the disproportionate risk of poor sleep and obesity among adolescents facing adversity, our findings extend prior work by uncovering how ACEs interacts with sleep on overweight/obesity among a national sample of U.S. adolescents. Longer sleep is associated with lower risk of overweight and obesity among adolescents who experience no ACEs compared to adolescents who experience ≥ 1 ACEs. Upon verification of these findings longitudinally, future work is needed to explore additional mechanisms for obesity development that will inform meaningful and culturally sensitive interventions for adolescents living within adverse environments. Interdisciplinary research and healthcare teams are particularly poised to move this agenda forward and enact trauma-informed care to address sleep and weight problems among adolescents facing adversity.

Author Contributions Lauren Covington conceptualized and drafted the initial article, assisted with interpreting data, and reviewed and revised the article. Xiaopeng Ji conceptualized and assisted in drafting the initial article, completed data analysis and interpretation, and reviewed and revised the article. Janeese Brownlow conceptualized, assisted with the draft of the initial article, and reviewed and revised the article. Ming Ji assisted with data analysis and interpretation, and critically reviewed and revised the article. Freda Patterson conceptualized, reviewed, and revised the article. All authors approve the final article as submitted and agree to be accountable for all aspects of the study.

Funding Dr. Covington's work is supported by the Rockefeller University Heilbrunn Family Center for Research Nursing through the generosity of the Heilbrunn Family and the National Center for Advancing Translational Sciences, National Institutes of Health, through Rockefeller University, under Grant [# UL1 TR001866], and an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences of the National Institutes of Health under Grant [U54-GM104941] (PI: Hicks). Dr. Patterson's work is supported by the National Institute of Drug Addiction [R01DA051321],

the National Institute of Minority Health and Health Disparities [R01MD012734] and the National Institute of General Medical Sciences [2P20GM113125] of the National Institutes of Health.

Data Availability Data and materials to replicate the findings here are available at the following URL: https://www.childhealthdata.org/help/dataset. The analytic code to reproduce the analyses presented in this paper is available from the second author.

Declarations

Competing Interests The authors report there are no competing interests to declare.

Ethical Approval All procedures performed in studies involving human participants were in accordance with ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study was deemed exempt by University of Delaware Institutional Review Board.

Informed Consent Informed consent was not required by this secondary data analysis, which was deemed exempt by University of Delaware Institutional Review Board.

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