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Associations of Adverse Childhood Experiences with Adolescent Total Sleep Time, Social Jetlag, and Insomnia Symptoms

Darlynn M. Rojo-Wissar, MPH[a,*], David W. Sosnowski, PhD[a,*], Maggie M. Ingram, MHS[a], Chandra L. Jackson, PhD[b,c], Brion S. Maher, PhD[a], Candice A. Alfano, PhD[d], Lisa J. Meltzer, PhD[e], Adam P. Spira, PhD[a,f,g]

[a]Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

[b]Epidemiology Branch, National Institute of Environmental Health Sciences, National Institutes of Health, Department of Health and Human Services, Research Triangle Park, NC

[c]Intramural Program, National Institute on Minority Health and Health Disparities, National Institutes of Health, Department of Health and Human Services, Bethesda, MD

[d]Department of Psychology, University of Houston, Houston, TX

[e]Department of Pediatrics, National Jewish Health, Denver, CO

[f]Department of Psychiatry and Behavioral Sciences, Johns Hopkins School of Medicine, Baltimore, MD

[g]Johns Hopkins Center on Aging and Health, Baltimore, MD

Abstract

Study Objective: Adverse childhood experiences (ACEs) are associated with sleep problems in adulthood, but less research has focused on ACEs and sleep during adolescence. The goal of the present study was to explore associations between ACEs reported at ages 5 and 9 years, and sleep (i.e., total sleep time (TST), social jetlag, and insomnia symptoms) at age 15.

Methods: Participants comprised 817 families from the Fragile Families and Child Wellbeing Study, a nationally representative sample of children born to unwed parents. Number of ACEs was constructed from primary-caregiver reports at ages 5 and 9, and sleep measures (i.e., TST, social jetlag, and insomnia symptoms) were derived from adolescent-reported sleep behaviors at age 15.

Results: Adjusting for sex and race/ethnicity, ACEs at age 9 were associated with longer weekend TST (B = 0.16, 95% CI = 0.04, 0.28), more social jetlag (B = 0.17, 95% CI = 0.07, 0.27), and higher odds of trouble falling asleep ≥ 3 times per week (Odds Ratio = 1.24, 95% CI = 1.01, 1.53). In females only, ACEs were associated with greater school night TST (B = 0.12, 95% CI = 0.01, 0.23). Results were similar after further adjustment for symptoms of anxiety and

Address correspondence to: Darlynn Rojo-Wissar, Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, 624 North Broadway, Hampton House, Baltimore, MD, 21205, drojowi1@jhu.edu.
*indicates joint first authorship

depression. Associations among ACEs, social jetlag, and insomnia symptoms appeared strongest among Non-Hispanic Black adolescents.

Conclusion: ACEs appear to be related to multiple aspects of sleep in adolescence. Additional research is needed to confirm these associations and examine the extent to which sleep disturbances associated with ACEs account for later health outcomes.

Keywords

adverse childhood experiences; adolescence; sleep; insomnia; social jetlag

Introduction

Adverse childhood experiences (ACEs) refer to a set of potentially traumatic experiences that occur prior to age 18 and confer risk for negative health outcomes and health-risk behaviors. Data from the 2016 National Survey of Children's Health suggest that nearly half of all U.S. children (34 million) are exposed to at least one ACE, while more than 20 percent experience two or more ACEs.[1] Felitti et al. (1998) originally identified seven ACEs across domains of child maltreatment (psychological, physical, and sexual abuse) and household dysfunction (substance abuse, mental illness, violent treatment of mother, and criminal behavior in the household).[2] Subsequent data collection added assessments of physical and emotional neglect and parent separation or divorce.[3,4] Recent efforts have expanded upon the original ACE measure to include other developmentally relevant experiences such as peer victimization, low socioeconomic status, and parent death.[5] Regardless of which ACEs are measured, there is a consistent, robust association between ACEs and numerous health and behavior problems (e.g., internalizing problems, substance use) throughout life.[6,7]

Traditionally, ACEs are assessed using either a dose-response (i.e., categorical) framework (i.e., coding ACE exposure as 0 [ref], 1, 2, 3, or 4+), or a cumulative (i.e., continuous) scoring framework. Using a dose-response framework, Felitti et al. (1998) found that individuals who reported at least four ACEs had a four- to 12-fold increase in poor self-rated health compared to individuals reporting no ACEs. Similar results have emerged when researchers use a cumulative risk index. Cumulative exposure to ACEs is linked with future victimization (e.g., sexual victimization), health risk behaviors (e.g., alcohol consumption), chronic health conditions, adverse mental health outcomes (i.e., depression, anxiety, and suicidality), and premature mortality.[2,8–11] Moreover, evidence suggests that adversity experienced during childhood remains independently associated with poor health after accounting for adversity during adulthood,[12] and may sensitize individuals to maladaptive or harmful coping behaviors (e.g., substance use) in response to stress.[13] These findings underscore the deleterious effects of ACEs, and research on the shorter-term consequences of ACEs is needed to identify malleable targets for intervention to interrupt harmful trajectories.

Adverse childhood experiences are often assessed retrospectively in adulthood, reflecting lifetime exposure to adversity. Although ACEs assessed in adulthood have demonstrated robust associations with adult health outcomes,[7] recent investigations have focused on assessing ACEs occurring in childhood and adolescence.[14,15] These efforts to assess ACEs may not cover the child's lifetime exposure and may rely on parent reports of children's adversity, yet they allow for examination of shorter term consequences of ACEs. In doing so, researchers can identify malleable intervention targets that may underlie the development of later health problems. One such malleable target is sleep.

As with ACEs, sleep disturbances are associated with adverse physical and mental health outcomes and premature mortality.[16–20] However, little is known about the association of cumulative ACEs with sleep health, specifically during adolescence. Using a nationally representative sample of adolescents, Wang et al. (2016) found a dose-response relationship between number of childhood adversities reported and risk of insomnia disorder.[21] Other aspects of sleep that are known to be important during adolescence, such as sleep duration and social jetlag, were not examined. Furthermore, number of ACEs was assessed using a dose-response framework (0 [ref], 1, 2, 3, 4, 5+). Although this approach is valuable in identifying graded associations between ACEs and health, recent evidence suggests that modeling ACEs using a cumulative score performs better in models examining the relationship between ACEs and sleep.[22] Most studies that have examined associations between cumulative ACE exposure and sleep have focused on sleep in adulthood. These studies have found that a higher ACE score is associated with poorer self-reported sleep quality, more severe insomnia symptoms, and poorer sleep indices (e.g., wake after sleep onset, sleep onset latency, and sleep efficiency).[22–27]

Though the literature on the relationship between ACEs and sleep is still growing, several theoretical frameworks linking them have been proposed.[28–30] These conceptual models posit that various biopsychosocial mechanisms lead to poor sleep following exposure to early life stress, and these sleep disturbances may be maintained into adulthood. Potential mechanisms linking ACEs to sleep problems include: disruption to the stress response and circadian timing systems, increased neuronal arousal, and failure to develop healthy sleep habits (e.g., a regular sleep schedule) due to family/household disruption.[28–30] Various aspects of sleep may be affected, such as duration, timing, onset and maintenance. ACE-related health conditions that manifest later in development may also then exacerbate existing sleep disturbances in individuals with ACEs.[28–30] Some of these health conditions (e.g., psychiatric disorders), may emerge as a result of the interaction between stress-related disruptions in the stress response system, circadian timing system, and sleep.[31]

Although many chronic health consequences of ACEs materialize in adulthood, sleep problems may manifest earlier in development. Consequently, these sleep problems may further increase the risk of developing medical or psychiatric conditions later in life.[32] Understanding the short-term relationship between ACEs in early and middle-childhood and subsequent sleep in adolescence is of particular interest given that adolescence is a period of rapid developmental growth and change, including changes to the sleep and circadian systems.[33] Further, sleep problems among adolescents are common.[34–36] One change that occurs in adolescence, related to the circadian system, is a biological shift toward a preference for later sleep timing.[16] Paired with early school start times, this preference for later sleep timing may increase risk for insufficient sleep and social jetlag. Social jetlag can be defined as a sleep-timing misalignment between school nights and weekend nights.[37] Adolescents with ACEs may be at greater risk for experiencing these sleep disturbances as well as insomnia symptoms (i.e., trouble falling asleep) as a result of factors like stress-related hyperarousal.[28,38] However, adolescence represents a key developmental window in which intervention can have lasting effects on lifelong health.[33] Examining how cumulative ACE exposure is related to sleep in adolescence is an important first step in informing targeted sleep interventions for this population.

The current study aimed to reduce a notable gap in the literature concerning the relationship between ACEs in early and middle childhood and subsequent sleep in adolescence. We conceptualized cumulative exposure via a count of the following ACEs: parent substance use, parent mental or physical disability, parent incarceration, family violence, parent separation/divorce, parent death, child abuse (physical and psychological) and neglect, and poverty status. To capture the multifaceted nature of sleep health,[39,40] we examined sleep duration (during school and work days), regularity of sleep timing (via social jetlag), and sleep quality (i.e., trouble falling asleep or staying asleep). Based on prior work in adults,[22–27] we generally hypothesized that a greater number of ACEs reported by primary caregivers (PCG; through the age of 9 years) would be associated with poorer adolescent-reported sleep health. However, given the scarcity of research on ACEs and sleep during this important developmental period, we considered this study to be hypothesis-generating, and did not have more specific hypotheses regarding the sleep parameters with which ACEs would be associated or the direction of associations. Much of the research examining associations between ACEs and sleep has been limited by the inclusion of only females and little to no representation of racial/ethnic minorities.[29] Females have been shown to be more susceptible to poor outcomes related to ACEs,[29] and racial/ethnic minorities are at higher risk for both ACEs and poor sleep.[41,42] As a result, we also explored whether there were disparities in these relationships by sex or race/ethnicity. Previous research also suggests that the effects of ACEs may vary across development due to age-associated neurobiological changes.[43,44] Thus, we separately determined the association of ACEs reported at ages 5 (early childhood) and 9 years (middle childhood) to clarify the influence of age of ACE exposure on adolescent sleep outcomes.

Method

Participants and Procedure

The present study is a secondary analysis of publicly available, de-identified data from the Fragile Families and Child Wellbeing Study (FFCWS).[45] A detailed description of the original study sample, goals, and sampling methodology can be found in prior work.[45] Briefly, the FFCWS was conducted to characterize the conditions and capabilities of new unwed parents, particularly fathers. The original study sampled approximately 5,000 families, a majority of which were unwed couples. The sample is nationally representative of non-marital births occurring in large U.S. cities between 1998 and 2000. When applying survey weights, the sample is nationally representative of all births in large U.S. cities between 1998 and 2000. Sampling weights are used to obtain unbiased statistical estimates when a study's sample members were either not selected independently or not selected with equal probability. In the case of the Fragile Families Study, nonmarital births, racial/ethnic minorities, and low-income families were oversampled. Data were collected at the child's birth and follow-up interviews were conducted at ages 1, 3, 5, 9, and 15 years. The present analysis uses data from children for whom the PCG was a biological parent, sampling weights were available, and who had complete data on variables of interest (N = 817). Specifically, primary caregivers reported on ACEs at the Year 5 and Year 9 follow-up waves, and adolescents reported on their own sleep at the Year 15 follow-up wave.

Data collection for the Year 5 follow-up wave was conducted from 2003 through 2006. Biological parents completed a portion of the surveys via computer-assisted telephone interviews. In-home interviews were then conducted with the child and the PCG (i.e., the biological parent who had custody of the child half or more of the time). The Year 9 follow-up wave was conducted from 2007 through 2010. Biological parents completed a portion of the surveys (i.e., maltreatment and neglect surveys) at this wave via computer-assisted telephone interview. In-home assessments also were conducted, in which children completed surveys using computer-assisted personal interview technology while the PCG completed a self-administered, written questionnaire. Data collection for the Year 15 follow-up wave was conducted from 2014 through 2017. Only the focal child and their PCG were interviewed at this wave. The PCG and child surveys were administered via telephone, although some were completed at the home, or online in the case of severe scheduling challenges. Informed consent and child assent was obtained from all study participants. Descriptive information for study participants at all waves is presented in Table 1.

Measures

Adverse Childhood Experiences—Although no formal ACE measure was included in the FFCWS, we constructed a cumulative ACE score based on parent-reported items from the Year 5 and Year 9 follow-up waves, consistent with prior reports using these data.[14,46] All ACEs at Year 5 were available at Year 9, albeit with slight wording variation for some items. Supplemental Table 1 provides detailed information on the ACE items used in the present analysis, response options, and how items were scored to create each dichotomous ACE item. Adverse childhood experiences in the present study were comparable to those assessed in the original ACE Study[2] with the notable exception of this study not including data on child sexual abuse. Suspected sexual abuse reported by Child Protective Services was queried, however, data on whether these cases were confirmed are not available. In addition, most ACE items in our study reflect past-year parent reports of childhood adversity, as opposed to self-reported lifetime experiences. ACE scores were calculated at Year 5 and Year 9, separately. A combined score was also computed for both years, such that, if a child experienced a given ACE at either Year 5 or Year 9, they received a point toward their cumulative ACE score (i.e., number of adversities experienced up to age 9). Scores could range from 0-10.

Parent Substance Abuse.: Parent substance abuse was assessed at Year 5 and Year 9 via two items asking each parent if their use of alcohol or other drugs interfered with work at school, home, or their job within the past 12 months. Item responses were coded as 0 (*no*) and 1 (*yes*). A response of *yes* to either item by either parent counted as a point toward the child's ACE score.

Parent Disability.: Parent disability was assessed at Year 5 and Year 9 via two items. The first item, constructed by the FFCWS investigators, indicated whether or not each parent had experienced a Major Depressive Episode in the past year using the Composite International Diagnostic Interview Short Form (CIDI-SF).[47] The CIDI-SF has been used in past studies and categorizes respondents as having experienced a depressive episode in the past year based on DSM-IV criteria. The second item asked each parent whether they had a psychological or physical condition that affected the type or amount of work they could do. Item responses for both items were coded as 0 (*no*) and 1 (*yes*). A response of *yes* to either item counted as a point toward the child's ACE score.

Parent Incarceration.: Parent incarceration at Year 5 was assessed via a single item asking each parent if the other parent had spent time in jail during the last two years. Mother incarceration at Year 9 was assessed via a single item asking fathers if the mother had spent time in jail during the past four years. Father incarceration at Year 9 was assessed via a single item asking mothers if the father had spent any time in jail since the study's inception. Item responses were coded as 0 (*no*) and 1 (*yes*). A response of *yes* from either parent counted as a point toward the child's total ACE score.

Family Violence.: Family violence at Year 5 was assessed via a single item asking mothers if the child had witnessed a physical fight between her and the father within the past two years. Family violence at Year 9 was assessed via a single item asking mothers if the father ever hurt them in front of the child in the past four years. Item responses were coded as 0 (*no*) and 1 (*yes*).

Parent Separation/Divorce.: Parent separation/divorce at Year 5 and Year 9 was assessed via a single item asking each biological parent what their current relationship is with the child's other biological parent. Responses were used to create codes of 0 (*separated or divorced*) and 1 (*married or living together romantically*).

Parent Death.: Death of either biological parent at Year 5 and Year 9 was determined using a single item asking each biological parent what their current relationship is with the child's other biological parent. Responses were used to create new codes of 0 (*no death of biological mother or father*) and 1 (*death of a biological mother or father*).

Child Abuse and Neglect.: Past-year physical and psychological abuse and neglect were assessed using a subset of items from the Parent Child Conflicts Tactics Scale (CTSPC).[48] The child's PCG responded to five items about physical abuse (e.g., "shook him/her"), five items about psychological abuse (e.g., "called him/her dumb or lazy or some other name like that") and five items about neglect (e.g., "Were not able to make sure your child got the food he/she needed"). Response options range from 0 (*never*) to 6 (*20 or more times*). Similar to prior work with these data,[49] and to mirror the original ACE measurement,[2] midpoints were calculated for each response option and scores on each construct summed for separate total scores for physical abuse, emotional abuse, and neglect. Next, individuals in the top 10th percentile of total scores on each construct were categorized as abused or neglected and received a point toward their total ACE score.[49]

Poverty Status.: At the Year 9 follow-up wave, parents provided an exact dollar amount of their total household income. Poverty status was then calculated by taking the ratio of total household income to the official poverty thresholds established by the U.S. Census Bureau for the previous year, which accounts for family size, composition, and age of the householder. The resulting categories were: 0-49% (*severe poverty*), 50-99% (*in poverty*), 100-199% (*near poverty*), 200-299%, and +300% (*above the poverty level*). Responses were coded such that those individuals whose household annual income that was at least 200% above their poverty threshold were coded as 0 (*not living in poverty*) and all others were coded as 1 (*living in or near poverty*), consistent with the U.S. Census Bureau's definition of low-income families.[50]

Sleep—During a single interview at Year 15, adolescents reported their usual bed and wake times on school days and weekend days. Consistent with previous analyses of these sleep data,[51,52] we calculated school and weekend night TST as the interval between reported habitual bed and wake times in hours. Given known differences in TST on school days versus weekend days in adolescence as a result of school schedules,[53] school TST and weekend TST were each assessed as separate outcomes. To calculate social jetlag, we subtracted the midpoint of sleep on school days from the midpoint of sleep on weekend days and took the absolute value.[37,54] Adolescents also reported the number of nights during the week they have problems falling asleep and the number of nights they have problems staying asleep. Trouble falling asleep and trouble staying asleep were each dichotomized as 0 (< 3 times per week) and 1 (≥ 3 times per week). These two symptoms were examined individually, given past research suggesting a stronger association of childhood trauma exposure[55] and incident depression[56] with trouble falling asleep.

Depressive Symptoms—Adolescent depressive symptoms during the past month were assessed at Year 15 using a subset of five items from the Center for Epidemiological Studies Depression Scale (CES-D).[57] Response options range on a scale from 1 (*strongly agree*) to 4 (*strongly disagree*). Although only five items from the CES-D were available, evidence supports the validity of this subset of items in measuring children's depressive symptoms.[58] Responses were recoded so that higher scores reflect more depressive symptoms. Reliability for this scale in this sample was acceptable (α = .78).

Anxiety Symptoms—Adolescent anxiety symptoms during the past month were assessed at Year 15 using a subset of six items from the Brief Symptoms Inventory 18 (BSI 18).[59] A sample item is, "I feel nervous or shaky inside." Response options range on a scale from 1 (*strongly agree*) to 4 (*strongly disagree*). Responses were recoded so that higher scores reflect more anxiety symptoms. Although not all 18 BSI items were available for analysis, reliability for this scale in this sample was acceptable (α = .78).

Additional covariates—Adolescents reported on their own race/ethnicity via a single question during the Year 15 interview: "What is your race and ethnicity?" Interviewers recorded the adolescent's verbal response verbatim, up to 80 characters. Using these responses, the FFCWS investigators constructed an item with the following race/ethnicity codes: Non-Hispanic (NH) White; NH Black; Hispanic/Latino; some other NH race/

ethnicity; and NH multi-racial. The latter two groups were combined in the present study due to small sample sizes. Since adolescent sex and/or gender were not assessed during the Year 15 interview, we used mother's reports of the child's sex at the baseline interview (i.e., birth of the child). This practice is consistent with how other gender- and sex-related variables (e.g., body mass index) were constructed during this wave of data collection.

Data Analysis

Design-based analyses were used to account for the complex sampling design of the FFCWS, and to generate unbiased statistical estimates, using the *survey* software package in Stata version 15.0 (StataCorp, College Station, TX). Specifically, national basic and replicate weights adjusting for probability of selection, non-response at baseline, and observed characteristics associated with loss to follow-up were applied. Design-based descriptive characteristics were computed for those in our analytic sample with complete data on all variables of interest (N = 817). We also computed design-based descriptive characteristics for the entire sample that had sample weights (N = 2,532) and used t-tests and chi-square tests were to compare descriptive characteristics between those in our analytic sample and those excluded from analyses (N = 1,715 [Supplemental Tables 2 and 3]). Of the 2,532 participants with sample weights, 1,721 had a biological parent as their PCG. Of these 1,721 participants, 817 had complete data on our variables of interest. The subsample feature from the *survey* software package was used to identify our analytic sample in analyses.

To examine associations of ACEs through Year 9 with each continuous sleep outcome of interest at Year 15 (i.e., school TST, weekend TST, and social jetlag in hours), we fit linear regression models. To examine associations of ACEs through Year 9 with each binary insomnia symptom outcome of interest at Year 15 (i.e., trouble falling asleep, and trouble staying asleep), we fit logistic regression models. For each outcome, the following models were fit: Model 1, adjusted for sex and race/ethnicity; and Model 2, which further adjusted for depressive and anxiety symptoms at Year 15. This was done to evaluate the effect of ACEs on sleep beyond the effects of internalizing symptoms.

To investigate whether there were disparities in the associations of ACEs and sleep by sex or race/ethnicity, we tested interaction terms (including global Wald tests) and stratified results by sex and race/ethnicity. We did not rely solely on significant interaction terms because they are often underpowered and over-relied upon in research examining disparities.[60] It has been suggested that assessment of disparities should include the examination of differences in the prevalence of the outcome and exposure, as well as the effect size by group.[60] Thus, in addition to examining results stratified by sex and race/ethnicity, we also examined descriptive characteristics by sex and race/ethnicity, which are provided in supplemental tables (Supplemental Tables 4 and 5). Interaction terms were added to Model 1 for each sleep outcome of interest (i.e., ACE x sex, ACE x race/ethnicity).

Lastly, to determine the influence of developmental timing on sleep-related outcomes, all models were also run with ACEs reported at Year 5 and ACEs reported at Year 9 as independent predictors in separate models.

Results

The survey-weighted mean age for children at Year 5 was 5.06 years (*SE* = .02 years) and the majority (57%) were male (Table 1). The sample was racially and ethnically diverse, with 19% of children identifying as NH Black, 29% as Hispanic/Latino, 41% as NH White, and 11% as NH Other. A majority of mothers (~86%) and fathers (~81%) had a least a high school degree, and mothers' mean annual income (*M* = $59,334.04, *SE* = $3,333.40) was lower than fathers' mean annual income (*M* = $67,432.14, *SE* = $4,362.61) at baseline (i.e., Year 5). AverageTST for the analytic sample at age 15 was 8.17 hours on school nights, and 9.52 hours on weekend nights. Approximately 25% reported trouble falling asleep at least three times per week, 15% reported trouble staying asleep at least three times per week.

When comparing our analytic sample to those excluded from analyses, mothers in the analytic sample reported a significantly higher annual household income compared to mothers excluded (Supplemental Table 2); children in the analytic sample had fewer ACEs compared to children excluded from analyses; and children in the analytic sample reported a lesser amount of social jetlag compared to children excluded from analyses (Supplemental Table 3). Specifically, children in the analytic sample reported, on average, approximately 20 fewer minutes of social jetlag compared to those not included in analyses.

ACEs and Total Sleep Time

In the demographic-adjusted models for weekend TST, Year 9 ACEs were significantly associated with longer weekend TST (B = .16, 95% CI = .04, .28); each additional ACE reported at Year 9 was associated with approximately 10 minutes more of weekend TST (Table 2). This association remained significant after adjusting for anxiety and depressive symptoms at Year 15 (B = .17, 95% CI = .04, .29).

ACEs and Social Jetlag

In the demographic-adjusted models assessing social jetlag, there was a significant association between ACEs and social jetlag in the model with Year 9 ACEs as a unique predictor (B = .17, 95% CI = .07, .27); each additional ACE at Year 9 was associated with approximately 10 additional minutes of social jetlag (i.e., 10 minute difference between average weekend and school day midpoint of sleep [Table 2]). Results were identical when accounting for Year 15 anxiety and depressive symptoms (B = .17, 95% CI = .07, .27).

ACEs and Insomnia Symptoms

In the demographic-adjusted models, there was a significant association between the combined Year 5 and Year 9 ACE score and trouble falling asleep, OR = 1.22, 95% CI = 1.01, 1.47, such that each additional ACE was associated with a 22% increased likelihood of children reporting trouble falling asleep ≥ 3 times per week (Table 2). Similar results were found with the model using Year 9 ACEs as the primary predictor (OR = 1.24, 95% CI = 1.01, 1.53); however, neither of these associations was significant after accounting for Year 15 anxiety and depressive symptoms. No significant associations were detected in models assessing the association between ACEs and trouble staying asleep.

Examination of Potential Disparities by Sex and Race/Ethnicity

Survey-adjusted descriptive characteristics stratified by sex and race/ethnicity are available in Supplementary Tables 4 and 5. Females had a significantly higher CES-D score (M = 8.52, SE = .46) compared to males (M = 7.08, SE = .19; p = .005 [Supplementary Table 4]). Females were also significantly more likely to report trouble falling asleep at least three times per week (36%) compared to males (17%, p = .026). Several differences in demographic, ACE and sleep characteristics by race/ethnicity were present (Supplemental Table 5). Mother and father education levels and annual income were significantly higher for Non-Hispanic White participants compared to racial/ethnic minority participants. With regard to our main variables of interest, Non-Hispanic White participants also had significantly lower ACE scores and less social jetlag.

ACEs and Total Sleep Time—In the demographic-adjusted models for school day TST, there was a significant interaction between ACEs and sex in both the combined Y5 and Y9 ACE score model (Wald test for overall interaction: F(1,32) = 5.08, p = .031), and the model using Year 9 ACEs as an independent predictor (Wald test for overall interaction: F(1,32) = 10.64, p = .003). Results stratified by sex are presented in Table 3. Stratified analyses revealed a significant association between the Year 9 ACE score and school TST in females only (Model 1: B = .12, 95% CI = .01, .23); each additional ACE at Year 9 was associated with approximately seven minutes of more school day TST. This association in females remained after controlling for Year 15 anxiety and depressive symptoms (B = .14, 95% CI = .02, .26). Although stratified results using the combined Year 5 and Year 9 ACE score did not reveal significant associations among males or females, the combined ACE score was associated with increased school day TST in females (~ 6 minutes more TST) and decreased school day TST in males (~ 5 minutes less TST). Results were similar after adjusting for Year 15 anxiety and depressive symptoms. There were no significant interactions by race/ethnicity or significant associations between ACEs and TST among any racial/ethnic groups when stratifying by race/ethnicity (Table 4).

ACEs and Social Jetlag—There were no significant interactions of ACEs with sex or race/ethnicity. In analyses stratified by sex, Year 9 ACE score was associated with an additional ~10 minutes of social jetlag for each additional ACE for both males (B = .17, 95% CI = .01, .32) and females (B = .17, 95% CI = .02, .32) in Model 1 (Table 3). These associations were no longer significant in Model 2. When stratifying by race/ethnicity, there was a significant association between the combined Year 5 and Year 9 ACE score and social jetlag among Black adolescents (B = .24, 95% CI = .02, .47); each additional ACE was associated with 14 additional minutes of social jetlag (Table 4). This association remained after accounting for Year 15 anxiety and depressive symptoms. Similar results were detected in the model using the Year 9 ACE score as an independent predictor (B = .26, 95% CI = .02, .52), although this association was not significant after accounting for Year 15 anxiety and depressive symptoms. Lastly, there was a significant association in the model using the Year 9 ACE score as an independent predictor and social jetlag among White adolescents (B = .16, 95% CI = .04, .28); each additional ACE was associated with an additional 10 minutes of social jetlag. This association was not significant after accounting for Year 15 anxiety and depressive symptoms.

ACEs and Insomnia Symptoms—There were no significant interactions of ACEs with sex or race/ethnicity. When stratifying by sex, there was a significant association between Year 9 ACE score and trouble falling asleep among males only, which held in Model 2 (OR = 1.47, 95% CI = 1,09, 1.97 [Table 3]). When stratifying by race/ethnicity, several notable differences were observed. There was a significant association between the both the Year 5 and Year 9 ACE score and trouble falling asleep among Black adolescents. Each additional Year 5 ACE was associated with an 85% increased odds of Black adolescents reporting trouble falling asleep ≥ 3 times per week (OR = 1.85, 95% CI = 1.07, 3.19). Similarly, each additional Year 9 ACE was associated with a 48% increased odds of trouble falling asleep (OR = 1.48, 95% CI = 1.00, 2.18). Among White adolescents, there was a significant association between the Year 9 ACE score and trouble falling asleep (OR = 1.44, 95% CI = 1.02, 2.03) only in Model 2. Each additional ACE was associated with a 44% increased odds of White adolescents reporting troubling falling asleep ≥ 3 times per week. Lastly, there was a significant association between the Year 5 ACE score as an independent predictor and trouble staying asleep among Black adolescents (OR = 1.80, 95% CI = 1.10, 2.94). Each additional ACE was associated with an 80% increased odds of Black adolescents reporting trouble staying asleep. Among Black adolescents, none of these associations of ACEs with insomnia symptoms remained significant after accounting for Year 15 anxiety and depressive symptoms in Model 2.

Post Hoc Analyses

To aid in evaluation of the magnitude of all observed effects, we also report descriptive characteristics of sleep outcomes by median ACE score for the full sample (Supplemental Table 6), stratified by sex (Supplemental Table 7), and stratified by race/ethnicity (Supplemental Table 8). We used the Year 9 ACE score since it tended to have the most significant associations with sleep. The median Year 9 ACE score was 1 for the full sample, males, females, and NH White participants. For NH Black participants, the median Year 9 ACE score was 2. Since there were no significant associations of ACEs with sleep for Hispanics or NH Other participants, values were not reported for these groups.

Since ACE exposure over time is correlated, we also conducted post hoc analyses in which ACE scores at age 5 and 9 were entered into models simultaneously. This was done only in the case of significant findings for Model 1 and/or 2, to determine if observed associations between age 9 ACE scores and sleep held when controlling for ACEs at age 5. Among the full sample, findings held for weekend TST and social jetlag. For analyses by sex, associations for trouble falling asleep held for males, and associations for school TST held for females in model 2. For analyses by race, the social jetlag findings held for NH White participants, but not NH Black participants.

Discussion

The current study builds upon an emerging literature on the association between cumulative ACEs and sleep problems[22–27] by focusing on sleep in adolescence. Adolescence represents a developmental period marked by significant biological, socio-emotional, and physical maturation, and has implications for a range of subsequent health and behavioral outcomes.[33] We found that ACEs experienced at or around age 9 were associated with increases in weekend TST, increased social jetlag, and greater odds of adolescents having difficulty falling asleep, but not staying asleep, in the full sample. When stratifying results by race/ethnicity, associations of age 9 ACE score with social jetlag and trouble staying asleep appeared to be stronger in NH Black participants compared to NH White participants. Further, ACE score at age 5 was associated with trouble falling asleep and trouble staying asleep among NH Black participants. When assessing these relationships by sex, ACEs at age 9 were associated with school night TST among females, trouble falling asleep among males, and social jetlag among both males and females. These results provide further evidence of the detrimental influence of ACEs on subsequent sleep health, and suggest that there may be important differences in these relationships by sex and race/ethnicity. Findings also underscore a need to understand which particular aspects of sleep are affected by early adversity, which will inform mechanistic research linking ACEs to sleep and health.

Aligning with a growing body of research, our results suggest the pre/peri-pubertal stage (i.e., age 9) of development may be uniquely sensitive to the effects of adversity. The pre-pubertal period is notably marked by significant changes in biological systems that may render the effects of ACEs on sleep more severe and/or enduring.[61] Indeed, a growing body of evidence reveals the timing of early adversity, trauma and/or abuse to be associated with differential effects on brain development.[62,63] In some cases, younger children (i.e., age 5) may be more resilient to some forms of ACEs, perhaps by virtue of their neurobiological immaturity. This is speculative, given certain types of early adversity, including childhood sexual abuse were not included in this study. Alternatively, it may be that the occurrence of fewer ACEs at age 5 account for lack of significant associations with sleep outcomes. Indeed, when stratifying results by race/ethnicity, ACEs at age 5 were associated with greater odds of both trouble falling asleep and staying asleep among Non-Hispanic Black participants only. Notably, NH Black participants, on average, experienced approximately 1 more ACE than NH White children at age 5. Our post hoc analyses appear to support this hypothesis in part. When controlling for ACEs at age 5, associations between ACEs at age 9 and sleep disturbances often became null, except in the case when mean ACE score at age 5 was low in the group being examined (e.g., for males, who had a lower mean ACE score than the total sample).

In the current study, ACEs were associated with approximately 10 minutes longer weekend TST per each additional ACE. This same association was observed for school TST in females only. These findings are contrary to previous studies in adults that have shown no significant association of ACEs with TST.[22,26] Of note, these previous studies did not examine whether sex differences were present. Though a 10 minute increase may by itself not appear clinically significant, in the context of the number of ACEs, this translates to a 30 minute difference in weekend TST for those with three ACEs compared to those with none. In our sample, this corresponds to approximately 14% (n = 158) of adolescents who experienced at least a 30 minute difference in weekend TST. It may be that ACEs are only associated with TST during adolescence (i.e., a period marked by both homeostatic and circadian sleep alteration), whereas later, they are more strongly associated with sleep quality and subjective reports of sleep disturbance. One study in children found that greater parental depressive symptoms and interparental conflict was associated with shorter child

TST.[64] Thus, the specific type of adversity–exposure may be important to consider in our results. Again, our study did not include assessment of childhood sexual abuse which is more common among girls than boys[65] and has specifically been linked with subsequent sleep disturbances.[66–68]

One explanation for our counterintuitive finding that ACEs were associated with longer TST is that our measure of sleep duration, which was derived from reports of bed and wake times, may be more reflective of time in bed versus sleep duration, which has been shown to be longer in adolescents previously exposed to trauma.[69] Thus, our findings that ACEs are associated with longer weekend TST among everyone and longer school night TST among females may actually reflect longer time spent in bed trying to sleep, and not longer sleep duration. Those who experience a longer time in bed or sleep duration may have a trauma response consistent with sickness behavior, where more time is spent in bed as a way to combat inflammation produced as a result of trauma exposure[70]. If we are capturing sleep duration and not time in bed, previous work has shown that psychological distress is associated with both short and long sleep in adults.[71] It may be that during this particular developmental period, there is more likely to be an association between ACEs and slightly longer sleep, however, this is speculative. In these contexts, our findings do not necessarily suggest that ACEs are associated with better sleep, but potentially a greater psychobiological need for rest and recovery. Additional research is needed to further understand the relationship between ACEs and adolescent sleep duration.

To our knowledge, no previous studies have examined the relationship between ACEs and social jetlag. In the current study, we found that each unit increase in ACE score was associated with approximately 10 more minutes of social jetlag among adolescents, which translates to an individual with three ACEs having 30 more minutes of social jetlag compared to an adolescent with no ACEs. Previous studies in the FFCWS found that more social jetlag was associated with greater symptoms of anxiety,[52] and among females, greater depressive symptoms. [51] Other studies have linked social jet lag with various health problems and poor health behaviors (e.g., cardiovascular disease, substance abuse).[37,72] Examining associations between ACEs and social jetlag will therefore be an important target for additional research among adolescents.

Our findings that ACEs are associated with adolescent insomnia symptoms is consistent with previous research in adults[23–25] and children.[21] Of the insomnia symptom outcomes examined, ACEs were only associated with trouble falling asleep among the full sample. This makes sense because problems falling asleep are more common than trouble staying asleep during adolescence,[73] are more closely linked with trauma-exposure in childhood,[55] and have been identified as a primary target for depression prevention.[56] The association between ACEs and trouble falling asleep became non-significant when further adjusting for symptoms of depression and anxiety. This suggests that sleep may partially mediate the association of ACEs with mental health, or mental health may partially mediate association of ACEs with sleep.[24] Future prospective research would help elucidate the temporal relationship among ACEs, mental health symptoms, and sleep, and allow for formal tests of mediation. These associations may in part differ by race/ethnicity. For NH Black participants only, significant associations between ACEs at age 5 and trouble staying asleep emerged.

However, this association also became non-significant when further adjusting for symptoms of anxiety and depression.

The current study did not find any statistically significant differences (i.e., interaction terms) in the relationships between ACEs and sleep by race/ethnicity. However, examination of interaction terms on their own is not sufficient to assess health disparities.[60] When stratifying analyses by race/ethnicity, the magnitude of the effects of ACEs on social jetlag and all of the insomnia symptoms were magnified among NH Blacks and sometimes in the reverse direction of NH Whites. Non-Hispanic Blacks also had the highest number of ACEs, the most severe social jetlag, shortest TST, and the highest prevalence of trouble staying asleep. Additional research is needed to examine potential differences in the relationship between cumulative ACEs and sleep by race/ethnicity. Use of both subjective and objective measures will be critical, given previous findings of differential measurement error of sleep by race/ethnicity in adults.[74] More research is needed to understand measurement error across racial/ethnic groups among adolescents.

There may also be some disparities in the relationship between ACEs and sleep by sex. Though none of these group differences were statistically significant, females had higher ACE scores, shorter TST, and were more likely to report trouble staying asleep compared to males. Females were statistically significantly more likely to report trouble falling asleep (36%) compared to males (17%). In addition to the TST finding where ACEs at age 9 were associated with longer school night TST, stratified analyses revealed differential associations of ACEs at age 9 with trouble falling asleep. Though there was not a significant interaction of Year 9 ACE score with sex, ACEs at age 9 were only associated with a greater odds of trouble falling asleep among males in stratified analyses. More research is needed examining potential sex differences in the relationship between ACEs and sleep to clarify these associations.

Various biological, psychological, and social mechanisms can help explain how ACEs may influence sleep. Biologically, ACEs may lead to altered hypothalamic-pituitary-adrenal (HPA) axis functioning via increased allostatic load by increasing threat vigilance, and through prolonged stress response system activation over time.[75] In addition to alterations to the stress response system, alterations in the circadian timing system, as well as interactions between the two, may also lead to sleep disruption.[28–30] Children with ACEs may also be less likely to have optimal parental bonding/attachment styles,[76] which has been shown to be associated with sleep problems.[77,78] Feelings of warmth and safety/security generated from optimal parent bonding allows for adolescents to downregulate vigilance and threat response systems, facilitating sleep.[79] Parents of children with cumulative ACEs may also be less likely to have set bedtime routines and schedules, which have been associated with better adolescent sleep.[80] One ACE examined in the current study involves low socio-economic status, which may be related to increased sleep disturbances in children in part through disruptive environmental conditions (e.g., noise, light, and uncomfortable temperature levels).[81] Internalizing symptoms resulting from ACEs can also lead to worse sleep, though the literature suggests that sleep problems most often precede internalizing symptoms in adolescence.[82] Lastly, early school start times may have an even greater negative effect on sleep (e.g., social jetlag) in adolescents with ACEs who may be particularly vulnerable to going to bed later due to stress-related hyperarousal and trouble falling asleep.

Strengths and Limitations

The current study has several strengths, the first of which is the use of sleep measures during adolescence. Although there is a growing literature on the association between ACEs and sleep, most of these studies involve retrospective reports of ACEs and adult sleep outcomes.[22–27] Examining sleep problems during adolescence allows us to better characterize the short-term effects of ACEs on sleep and thus identify specific sleep targets for intervention. Another strength of the present study is the use of a nationally representative sample. Although the FFCWS originally oversampled low-income, single parent households, the sample is racially and ethnically diverse, which allowed for testing of disparities by race/ethnicity. Conducting minority health research is necessary to advance our understanding of the mechanisms by which health disparities manifest and ways to reduce them.[83] This study benefited from a longitudinal design, which allows for a stronger test of causality compared to the more common cross-sectional ACE studies using adult retrospective reports.[84] Lastly, the examination of ACEs separately at age 5 and 9 allowed for testing of potential sensitive periods for experiencing adversity. As previous evidence suggests, the effects of ACEs may vary across child development.[43] However, the prevalence of ACEs at different ages may also contribute to their effects at different developmental periods.

The current study also has several limitations that should be considered when interpreting the findings. First, no formal ACE survey was administered in the FFCWS. We constructed an ACE scale reflecting common ACE constructs from parent-reported items, which may underestimate the occurrence of ACEs and their influence on child outcomes, given the child's subjective experience was not captured.[85] The cumulative ACE score may also underestimate the actual ACE score, since adversities in the past year at ages 5 and 9 were examined, and not adversities at any age through age 9. Further, additional adversity items not included in the original ACE survey, and not measured in the current study, may be more predictive of poor health outcomes for younger generations and populations.[5,86,87] The inclusion of additional items such as items assessing adversities such as peer victimization, parental job loss, and involvement in the child welfare system,[87] may yield stronger associations of ACEs with sleep outcomes. There are also some limitations to the sleep measures in the current study. Specifically, the TST measures were based on adolescent-reported bed and wake times, but may be more reflective of time in bed, thus likely overestimating time spent sleeping. In addition, we were limited to measures of insomnia symptoms that do not reflect a clinical diagnosis of insomnia. However, the increases in sleep problems and probability in endorsing insomnia symptoms seen in the present analysis align with prior evidence,[21,23–25] and suggest clinically significant deviations in sleep patterns. Missing data led to our analytic sample consisting of participants who had higher incomes, were slightly more highly educated, and more likely to be non-Hispanic White. Though we used sample weights that accounted for non-response at baseline and loss to follow-up, results may still be subject to selection bias. As a result, our findings may underestimate the effect of ACEs on sleep. Lastly, because we considered this study hypothesis-generating and did not adjust for multiple testing to avoid committing Type II error at this early stage of research, findings should be interpreted with caution and examined further in future confirmatory studies.[88,89]

Clinical Significance and Future Directions

Although prior research has focused on associations between ACEs and sleep in adulthood, the current study provides evidence that ACEs are associated with subsequent social jetlag, insomnia symptoms, and sleep duration in adolescence. Though these findings should be confirmed in future studies early interventions targeting sleep may help reduce downstream health consequences of ACEs. For example, trouble falling asleep, with which ACEs were associated in this study, has been identified as a target for preventing depression.[56,90] A recent review highlights research showing that cognitive behavioral sleep interventions improve not only sleep, but also anxiety and depression among adolescents and adults. Though they are bidirectionally related, sleep disruption tends to precede development of anxiety and depression in adolescence.[82] Delaying school start times, which has been shown to benefit virtually all adolescents,[91] may be particularly beneficial for adolescents with a history of adversity given their heightened risk for sleep problems.

Additional prospective research is needed that includes formal measures of sample-relevant ACE data, and both subjective and objective measures of sleep collected at multiple timepoints. These studies will confirm specific aspects of sleep that should be targeted in future intervention studies. We used a cumulative risk (i.e., the number of adversities experienced) approach to study the relationship between ACEs and sleep because it has been the prevailing approach when studying the effects of early life adversity on subsequent health outcomes, and has been useful for demonstrating the public health significance of ACEs.[92] However, it will also be important for future studies to begin to examine the relationship between distinct types of ACEs and sleep, which will advance our understanding of mechanisms linking ACEs to sleep and other health outcomes.[92] Another direction for future research is the extent to which sleep disturbances related to ACE exposure contribute to long term mental and physical health outcomes known to be associated with ACEs. Several studies suggest that sleep may partially account for known associations between ACEs and poor mental and physical health outcomes. However, prospective studies are needed in order to explore this relationship, as are studies in children.[24,93–95] Lastly, studies are needed examining whether sleep interventions that have been shown to prevent the development of depression and anxiety,[82] and improve symptoms of these conditions once they have already manifested,[96] are efficacious in groups exposed to cumulative ACEs.

Conclusion

More caregiver-reported ACEs during childhood were associated with greater social jetlag, greater odds of trouble falling asleep three or more nights per week, and longer weekend sleep duration at age 15. These findings suggest that sleep disturbances that are associated with ACEs may manifest early in development and contribute to the development of later health problems. Many observed associations appeared to be strongest in NH

Black participants. Future research should continue to evaluate potential disparities in the relationship between ACEs and sleep. Importantly, these sleep outcomes are malleable and responsive to intervention. Prospective confirmatory studies measuring population-relevant ACEs and subjective and objective sleep outcomes will clarify these associations and inform intervention efforts.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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The datasets were derived from sources in the public domain: http://www.fragilefamilies.princeton.edu

Abbreviations:

ACEs	Adverse childhood experiences
TST	total sleep time
FFCWS	Fragile Families and Child Wellbeing Study
PCG	primary caregiver
CIDI-SF	Composite International Diagnostic Interview Short Form
CTSPC	Parent Child Conflicts Tactics Scale
CES-D	Center for Epidemiological Studies Depression Scale
BSI 18	Brief Symptoms Inventory 18

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Table 1

Survey-adjusted descriptive characteristics, N = 817 [Mean ± standard error or n (weighted %)]

Sex	
Female	402 (43.16)
Male	415 (56.84)
Race/Ethnicity	
Non-Hispanic White	216 (40.95)
Non-Hispanic Black	317 (18.86)
Hispanic/Latino	213 (29.08)
Non-Hispanic Other	71 (11.10)
Mother Education	
< High School	144 (13.41)
High School or GED	158 (21.07)
Some College/Tech	338 (31.44)
College or Grad	177 (34.07)
Father Education	
< High School	166 (18.09)
High School or GED	190 (16.76)
Some College/Tech	298 (33.08)
College or Grad	159 (32.07)
Father annual income, dollars	67,432.14 ± 4,362.61
Mother annual income, dollars	59,344.04 ± 3,333.40
ACE score age 5 and 9 combined	1.99 ± 0.13
ACE score age 5	1.28 ± 0.10
ACE score age 9	1.48 ± 0.11
Depression score	7.70 ± 0.27
Anxiety score	10.53 ± 0.31
Social jetlag, hours	2.41 ± 0.11
School TST, hours	8.17 ± 0.11
Weekend TST, hours	9.52 ± 0.13
Trouble falling asleep	
No	596 (74.87)
Yes	221 (25.13)
Trouble staying asleep	
No	670 (84.93)
Yes	147 (15.07)

Table 2

Associations between ACEs and sleep in the full sample: Main effects

Sleep outcome	*ACE predictor*	Analytic sample (N = 817)			
		Model 1		**Model 2**	
		B	**95% CI**	**B**	**95% CI**
School TST	ACE score age 5 and 9 combined	−0.02 †	[−0.14,0.10]	−0.01	[−0.13,0.11]
	ACE score age 5	−0.01	[−0.18,0.16]	−0.01	[−0.17,0.16]
	ACE score age 9	−0.04 †	[−0.16,0.09]	−0.02	[−0.14,0.09]
Weekend TST	ACE score age 5 and 9 combined	0.09	[−0.01,0.19]	0.10	[−0.00,0.20]
	ACE score age 5	0.07	[−0.09,0.24]	0.08	[−0.09,0.26]
	ACE score age 9	0.16 *	[0.04,0.28]	0.17 **	[0.04,0.29]
Social jetlag	ACE score age 5 and 9 combined	0.09	[−0.02,0.19]	0.08	[−0.03,0.19]
	ACE score age 5	0.03	[−0.12,0.18]	0.02	[−0.13,0.17]
	ACE score age 9	0.17 **	[0.07,0.27]	0.17 **	[0.07,0.27]
		Model 1		Model 2	
		OR	**95% CI**	**OR**	**95% CI**
Trouble falling asleep	ACE score age 5 and 9 combined	1.22 *	[1.01,1.47]	1.17	[0.94,1.44]
	ACE score age 5	1.19	[0.83,1.71]	1.11	[0.78,1.59]
	ACE score age 9	1.24 *	[1.01,1.53]	1.20	[0.95,1.52]
Trouble staying asleep	ACE score age 5 and 9 combined	1.10	[0.91,1.33]	1.06	[0.88,1.28]
	ACE score age 5	1.28	[0.96,1.70]	1.22	[0.93,1.62]
	ACE score age 9	1.19	[0.95,1.49]	1.15	[0.90,1.47]

Note. Survey-adjusted linear and logistic regression models. Model 1: adjusted for sex and race/ethnicity; Model 2: adjusted for Model 1 covariates, depressive symptoms, and anxious symptoms.

* $p < .05$,

** $p < .01$,

*** $p < .001$;

†indicates a statistically significant interaction term of ACE score by sex ($p < .05$).

Table 3

Associations between ACEs and sleep, stratified by sex

Sleep outcome	*ACE predictor*	**Males (N = 415)**				**Females (N = 402)**			
		Model 1		**Model 2**		**Model 1**		**Model 2**	
		B	**95% CI**	**B**	**95% CI**	**B**	**95% CI**	**B**	**95% CI**
School TST	ACE score age 5 and 9 combined	−0.09†	[−0.24,0.05]	−0.09	[−0.24,0.06]	0.10†	[−0.08,0.28]	0.12	[−0.07,0.30]
	ACE score age 5	−0.07	[−0.32,0.18]	−0.07	[−0.33,0.18]	0.06	[−0.15,0.28]	0.08	[−0.15,0.31]
	ACE score age 9	−0.15†	[−0.31,0.01]	−0.15	[−0.30,0.01]	0.12*†	[0.01,0.23]	0.14*	[0.02,0.26]
Weekend TST	ACE score age 5 and 9 combined	0.04	[−0.11,0.19]	0.04	[−0.12,0.20]	0.19	[−0.05,0.43]	0.21	[−0.04,0.47]
	ACE score age 5	0.08	[−0.19,0.35]	0.08	[−0.20,0.35]	0.07	[−0.15,0.28]	0.09	[−0.15,0.33]
	ACE score age 9	0.10	[−0.06,0.26]	0.10	[−0.07,0.27]	0.24	[−0.05,0.54]	0.26	[−0.04,0.56]
Social jetlag	ACE score age 5 and 9 combined	0.11	[−0.04,0.25]	0.11	[−0.04,0.25]	0.04	[−0.16,0.24]	0.03	[−0.19,0.24]
	ACE score age 5	0.10	[−0.11,0.31]	0.10	[−0.11,0.31]	−0.08	[−0.32,0.17]	−0.10	[−0.38,0.18]
	ACE score age 9	0.17*	[0.01,0.32]	0.16	[−0.00,0.33]	0.17*	[0.02,0.32]	0.16	[−0.00,0.32]
		Model 1		Model 2		Model 1		Model 2	
		OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Trouble falling asleep	ACE score age 5 and 9 combined	1.28	[0.97,1.71]	1.27	[0.94,1.70]	1.15	[0.81,1.63]	1.02	[0.68,1.52]
	ACE score age 5	1.17	[0.74,1.86]	1.14	[0.72,1.82]	1.21	[0.66,2.20]	1.04	[0.56,1.93]
	ACE score age 9	1.49**	[1.12,1.99]	1.47*	[1.09,1.97]	1.03	[0.76,1.39]	0.93	[0.63,1.37]
Trouble staying asleep	ACE score age 5 and 9 combined	1.13	[0.86,1.48]	1.11	[0.82,1.50]	1.07	[0.75,1.52]	1.02	[0.71,1.48]
	ACE score age 5	1.23	[0.91,1.67]	1.20	[0.85,1.68]	1.34	[0.76,2.34]	1.27	[0.75,2.14]
	ACE score age 9	1.33	[0.96,1.84]	1.31	[0.90,1.92]	1.02	[0.73,1.43]	0.97	[0.68,1.38]

Note. Survey-adjusted linear and logistic regression models. Model 1: adjusted for race/ethnicity; Model 2: adjusted for Model 1 covariate, depressive symptoms, and anxious symptoms.

* $p < .05$,

** $p < .01$,

*** $p < .001$;

† indicates a statistically significant interaction term of ACE score by sex ($p < .05$).

Table 4

Associations between ACEs and sleep, stratified by race/ethnicity

Sleep outcome	***ACE predictor***	**Non-Hispanic Blacks (N = 317)**				**Non-Hispanic Whites (N = 216)**			
		Model 1		**Model 2**		**Model 1**		**Model 2**	
		B	**95% CI**	**B**	**95% CI**	**B**	**95% CI**	**B**	**95% CI**
School TST	ACE score age 5 and 9 combined	−0.09	[−0.30,0.12]	−0.08	[−0.30,0.13]	0.13	[−0.10,0.35]	0.15	[−0.07,0.37]
	ACE score age 5	−0.12	[−0.36,0.11]	−0.12	[−0.36,0.13]	0.23	[−0.15,0.60]	0.22	[−0.13,0.58]
	ACE score age 9	−0.09	[−0.32,0.14]	−0.08	[−0.31,0.15]	0.00	[−0.17,0.17]	0.04	[−0.14,0.23]
Weekend TST	ACE score age 5 and 9 combined	0.09	[−0.16,0.33]	0.07	[−0.18,0.32]	0.15	[−0.04,0.35]	0.19	[−0.03,0.40]
	ACE score age 5	0.06	[−0.22,0.34]	0.01	[−0.31,0.33]	0.23	[−0.12,0.58]	0.24	[−0.11,0.59]
	ACE score age 9	0.11	[−0.15,0.38]	0.09	[−0.18,0.37]	0.11	[−0.10,0.31]	0.15	[−0.08,0.38]
Social jetlag	ACE score age 5 and 9 combined	0.24 *	[0.02,0.47]	0.24 *	[0.01,0.46]	−0.04	[−0.24,0.17]	−0.06	[−0.28,0.16]
	ACE score age 5	0.25	[−0.06,0.56]	0.25	[−0.05,0.55]	−0.16	[−0.44,0.11]	−0.17	[−0.42,0.09]
	ACE score age 9	0.26 *	[0.01,0.52]	0.26	[−0.00,0.51]	0.16 *	[0.04,0.28]	0.13	[−0.01,0.26]
		Model 1		Model 2		Model 1		Model 2	
		OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Trouble falling asleep	ACE score age 5 and 9 combined	1.43	[0.99,2.05]	1.34	[0.94,1.91]	1.33	[0.89,1.98]	1.30	[0.82,2.06]
	ACE score age 5	1.85 *	[1.07,3.19]	1.58	[0.95,2.64]	1.04	[0.59,1.83]	0.98	[0.54,1.75]
	ACE score age 9	1.48 *	[1.00,2.18]	1.39	[0.95,2.04]	1.32	[0.97,1.79]	1.44 *	[1.02,2.03]
Trouble staying asleep	ACE score age 5 and 9 combined	1.34	[0.86,2.10]	1.24	[0.80,1.92]	1.07	[0.65,1.76]	1.10	[0.68,1.80]
	ACE score age 5	1.80 *	[1.10,2.94]	1.50	[0.97,2.33]	1.30	[0.72,2.34]	1.31	[0.74,2.33]
	ACE score age 9	1.36	[0.82,2.26]	1.25	[0.74,2.10]	1.11	[0.68,1.82]	1.23	[0.80,1.91]
Sleep outcome	***ACE predictor***	**Hispanic/Latinos (N = 213)**				**Non-Hispanic Others (N = 71)**			
		Model 1		**Model 2**		**Model 1**		**Model 2**	
		B	**95% CI**	**B**	**95% CI**	**B**	**95% CI**	**B**	**95% CI**
School TST	ACE score age 5 and 9 combined	−0.08	[−0.29,0.13]	−0.07	[−0.29,0.15]	−0.07	[−0.38,0.25]	−0.06	[−0.35,0.23]
	ACE score age 5	−0.17	[−0.41,0.06]	−0.16	[−0.41,0.10]	−0.20	[−0.58,0.17]	−0.17	[−0.47,0.14]

Sleep outcome	***ACE predictor***	**Non-Hispanic Blacks (N = 317)**				**Non-Hispanic Whites (N = 216)**			
		Model 1		**Model 2**		**Model 1**		**Model 2**	
		B	**95% CI**	**B**	**95% CI**	**B**	**95% CI**	**B**	**95% CI**
	ACE score age 9	−0.07	[−0.39,0.25]	−0.06	[−0.36,0.25]	0.02	[−0.38,0.41]	0.02	[−0.34,0.37]
Weekend TST	ACE score age 5 and 9 combined	0.04	[−0.21,0.29]	0.06	[−0.22,0.33]	0.09	[−0.32,0.51]	0.10	[−0.32,0.51]
	ACE score age 5	−0.08	[−0.46,0.30]	−0.09	[−0.49,0.31]	−0.10	[−0.58,0.38]	−0.08	[−0.54,0.37]
	ACE score age 9	0.17	[−0.07,0.41]	0.21	[−0.06,0.48]	0.26	[−0.33,0.86]	0.26	[−0.33,0.86]
Social jetlag	ACE score age 5 and 9 combined	0.05	[−0.19,0.28]	0.04	[−0.20,0.29]	0.11	[−0.07,0.30]	0.12	[−0.03,0.28]
	ACE score age 5	−0.02	[−0.32,0.29]	−0.01	[−0.34,0.32]	0.14	[−0.09,0.36]	0.11	[−0.09,0.32]
	ACE score age 9	0.10	[−0.11,0.32]	0.10	[−0.14,0.34]	0.16	[−0.18,0.50]	0.18	[−0.10,0.45]
		Model 1		Model 2		Model 1		Model 2	
		OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Trouble falling asleep	ACE score age 5 and 9 combined	0.96	[0.66,1.39]	0.86	[0.53,1.40]	1.14	[0.70,1.85]	1.20	[0.71,2.04]
	ACE score age 5	0.87	[0.51,1.49]	0.79	[0.44,1.42]	1.32	[0.72,2.43]	1.33	[0.71,2.49]
	ACE score age 9	1.05	[0.63,1.77]	0.94	[0.49,1.80]	1.13	[0.49,2.59]	1.18	[0.47,2.95]
Trouble staying asleep	ACE score age 5 and 9 combined	0.91	[0.62,1.34]	0.83	[0.53,1.31]	1.18	[0.58,2.41]	1.28	[0.59,2.78]
	ACE score age 5	0.75	[0.47,1.19]	0.73	[0.43,1.23]	1.37	[0.50,3.75]	1.41	[0.43,4.58]
	ACE score age 9	1.08	[0.68,1.71]	0.99	[0.58,1.70]	1.25	[0.41,3.79]	1.34	[0.34,5.32]

Note. Survey-adjusted linear and logistic regression models. Model 1: adjusted for sex; Model 2: adjusted for Model 1 covariate, depressive symptoms, and anxious symptoms.

* $p < .05$,

** $p < .01$,

*** $p < .001$.