2021-06-04

Daytime Sleepiness Underlies the Link Between Adverse Parenting and Youth Psychopathology Among Adolescent Girls

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Recommended Citation

Sasser, J., Oshri, A., Duprey, E. B., Doane, L. D., & Peltz, J. S. (2021). Daytime sleepiness underlies the link between adverse parenting and youth psychopathology among adolescent girls. *Journal of Adolescence*, *90*, 32–44. https://doi.org/10.1016/j.adolescence.2021.05.010

Abstract

Introduction: Adverse parenting is associated with sleep problems in adolescence, including sleep quality, inadequate sleep, and daytime sleepiness. Adolescents who experience sleep problems are at greater risk for developing internalizing and externalizing problems. However, research on the intervening role of sleep in the link between adverse parenting and youth psychopathology remains limited. The present study aimed to examine the indirect effects of adverse parenting on youth internalizing and externalizing psychopathology via sleep problems, and to examine the moderating role of gender in associations between parenting and sleep. **Methods:** Participants were 101 low-income youth aged 9-12 (52.5% female; 75.2% African-American) and their primary caregivers. Families were from a non-metropolitan region in the Southeastern United States. Data were collected at two time points (T1; M_{age} = 10.28, *SD* = 1.2; T2; M_{age} = 12.08, *SD* = 1.2). Adverse parenting was measured at T1, youth-reported sleep problems (inadequacy, disturbance) and daytime sleepiness were assessed at T2, and parent-reported internalizing and externalizing symptoms were measured at T2. **Results:** Daytime sleepiness served as an intervening variable in associations between adverse parenting and internalizing and externalizing problems, but sleep problems did not. This indirect association was moderated by gender, such that the association between adverse parenting and daytime sleepiness only emerged as significant for girls. **Conclusions:** These findings suggest that daytime-related sleep behaviors may serve as a mechanism through which harsh or neglectful parenting is related to internalizing and externalizing psychopathology in adolescence, particularly for adolescent girls.

Keywords: Sleep Problems, Daytime Sleepiness, Adverse Parenting, Psychopathology, Gender

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Adverse rearing environments, characterized by harsh and neglectful parenting, are a significant form of early-life stress associated with the development of sleep problems (McPhie, Weiss, & Wekerle, 2014; Turner et al., 2020) and psychopathology in adolescence (Bender et al., 2007; McKee et al., 2007). Sleep, a key bioregulatory mechanism that promotes essential cognitive and emotional functions, is particularly important in adolescence (Dahl & Lewin, 2002; Galván, 2019). Sleep problems, including sleep disturbance, inadequacy of sleep, and daytime sleepiness, increase in prevalence during this time period (Sadeh & Gruber, 2002), and youth who report sleep problems are at heightened risk for a range of mental and physical health disorders (Zhang et al., 2017). Research suggests that the family context plays a crucial role in youths' sleep behaviors (El-Sheikh & Kelly, 2017), with stressful home environments resulting in greater sleep problems. Thus, it is plausible that sleep may underpin the connection between adverse parenting and internalizing (e.g., depressive, anxious) and externalizing (e.g., disruptive, aggressive) psychopathology in adolescence. Further, this connection may vary by gender, as emerging research suggests that the effect of adverse parenting on adolescent sleep problems is particularly evident in girls (Xiao et al., 2019). The present study investigated the indirect effect of adverse parenting on internalizing and externalizing behaviors via youths' sleep problems and daytime sleepiness, and examined the moderating role of gender in the link between parenting and sleep.

Adverse Parenting and Youth Psychopathology

The developmental psychopathology perspective focuses on developmental processes and mechanisms that underlie typical and atypical psychological outcomes (e.g., psychopathology;

Cicchetti & Rogosch, 2002). Based on this view, stress related to adverse parenting can disrupt normative bioregulatory and socioemotional developmental processes, ushering in risk for psychopathology in adolescence (Toth & Cicchetti, 2013). Youth who experience adverse parenting during early adolescence, a formative period that sets the stage for future transitions throughout adolescence, may be particularly vulnerable to developing internalizing and externalizing psychopathology (Bender et al., 2007; McKee et al., 2007; Oshri, Rogosch, & Cicchetti, 2013). For example, prior work suggests that changes in parent-child relationships across the transition to adolescence predict problem behaviors (e.g., internalizing symptoms) later in adolescence (Ebbert, Infurna, & Luthar, 2019). Importantly, normative physiological changes that accompany the transition to adolescence (e.g., pubertal maturation) render this a sensitive period for the development of psychopathology, in particular among youth who experience adverse parenting (Cicchetti, 2015). Although other physiological mechanisms have been examined as potential underlying mechanisms in the link between adverse parenting and problem behaviors (e.g., cortisol patterns and regulation; Stroud, Chen, Doane, & Granger, 2019; Duprey, Oshri, Liu, Kogan, & Caughy, 2020), less research has examined whether regulation of sleep across development may serve as a mechanism linking adverse parenting and the etiology of internalizing and externalizing problems (Kelly, Marks, & El-Sheikh, 2014).

Adverse Parenting and Sleep Problems

Sleep is a stress-sensitive state that is particularly vulnerable to the experience of early-life stressors like adverse parenting (Kajeepeta, Gelaye, Jackson, & Williams, 2015). Youth who are raised feeling unsafe and/or experiencing perceptions of threat at home may experience vigilance and arousal that can disrupt sleep (Dahl & Lewin, 2002). Indeed, a large body of evidence suggests that adverse parenting (e.g., child maltreatment) predicts sleep disturbances in

adolescence (McPhie et al., 2014; Noll, Trickett, Susman, & Putnam, 2006; Turner et al., 2020). In their longitudinal study, McPhie et al. (2014) found that severity of childhood maltreatment was a significant predictor of sleep issues in adolescence, including both nighttime sleep problems (e.g., waking up during the night) and daytime dysfunction. Notably, a recent cross-sectional study of nearly 3,000 adolescents ages 14-17 found that multiple types of maltreatment were associated with greater sleep latency, waking more often during the night, and sleeping fewer hours on weekdays (Turner et al., 2020). Despite the robust evidence for the impact of child maltreatment on sleep problems in adolescence, less is known regarding the effects of adverse parenting (e.g., occasional use of physical punishment, child-directed verbal aggression) on sleep in community samples of low-SES and largely minority youth and their families. Further, the connection between adverse parenting and related sleep problems in the development of youth psychopathology highlights an important area of investigation due to the relatively high prevalence of dysfunctional and adverse parenting.

Sleep and Adolescent Psychopathology

Adolescents who experience sleep problems are at an increased risk for adjustment problems, including internalizing and externalizing behaviors (Coulombe, Reid, Boyle, & Racine, 2010; Zhang et al., 2017). In the context of stressful life events, sleep may be viewed as a bioregulatory mechanism that precedes or explains youths' emotional and/or behavioral response to stress (El-Sheikh, Buckhalt, Mize, & Acebo, 2006; Kelly et al., 2014). Moreover, the co-occurrence of sleep problems across multiple mental health disorders provides evidence for sleep as a potential transdiagnostic process (i.e., a mechanism relating to the development of multiple disorders) underlying the development of youth psychopathology (Harvey, Murray, Chandler, & Soehner, 2011). Although prior literature suggests a bidirectional link between sleep

and youth problem behaviors (Kelly & El-Sheikh, 2014), there is evidence to suggest that these pathways may begin with sleep. Pieters et al. (2015) found that adolescent sleep problems were associated with both internalizing and externalizing behaviors a year later, but problem behaviors were not predictive of changes in sleep problems. Hence, a common mechanism, such as sleep, might explain the multifinality (i.e., diversity) of psychopathology outcomes associated with adverse parenting. Given established associations between multiple sleep indicators and adolescent psychopathology, including internalizing and externalizing problems (Coulombe et al., 2010; Kelly & El-Sheikh, 2014; Shimizu, Gillis, Buckhalt, & El-Sheikh, 2020), we predict that sleep problems (e.g., inadequacy/disturbance) and daytime sleepiness will both serve as mechanisms underlying the link between harsh parenting and internalizing and externalizing behaviors.

Sleep as a Mechanism in the Link Between Adverse Parenting and Psychopathology

The hypothesis that sleep may serve as an *intervening* mechanism in the pathway from adverse parenting to psychopathology, rather than a by-product of stress alongside psychopathology, is further supported when considering the role of sleep in emotion regulation. Substantial evidence supports the idea that sleep is a natural restorative process that may help youth cope with stressors and regulate their emotions (Dahl & Lewin, 2002; Palmer & Alfano, 2017). Further, sleep is a dynamic state that can be influenced by the daily experiences of adolescents, including stressful (e.g., demands from family, peers, school; Fuligni & Hardway, 2006) as well as positive experiences (e.g., interactions with parents; Peltz, Rogge, & Connolly, 2020; Sasser, Lecarie, Park, & Doane, 2020). These experiences can lead to changes in both daily and overall sleep health, and consequently influence psychological well-being (e.g., anxiety, depressive feelings; Fuligni & Hardway, 2006). Thus, less healthy sleep that occurs as a

result of adverse parenting may reduce youths' abilities to successfully regulate emotions and in turn lead to the development of internalizing and externalizing behaviors. Indeed, a study by Kelly and colleagues (2014) found evidence for sleep continuity/quality (i.e., efficiency, wake episodes) as a mechanism in the link between parent-child conflict (e.g., physical aggression) and youths' internalizing and externalizing problems. Further studying the intermediate role of sleep may help elucidate associations between adverse parenting and problem behaviors, while also pinpointing a potential area of intervention for youth enduring family stress.

Adverse Parenting, Sleep, and Psychopathology in Low-SES Families

Youth who experience poverty may be particularly at-risk for the experience of adverse parenting, sleep problems, and the development of psychopathology (Bradley & Corwyn, 2002; Doane et al., 2019; El-Sheikh et al., 2013; Farrell et al., 2017). Structural barriers, including a general lack of resources and increased levels of stressors, may underlie the heightened frequency of adverse parenting in low-SES families. Importantly, research has demonstrated a "biological embedding of poverty" (Doan & Evans, 2018), wherein lower socioeconomic status is linked with chronic psychological stress (e.g., allostatic load) and related self-regulation problems (Oshri et al., 2019). Thus, whereas youth who are reared in low-SES families may be at greater risk for negative health outcomes generally, family stressors like adverse parenting may serve as a potential pathway explaining the development of these outcomes, including disturbed sleep and psychopathology (e.g., Devenish, Hooley, & Mellor, 2017; Philbrook et al., 2020). Therefore, in our present study, we examined the associations between adverse parenting, sleep, and youth psychopathology in a low-SES sample of families.

Gender Differences in the Association between Adverse Parenting and Sleep

Because the transition into adolescence is a critical period of development during which gender differences in sleep patterns emerge (Laberge et al., 2001; Lee, Mcenany, & Weekes, 1999), it is important to consider gender in studies examining adverse parenting, sleep, and youth psychopathology. Although results are varied, some evidence suggests that adolescent girls are at greater risk for sleep problems, including lower quality sleep, less sleep duration, and greater daytime sleepiness when compared to males (de Matos et al., 2019; Matthews, Hall, & Dahl, 2014; Organek et al., 2015). These differences may be due in part to earlier pubertal timing, which has been linked with sleep-wake behaviors primarily among girls (e.g., delayed sleep schedule; shorter sleep duration; Foley et al., 2018; Hoyt et al., 2018). These findings suggest that preadolescent girls may be particularly vulnerable to sleep issues during this developmental period, and that gender differences may be present in the effect of early-life stress on adolescent sleep problems. Indeed, a recent study of over 153,000 Chinese adolescents found stronger associations between child maltreatment and sleep disturbances for girls than for boys (Xiao et al., 2019), providing evidence for potential gender differences in the link between adverse parenting and sleep problems in adolescence.

The Present Study

The present study investigated the intervening role of sleep in associations between adverse parenting and youth internalizing and externalizing psychopathology, and examined the moderating role of gender in the link between parenting and sleep (Figure 1). With the exception of one study (Kelly et al., 2014), no prior studies to our knowledge have examined sleep as an intervening process in associations between adverse parenting and youth psychopathology. Further, this is the first study to examine these research questions in a predominantly African-American sample (~75%) of youth from impoverished families (i.e., income at or below 200% of

the federal poverty level), a traditionally underserved community. These research aims are particularly salient for this population, as they experience increased family stressors, worse sleep, and heightened rates of psychopathology (Bradley & Corwyn, 2002; Doane et al., 2019; El-Sheikh et al., 2013). Importantly, though a large body of literature shows that childhood maltreatment impacts sleep in adulthood (i.e., Kajeepeta et al., 2015), less has been done empirically on this link during adolescence. This is a critical gap in the literature given that sleep in adolescence has important neurobiological functions and is linked with a range of mental and physical health outcomes (Galván, 2019; Zhang et al., 2017).

We examined two primary research questions in the current study. First, does sleep (e.g., sleep problems, daytime sleepiness) serve as an intervening process in the link between adverse parenting and problem behaviors among youth transitioning to adolescence? Second, does gender moderate the link between adverse parenting and sleep in this intervening model? Based on previous findings (Kelly et al., 2014; Xiao et al., 2019), we hypothesized that (H1) adverse parenting would predict internalizing and externalizing symptomology a year later via youth-reported sleep, and (H2) the link between adverse parenting and sleep problems would be moderated by gender, with females who experienced adverse parenting at a greater risk for sleep issues than males.

Methods

Sample

This study utilized data from a longitudinal sample of 101 youth aged 9-12 (M_{age} = 10.28, *SD* = 1.19; 52.5% female) and one of their primary caregivers. Families were from a non-metropolitan region in the Southeastern United States. Participants were recruited through community organizers and online and in-person advertisements. Inclusion criteria required that

youth were (a) between the ages of 9-12, (b) English speakers, and (c) able to read and answer questions at an elementary reading level. Additionally, families who participated were required to have had a household income at or below 200% of the federal poverty level. Participants were considered ineligible for the study if (a) the parent was pregnant or (b) the youth had type II diabetes or significant developmental disabilities. This exclusion criteria was due to the collection of psychophysiological data (e.g., heart rate variability and inflammation) in the larger study and the lack of resources to adapt study materials for youth with significant developmental disabilities. Approximately 8.8% (n = 8) and 16.5% (n = 16) of families within the full sample had an open or closed case with child protective services (CPS), respectively. The majority of primary caregivers who participated in the study were the youth's biological mother (n = 88), followed by grandmother (n = 3), stepfather (n = 3), stepmother (n = 2) biological father (n = 2), adoptive mother (n = 2), and aunt (n =1).

Data were collected at two time points (T1; M_{age} = 10.28, SD = 1.19; T2; M_{age} = 12.08, SD = 1.19). Data collection for T1 began in January 2017 and was completed in June 2018. Data collection for T2 took place approximately one year later, January through June 2018. There were 71 families who participated at T2 (70% retention). A majority of the participants were African-American (n = 76, 75.2%), followed by European-American (n = 11, 10.9%), Latinx (n = 9, 8.9%), Native American (n = 1, 1.0%), and Other (n = 4, 4.0%). Nearly half (45.6%) of primary caregivers reported having attained less than a high school degree or a high school degree, 37.6% reported having completed some college education, and 16.8% reported having completed a college degree or higher.

Procedure

The university's Institutional Review Board approved all procedures before data collection began. At T1, data collection took place at a clinical research setting affiliated with the university. Before any study procedures took place, parents provided their written informed consent and youth provided their informed assent. Youth and their primary caregivers completed a battery of survey measures, including measures of parenting behaviors, subjective sleep, and child behavioral problems. Youth completed their surveys with the assistance of a trained research assistant who read each item and the corresponding answer choices to the child. Parents completed surveys independently in a separate room from the child. Approximately one year later, trained study personnel visited families at their homes to conduct follow-up appointments. Some participants (n =16) elected to have their meeting in an on-campus laboratory, instead of their home. Parents and youth completed questionnaires separately using a handheld device.

Measures

Adverse Parenting. Adverse parenting was measured at T1, utilizing subscales from the Parent-Child Conflict Tactic Scale (CTS-PC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). Primary caregivers were asked to indicate the frequency of specific behaviors towards their child that had occurred in the past year, with answer choices ranging from "0" (*this has not happened in the past year*) to "6" (*more than 20 times in the past year*). Emotional abuse was measured using five items from the Psychological Aggression subscale (e.g., "swore or cursed at him/her"; α = .72). Neglect was measured using four items from the Neglect subscale (e.g., "had to leave your child home alone, even when you thought some other adult should be with him/her"; α = .70). Physical abuse was measured using six items from the Corporal Punishment subscale (e.g., "hit him/her on the bottom with something like a belt, a hairbrush, a stick or some other hard object"; α = .83). An adverse parenting composite was created by summing the

corporal punishment, neglect, and psychological aggression subscales (α = .78). Higher scores indicated higher levels of adverse parenting. Prior studies have utilized the CTS-PC subscales together as a measure of family stress (see Oshri, Duprey, Liu, & Ehrlich, 2020).

Sleep. Sleep was measured at T2 using the Medical Outcomes Study (MOS) Sleep Scale (Hays & Stewart, 1992). Youth responded to questions regarding their sleep quality, adequacy, and problems during the previous four weeks. Items were assessed on a 6-point Likert scale ranging from "1" (*None of the time*) to "6" (*All of the time*). Two indicators of sleep were measured: sleep problems and daytime sleepiness. Sleep problems were measured using two items assessing sleep adequacy (e.g., "How often during the past four weeks did you get enough sleep to feel rested upon waking in the morning?") and four-items assessing sleep disturbance (e.g., "How often during the past four weeks did you have trouble falling asleep?"). Adequacy items were reverse coded so that greater values reflected more inadequate sleep. Mean scores were computed across the six items (α = .65). Daytime sleepiness was measured using two items assessing daytime sleep problems: "How often during the past four weeks did you feel drowsy or sleepy during the day?" and "How often during the past four weeks did you have trouble staying awake during the day?" A composite was created based on these two items (r = .22). Previous studies have established the reliability and validity of the MOS in non-clinical populations (e.g., Hays, Martin, Sesti, & Spritzer, 2005; α's ranging from 0.63 to 0.83). Further, the MOS has demonstrated strong convergent validity with other self-report sleep scales assessing healthy young adults (Kato, 2014) and good reliability in healthy adolescent samples (α = .83 for an overall sleep problem index; Przepiorka & Blachnio, 2019).

Internalizing and Externalizing Symptoms. Primary caregivers reported on youth internalizing and externalizing symptoms at T1 and T2 using the Child Behavior Checklist

(CBCL; Achenbach, 1991). Parents were instructed to report on whether items described their child's behavior during the last six months. Items were assessed on a Likert scale ranging from "0" (*not true, as far as you know*) to "2" (*very true or often true*). The externalizing subscale is composed of delinquent and aggressive syndromes (e.g., argues a lot, steals at home, gets in many fights) and was calculated by summing the total score of these two subscales. The internalizing subscale was calculated by summing the total score of the anxiety/depression, social withdrawal, and somatic complaints scales (e.g., feels worthless, fearful or anxious). The internal consistencies for the internalizing and externalizing scales of the CBCL were good at both T1 (α = .88; α = .90, respectively) and T2 (α = .85, α = .88, respectively). Raw total scores were utilized in the analysis, as recommended for longitudinal data analysis (Moeller, 2015). For descriptive purposes, these values were transformed into T-scores. At T1, 9% and 3% of youth met criteria for the 'Borderline' clinical range (60-63) and 13% and 9% were in the 'Clinical' range (64+) of internalizing and externalizing symptoms, respectively. At T2, 5.6% and 8.4% of participants met criteria for the 'Borderline' clinical range, and 11.2% and 5.6% of youth were in the 'Clinical' range of internalizing and externalizing symptoms, respectively.

Covariates. Based on prior empirical literature, in preliminary analyses we examined factors that could be confounding in the associations between adverse parenting, sleep, and youth psychopathology. Potential covariates included youths' gender, ethnicity, age at T1 and T2, pubertal stage at T1, family income at T1, and internalizing and externalizing symptoms at T1. Gender was included as a covariate based on prior research showing increases in internalizing symptoms across the transition to adolescence for girls, but not boys (Leve, Kim, & Pears, 2005). Age and pubertal status were examined given documented changes in sleep (e.g., delayed bedtime) and changes in internalizing and externalizing problems that occur as youth transition

into adolescence and experience pubertal maturation (Ge, Brody, Conger, & Simons, 2006; Laberge et al., 2001). Pubertal stage was measured using three self-report items from the Pubertal Development Scale (Petersen, Crockett, & Boxer, 1988). Ethnicity and family annual income were assessed due to prior research suggesting socioeconomic and race-based disparities in sleep and psychopathology (e.g., Bradley & Corwyn, 2002; El-Sheikh et al., 2013; Organek et al., 2015). Family income was measured via parents self-report of the total household annual income before taxes (e.g., "$0 - $5,000", "$5,001 - $10,000"), which was recoded to reflect the median value of the selected category (e.g., "$2,500", "$7,500").

Analytic Plan

The study hypotheses were tested in a structural equation modeling (SEM) framework using Mplus version 7.4 with maximum likelihood estimation (Muthén & Muthén, 2010). Missing data analyses revealed that missing data were related to observed study variables. At T1, the percentage of missing data ranged from zero to 1.0%. At T2, there were missing data for 30 participants (29.7%). All missing data at T2 were due to participant attrition. Analyses were conducted to investigate group differences between participants who participated at T2 and participants who dropped out of the study. There was a significant group difference by age, with greater attrition among youth who were younger at T1 t (98) = 2.49, p = .01. Chi-square tests were also utilized to determine if participants who dropped out at T2 differed in gender and ethnicity from those who participated at T2. There were no significant group differences by gender χ^2 (1) = 1.84, p = .17, African-American ethnicity, χ^2 (1) = .05, p = .83, Latinx ethnicity, χ^2 (1) = .27, p = .61, or other ethnicity, χ^2 (1) = .04, p = .83. Thus, subsequent analyses were performed under the assumption of data missing at random (MAR; Schafer & Graham, 2002). Based on published criteria (Hu & Bentler, 1999), good model fit was determined if CFI and TLI

values were at or above .95 and RMSEA values were less than .05. Adequate model fit was determined if CFI and TLI values were between .90 and .95 and RMSEA values were between .05 and .08.

Indirect effect models were first constructed to evaluate the association between adverse parenting and internalizing and externalizing symptomology via youths' sleep (e.g., sleep problems, daytime sleepiness; Models 1-2). Next, interaction terms between adverse parenting and sleep were introduced into the intervening model to examine the moderating role of gender in the link between parenting and sleep. Models 3-4 tested the indirect effect between adverse parenting and youth psychopathology via sleep, conditional upon gender. Due to our sample size and limits on statistical power, intervening variables (e.g., sleep problems, daytime sleepiness) were tested in separate models that included both internalizing and externalizing problems. To address concerns regarding statistical power, we conducted a post-hoc Monte Carlo power analysis (N=101) and found that the power for each parameter was greater than .92, which exceeds the commonly accepted value for sufficient power (e.g., .80, Muthén & Muthén, 2002). For the interaction analyses, variables were mean-centered and product terms were computed and modeled (e.g., adverse parenting*gender). Nonsignificant covariates were trimmed from the final models. Lastly, the simple slopes procedure was performed in order to probe significant interactions (Aiken & West, 1991). We reported 95% confidence intervals for all results.

Results

Descriptive Statistics and Bivariate Correlations

Table 1 presents descriptive statistics and bivariate correlations. Data were examined for normality and outliers. There was one outlier (+3 SD) on the adverse parenting variable, and this was winsorized (i.e., recoded to 3 SD above the mean; Ghosh & Vogt, 2012). Sensitivity

analyses were conducted both with and without the winsorized data and results were consistent. Thus, we present results here with the full (non-winsorized) data. Correlations showed that adverse parenting was significantly correlated with daytime sleepiness (r = .34, p = .004, 95% CI [.12, .56]), but not sleep problems (r = .004, p = .97, 95% CI [-.23, .24]). Adverse parenting was associated with internalizing and externalizing problems at T1 (r = .29, p = .003, 95% CI [.10, .48]; r = .35, p < .001, 95% CI [.17, .54], respectively) and T2 (r = .27, p = .02, 95% CI [.04, .50]; r = .42, p < .001, 95% CI [.21, .63], respectively). Sleep problems were correlated with daytime sleepiness (r = .42, p < .001, 95% CI [.21, .63]) and T2 internalizing and externalizing problems (r = .32, p = .01, 95% CI [.10, .54]; r = .34, p = .004, 95% CI [.12, .56], respectively). Daytime sleepiness was associated with T2 internalizing and externalizing problems (r = .33, p = .01, 95% CI [.11, .55]; r = .40, p = .001, 95% CI [.19, .62], respectively). None of the covariates (e.g., gender, age, ethnicity, family income, pubertal status) were significantly correlated with internalizing or externalizing symptoms at T2 (ps > .16) and thus were not retained as covariates for psychopathology in multivariate models.

Analyses of Indirect Effects

Model 1: Indirect effects via sleep problems. In Model 1, adverse parenting did not significantly predict sleep problems at T2 (β = –.06, *SE* = .00, p = .69, 95% CI [-.33, .22]), controlling for youth's gender (β = .17, *SE* = .22, p = .14, 95% CI [-.05, .38]) and age (β = .30, *SE* = .09, p = .01, 95% CI [.09, .51]; see Table 2, Model 1). Ethnicity, family income, and pubertal status were also tested as covariates for youths' sleep problems but were trimmed from the final model due to non-significance. Sleep problems were significantly associated with both internalizing (β = .30, *SE* = .56, p = .002, 95% CI [.12, .48]) and externalizing psychopathology at T2 (β = .27, *SE* = .53, p = .002, 95% CI [.09, .44]), controlling for T1

internalizing and externalizing (β = .47, *SE* = .08, *p* < .001, 95% CI [.33, .68]; β = .52, *SE* = .08, *p* < .001, 95% CI [.36, .68], respectively). The indirect effects of adverse parenting on internalizing and externalizing symptomology via sleep problems were not significant (α*β = -.02, *SE* = .04, *p* = .70, 95% CI [-.10, .07]; α*β = -.02, *SE* = .04, *p* = .70, 95% CI [-.09, .06], respectively). The model explained 12.1% of variance in sleep problems, 36.9% of variance in internalizing symptoms, and 45.1% of variance in externalizing symptoms at T2.

Model 2: Indirect effects via daytime sleepiness. In Model 2, adverse parenting significantly predicted daytime sleepiness at T2 (β = .40, *SE* = .00, *p* = .003, 95% CI [.16, .63]), controlling for youth's gender (β = .19, *SE* = .24, *p* = .07, 95% CI [-.02, .40]; see Table 2, Model 2). Ethnicity, family income, age, and pubertal status were also tested as covariates for daytime sleepiness but were not retained in the final model due to non-significance. Further, daytime sleepiness, in turn, predicted internalizing (β = .32, *SE* = .53, *p* = .002, 95% CI [.13, .52]) and externalizing problems at T2 (β = .28, *SE* = .50, *p* = .003, 95% CI [.10, .47]), controlling for symptoms of internalizing and externalizing at T1 (β = .49, *SE* = .08, *p* < .001, 95% CI [.34, .65]; β = .54, *SE* = .08, *p* < .001, 95% CI [.38, .69], respectively). The indirect effects of adverse parenting on internalizing and externalizing symptomology via daytime sleepiness were both significant (α*β = .13, *SE* = .06, *p* = .03, 95% CI [.02, .24]; α*β = .11, *SE* = .05, *p* = .03, 95% CI [.01, .21], respectively). The model explained 19.4% of variance in daytime sleepiness, 38.8% of variance in internalizing symptoms, and 46.0% of variance in externalizing symptoms at T2.

Conditional Indirect Effect Analyses

Model 3: Conditional indirect effects via sleep problems. In Model 3, there were no significant effects of adverse parenting, gender, or the interaction term (adverse parenting*gender) on sleep problems (see Table 3, Model 3 for full statistics). The model

explained 12.3% of variance in sleep problems, 37.1% of variance in internalizing symptoms, and 45.2% of variance in externalizing symptoms at T2.

Model 4: Conditional indirect effects via daytime sleepiness. In Model 4, adverse parenting was positively associated with daytime sleepiness at T2 (β = .34, *SE* = .00, *p* = .01, 95% CI [.08, .60]; see Table 3, Model 4). Gender was significantly associated with daytime sleepiness (β = .23, *SE* = .23, *p* = .03, 95% CI [.03, .44]), such that females were more likely to report greater sleepiness. Further, the interaction term (adverse parenting*gender) was significantly associated with daytime sleepiness (β = .35, *SE* = .01, *p* = .01, 95% CI [.09, .61]), which in turn predicted internalizing and externalizing symptomology at T2 (β = .31, *SE* = .53, *p* = .002, 95% CI [.13, .50]; β = .25, *SE* = .50, *p* = .003, 95% CI [.10, .45], respectively). The moderation was probed using the simple slopes technique (see Figure 2). High levels of adverse parenting predicted greater daytime sleepiness for female youth (*b* = .02, *p* < .001), but this association was not significant for males (*b* = -.001, *p* = .92). The indirect effect from adverse parenting to internalizing problems via daytime sleepiness, conditional upon gender, was significant (α*β = .11, *SE* = .06 , *p* = .04, 95% CI [.003, .22]) and the conditional indirect effect from adverse parenting to externalizing problems was marginally significant (α*β = .10, *SE* = .05 , *p* = .05, 95% CI [-.001, .19]). The model explained 21.3% of variance in daytime sleepiness, 38.1% of variance in internalizing symptoms, and 45.4% of variance in externalizing symptoms at T2.

Post-Hoc Analyses

Given well-documented gender differences in youth psychopathology (Chaplin & Aldao, 2013; Nolen-Hoeksema & Hilt, 2009), post-hoc analyses were conducted to test for potential gender differences in the link between sleep and internalizing and externalizing symptoms (e.g.,

path b). Model fit was adequate for both sleep problems and daytime sleepiness (χ^2 (15) = 24.90, p = .051, RMSEA = .08, CFI = .91, TLI = .87, SRMR = .04; χ^2 (10) = 15.98, p = .10, RMSEA = .08, CFI = .95, TLI = .91, SRMR = .09, respectively). The interaction between sleep problems and gender was not significantly associated with internalizing (β = -.05, *SE* = 1.19, p = .63, 95% CI [-.24, .15]) or externalizing symptoms (β = -.03, *SE* = 1.08, p = .71, 95% CI [-.21, .14]). Further, the interaction between daytime sleepiness and gender was also not significant for internalizing (β = .02, *SE* = 1.19, p = .85, 95% CI [-.19, .23]) or externalizing symptoms (β = .05, *SE* = 1.12, p = .63, 95% CI [-.15, .25]). Thus, we concluded that gender did not moderate the pathway between sleep problems and youth psychopathology.

Discussion

Youth exposed to harsh or abusive parenting are at significant risk for sleep problems and the development of psychopathology in adolescence (Kelly et al., 2014; McPhie et al., 2014). Although sleep problems are linked to a wide range of psychological adjustment in adolescence (Coulombe et al., 2010; Zhang et al., 2017), few studies have examined its intermediate role in the association between adverse parenting and youth psychopathology (Kelly et al., 2014). Results of the present study suggest that daytime sleepiness (e.g., drowsiness, trouble staying awake during the day) serves as an intervening process in associations between adverse parenting and internalizing and externalizing problems. Further, while sleep problems (e.g., inadequacy/disturbance) were associated with internalizing and externalizing problems, they did not play an intervening role in the relation between adverse parenting and psychopathology. In addition, we found gender differences in our model, with females but not males reporting greater daytime sleepiness following adverse parenting in the pathway to internalizing and externalizing psychopathology. These findings provide preliminary evidence for gender differences in the

effect of adverse parenting on daytime sleepiness, while highlighting a potential mechanistic role of sleep in the link between early-life stress and adolescent psychopathology.

The first aim of the study was to examine sleep as an intervening variable linking adverse parenting and internalizing and externalizing behaviors. Contrary to our hypothesis, we found that adverse parenting was not associated with sleep problems. However, adverse parenting was significantly related to youths' daytime sleepiness, with harsh and neglectful parenting predicting more feelings of daytime fatigue and drowsiness. Although unexpected, these divergent findings may reflect Sadeh's (1996) identification of the "turn-on" and "shut-off" modes of adaptation following childhood stress. Specifically, whereas the "turn-on" mode reflects hyperarousal that manifests in difficulty initiating or maintaining sleep (e.g., latency, disturbance), the "shut-off" mode reflects a hypoarousal response marked by decreased activity (e.g., exhaustion) and the extension and deepening of sleep (Sadeh, 1996). This exhaustion may manifest as daytime fatigue behaviors among school-aged youth, who may not have the option to extend their sleep due to early school-start times, but whose sleepiness reflects reduced activity and increased desire for sleep. Indeed, our findings indicated that youth who experienced adverse parenting reported increased drowsiness and reduced activity levels throughout the day, which may serve as an attempt to cope with this stressor. However, further research is needed examining this hypothesis using more comprehensive longitudinal assessments of sleep that can better capture aspects of this theory (e.g., actigraphy-derived sleep duration, polysomnography).

Given the considerable amount of research linking experiences of harmful parenting to less quality sleep (e.g., McPhie et al., 2014; Turner et al., 2020), the nonsignificant associations between adverse parenting and sleep problems were surprising. However, these results are consistent with recent studies that found little or no association between adverse parenting (e.g.,

child maltreatment) and adolescent sleep problems (Schneiderman, Ji, Susman, & Negriff, 2018; Wamser-Nanney & Chesher, 2018). Specifically, Schneiderman et al. (2018) found that maltreatment status did not significantly predict sleep disturbance, but was significantly linked to *longer* sleep duration, a finding that may yield support for the aforementioned "shut-off" mode (i.e., extension of sleep). Notably, whereas previous studies have shown that adverse parenting is associated with global indices of sleep problems (i.e., composites including both daytime and nighttime sleep problems; McPhie et al., 2014; Noll et al., 2006), the present study is innovative in the examination of daytime sleepiness and sleep problems as distinct responses to adverse parenting.

Next, the present study contributes to the hypothesis that sleep is a transdiagnostic mechanism underlying both internalizing and externalizing psychopathology (Harvey et al., 2011). Consistent with the concept of multifinality, or the notion that similar experiences can result in heterogeneous sequelae (Cicchetti & Rogosch, 1996), sleep was related to symptoms of both internalizing and externalizing problems. These results are not surprising, given that symptoms of internalizing and externalizing were highly correlated in the current study (r = .67). The findings that daytime sleepiness and sleep problems predicted internalizing psychopathology is supported by a large body of work establishing the co-occurrence of various sleep issues (i.e., sleepiness, difficulty falling asleep) with depressive and anxious symptomology (Kelly & El-Sheikh, 2014; Pieters et al., 2015). In addition, the findings that both forms of sleep issues were related to externalizing behaviors are consistent with previous findings linking multiple indicators of sleep to youth externalizing problems (Coulombe et al., 2010; Shimizu et al., 2020). These associations highlight the importance of sleep as a natural restorative process that is essential not only for adolescents' cognitive performance (Lo et al., 2016), but for

socioemotional adjustment as well (El-Sheikh, Saini, Gillis, & Kelly, 2019). Further, this investigation contributes to the limited literature examining sleep as an intervening mechanism between family stress and youth psychopathology (e.g., Kelly et al., 2014).

A secondary aim of this study was to examine gender differences in the link between adverse parenting and sleep. In contrast to our expectations, we observed no significant gender differences in the link between adverse parenting and sleep problems, such that harsh parenting was unrelated to sleep inadequacy/disturbance for both males and females. These results vary from a previous study demonstrating that females were at higher risk for sleep disturbances following the experience of child maltreatment (Xiao et al., 2019). It is possible that our results differ because we examined adverse parenting (as opposed to child maltreatment), which may have different effects on adolescents' sleep. However, as predicted, there were significant gender differences in levels of daytime sleepiness among youth experiencing adverse parenting, with females, but not males, reporting greater levels of sleepiness. These results are consistent with previous findings highlighting adolescent girls' risk for daytime sleepiness in particular (Lee et al., 1999; Matthews et al., 2014) and contributes to limited existing research examining gender differences in sleep following harsh or abusive parenting (Xiao et al., 2019). Importantly, these findings provide preliminary evidence that preadolescent girls may be particularly at-risk for daytime sleepiness following experiences of adverse parenting, which may in turn explain subsequent elevations in adolescent psychopathology. Future research should aim to extend this work by investigating the role in which pubertal maturation influences these gender differences. Indeed, although pubertal status was not significant in our final models, the positive correlation between pubertal status and both female gender and daytime sleepiness suggests that pubertal

change could have played a role in these findings, such that adolescent girls' heightened sleepiness may be due, in part, to their pubertal advancement (Carskadon, 2011).

Taken together, our results suggest that daytime sleep problems may serve as an underlying mechanism in the link between adverse parenting and internalizing and externalizing psychopathology. In conjunction with theories of childhood stress and sleep (e.g., Sadeh, 1996), these results may point to underlying physiological processes implicated in the maintenance of the sleep-wake cycle, such as the stress response system. Indeed, prior work has established a bidirectional relationship between sleep and HPA axis activity during adolescence (Zeiders et al., 2011), while other studies suggest that sleep may modulate HPA axis functioning in the context of family stress (Chiang et al., 2016). Importantly, a growing body of literature suggests that stress response dysregulation may serve as a mechanism in the association between early-life stress and adolescent psychopathology (Duprey et al., 2020; Stroud et al., 2019). Therefore, physiological stress activity may help further explain the associations between early life stress, sleep problems, and youth psychopathology. However, future work is needed to examine the degree to which these factors may transactionally influence youths' outcomes in the context of harsh or neglectful parenting (Gonzalez & Oshri, 2019).

The findings of the current study may help inform future intervention and prevention efforts that aim to reduce the impact of adverse parenting on youth psychopathology. Sleep issues are a possible target for intervention and prevention efforts that aim to decrease youth psychopathology. Indeed, evidence provided by randomized control trials suggest that cognitive-behavioral interventions to reduce sleep problems do improve mental health in adolescence (e.g., Blake & Allen, 2020). Prevention programs in middle school and high school may be particularly effective, as these youth are nearing the transition into adolescence and may be at

heightened risk for negative alterations in sleep (Laberge et al., 2001). In addition, we identified gender differences in the link between adverse parenting and sleepiness, but not in the association between sleepiness and adjustment. The gender specific effect suggests that early adolescent girls are at greater risk than boys for sleepiness following adverse parenting, whereas sleepiness is equally detrimental for adjustment across gender. This is an important finding, as the majority of existing sleep intervention programs focus on youths' sleep duration and sleep-wake schedules and less on the *consequences* of poor sleep (e.g., daytime sleepiness). Intervention programs responding to adverse parenting should consider allocating special attention to adolescent girls, as their risk for daytime sleep dysfunction may increase significantly in the context of adverse parenting. Nonetheless, sleepiness and sleep problems should both be considered promising targets for the reduction of internalizing and externalizing symptoms among adolescent boys and girls.

Recognizing sleep as both a risk factor (i.e., for psychopathology) and an outcome (i.e., of family stress) may aid educators and health professionals in identifying preadolescents for preventive intervention programs. From a practice standpoint, clinicians who are working with adolescents who have experienced adverse parenting may attempt to focus on aspects of the youth's sleep and involve parents in strategies to help reduce daytime sleep dysfunction and promote healthful sleep. Importantly, because adverse parenting is often the product of financial or structural barriers (e.g., less access to resources), it is critical that prevention and intervention programs appropriately target parents and families within these circumstances by providing accessible and digestible resources to help support parents and reduce instances of harmful or neglectful parenting.

Limitations

The results of this study should be interpreted while considering the study limitations. First, our sample was comprised of low-income and predominantly African-American families, which may limit the generalizability of our findings to youth from other racial/ethnic or socioeconomic backgrounds. Additionally, in contrast to the previous study examining objective sleep as an intervening variable in the link between adverse parenting and psychopathology (Kelly et al., 2014), the current study utilized a brief self-reported measure assessing daytime sleepiness (two items) and sleep problems (six items), which may not accurately represent the quality of youths' sleep, but instead, their perceptions of their sleep. Specifically, we utilized the MOS Sleep Scale (Hays & Stewart, 1992), which was originally adapted for use within clinical populations, but has been validated for use in the general population (Hays et al., 2005) and successfully administered in non-clinical adolescent samples (Przepiorka & Blachnio, 2019). Further, sleep problems and daytime sleepiness were only assessed at T2, limiting our ability to model the potential continuity of sleep problems. Future studies seeking to replicate the findings of the current study should incorporate longitudinal objective measures of sleep (e.g., actigraphy) or subjective reports that are more widely used in adolescent samples (e.g., Adolescent Sleep-Wake Scale; LeBourgeois, Giannotti, Cortesi, Wolfson, & Harsh, 2005).

The utilization of parent reports for youth psychopathology was an additional limitation of the current study, as previous studies have noted discrepancies between youth and parent-reported assessments of problem behaviors (De Los Reyes & Kazdin, 2005). Adverse parenting was also assessed using parent-report. It is likely that parents were underreporting their negative parenting practices (i.e., due to social desirability bias; Straus et al., 1998); yet, the present study did reveal associations between adverse parenting and youths' sleep, suggesting that these links may have been stronger if parents fully reported on adverse parenting practices. Despite the

benefits of the study's longitudinal design, sleep and psychopathology were both measured at T2, which limits our ability to determine causal inference of sleep on psychopathology (versus a bidirectional influence). However, adjusting for T1 psychopathology within our models helped further support the direction of this finding. Additionally, pubertal status was examined as a potential covariate at T1 but was not measured at T2; this would have been an important variable to account for alongside sleep and psychopathology. Last, other variables that were not considered in the present study (e.g., family structure, additional forms of family stress) could have impacted these results, and thus should be considered in future studies.

Conclusion

The present study suggests that daytime-related sleep behaviors (e.g., daytime sleepiness) may serve as a mechanism through which harsh or neglectful parenting is related to internalizing and externalizing psychopathology in adolescence. Further, the current study found that daytime sleepiness and sleep problems (e.g., inadequacy/disturbance) were independently associated with internalizing and externalizing psychopathology. Importantly, this study provides evidence that female adolescents may be particularly at-risk for daytime sleepiness following experiences of adverse parenting. Future research should aim to expand these findings by incorporating objective sleep data (e.g., actigraphy) and investigating sleep-relevant physiological processes that may further elucidate these associations. Implications for future prevention efforts include promoting the importance of sleep health for adolescents reared in families with harsh and/or neglectful parents and implementing intervention programs that aim to reduce adverse parenting behaviors among teen parents.

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Table 1. *Bivariate Correlations and Descriptive Statistics*

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Adverse Parenting	—												
2. Sleep Problems	.004	—											
3. Daytime Sleepiness	.34**	.42***	—										
4. Gender (1 = male, 2 = female)	.01	.17	.23†	—									
5. Ethnicity	-.08	-.09	.02	-.04	—								
6. Family Income	.20	.15	.01	.18	-.28*	—							
7. Age (T1)	.18†	.26*	.19	.12	-.20†	.24†	—						
8. Age (T2)	.11	.30*	.20†	.04	-.15	.13	.90***	—					
9. Pubertal Status (T1)	.16	.08	.30*	.36***	-.13	.14	.52***	.54***	—				
10. Internalizing (T1)	.29**	.03	-.00	.01	-.06	.03	.30**	.11	.14	—			
11. Externalizing (T1)	.35***	.11	.15	.00	-.01	-.03	.26**	.25*	.17†	.73***	—		
12. Internalizing (T2)	.27*	.32**	.33**	.15	-.11	-.23	.11	.02	.07	.48***	.23†	—	
13. Externalizing (T2)	.42***	.34**	.40**	.08	-.01	-.14	.16	.07	.08	.38**	.52***	.67***	—
M	33.56	2.53	2.45	1.52	.75	21.74	10.28	12.08	2.30	5.76	5.07	5.76	4.61
SD	36.86	.95	1.08	.50	.44	12.80	1.19	1.19	.65	5.99	6.49	5.52	5.37
Skewness	2.10	.35	.99	-.10	-1.14	.73	.10	-.01	.38	1.17	1.99	1.52	1.27
Kurtosis	6.92	-.97	1.70	-2.03	-.71	.19	-1.21	-.82	-.12	.68	4.26	3.30	.78

Notes. Ethnicity is coded as 1 = African-American, 0 = Other; Family Income = Annual family income in thousands; T1= Time 1, T2 = Time 2.
$^{\dagger}p < .10$, *$p < .05$, ** $p < .01$, *** $p < .001$

Table 2. *Parameters for Path Models Testing Indirect Effects*

Model 1 (Sleep Problems)	*B*	*SE*	β	*p*	95% CI
Adverse Parenting (AP) → T2 INT	.02	.02	.16	.20	[-.08, .39]
AP → T2 EXT	.03	.02	.21	.08	[-.02, .44]
AP → Sleep Problems	-.001	.004	-.06	.69	[-.32, .22]
T2 Age → Sleep Problems	.24	.09	.30	.008**	[.09, .51]
Gender → Sleep Problems	.31	.22	.17	.14	[-.05, .38]
Sleep Problems → T2 INT	1.77	.56	.30	.002**	[.12, .48]
Sleep Problems → T2 EXT	1.60	.53	.27	.002**	[.09, .44]
T1 INT → T2 INT	.44	.08	.47	<.001***	[.33, .68]
T1 EXT → T2 EXT	.45	.08	.52	<.001***	[.36, .68]
Indirect effects	α*β	*SE*	*p*	95% *CI*	
AP → SleepProb → T2 INT	-.02	.04	.70	[-.10, .07]	
AP → SleepProb → T2 EXT	-.02	.04	.70	[-.09, .06]	
Model 2 (Daytime Sleepiness)	*B*	*SE*	β	*p*	95% CI
Adverse Parenting (AP) → T2 INT	.002	.02	.01	.94	[-.24, .26]
AP → T2 EXT	.01	.02	.08	.54	[-.17, .32]
AP → Daytime Sleepiness	.01	.004	.40	.003**	[.16, .63]
Gender → Daytime Sleepiness	.43	.24	.19	.07	[-.02, .40]
Daytime Sleepiness → T2 INT	1.68	.53	.32	.002**	[.13, .52]
Daytime Sleepiness → T2 EXT	1.47	.50	.28	.003**	[.10, .47]
T1 INT → T2 INT	.47	.08	.49	<.001***	[.34, .65]
T1 EXT → T2 EXT	.47	.08	.54	<.001***	[.38, .69]
Indirect effects	α*β	*SE*	*p*	95% *CI*	
AP → DaySleep → T2 INT	.13	.06	.03*	[.02, .24]	
AP → DaySleep → T2 EXT	.11	.05	.03*	[.01, .21]	

Notes. AP = Adverse Parenting, INT = Internalizing, EXT = Externalizing, T1= Time 1, T2 = Time 2, SleepProb = Sleep Problems, DaySleep = Daytime sleepiness. *$p < .05$, **$p < .01$, ***$p < .001$

Model 1: $\chi^2 = 15.28$ ($df = 14$), $p = .36$, RMSEA = .03, CFI = .99, TLI = .99, SRMR = .07

Model 2: $\chi^2 = 7.86$ ($df = 9$), $p = .55$, RMSEA = .00, CFI = 1.00, TLI = 1.02, SRMR = .05

Table 3. *Parameters for Path Models Testing Conditional Indirect Effects*

Model 3	*B*	*SE*	β	*p*	95% CI
Adverse Parenting (AP) → T2 INT	.02	.02	.16	.20	[-.08, .39]
AP → T2 EXT	.03	.02	.21	.08	[-.02, .44]
AP → Sleep Problems	-.001	.004	-.04	.76	[-.33, .24]
T2 Age → Sleep Problems	.24	.09	.30	.008**	[.09, .51]
Gender → Sleep Problems	.30	.22	.16	.16	[-.06, .38]
AP*Gender → Sleep Problems	-.003	.01	-.07	.65	[-.35, .22]
Sleep Problems → T2 INT	1.77	.56	.30	.002**	[.12, .48]
Sleep Problems → T2 EXT	1.60	.53	.27	.002**	[.09, .44]
T1 INT → T2 INT	.44	.08	.47	<.001***	[.31, .63]
T1 EXT → T2 EXT	.45	.08	.52	<.001***	[.36, .68]
Conditional indirect effects	α*β	*SE*	*p*	95% *CI*	
AP*Gender → SleepProb → T2 INT	-.02	.04	.66	[-.11, .07]	
AP*Gender → SleepProb → T2 EXT	-.02	.04	.66	[-.10, .06]	
Model 4	*B*	*SE*	β	*p*	95% CI
Adverse Parenting (AP) → T2 INT	.002	.02	.01	.94	[-.25, .27]
AP → T2 EXT	.01	.02	.08	.54	[-.17, .33]
AP → Daytime Sleepiness	.01	.004	.34	.01*	[.08, .60]
Gender → Daytime Sleepiness	.50	.23	.23	.03*	[.03, .44]
AP*Gender → Daytime Sleepiness	.02	.01	.35	.01*	[.09, .61]
Daytime Sleepiness → T2 INT	1.68	.53	.31	.002**	[.13, .50]
Daytime Sleepiness → T2 EXT	1.47	.50	.25	.003**	[.10, .45]
T1 INT → T2 INT	.47	.08	.49	<.001***	[.34, .65]
T1 EXT → T2 EXT	.47	.08	.54	<.001***	[.39, .69]
Conditional indirect effects	α*β	*SE*	*p*	95% *CI*	
AP*Gender → DaySleep → T2 INT	.11	.06	.04*	[.003, .22]	
AP*Gender → DaySleep → T2 EXT	.10	.05	.05	[-.001, .19]	

Notes. AP = Adverse Parenting, INT = Internalizing, EXT = Externalizing, T1= Time 1, T2 = Time 2, SleepProb = Sleep Problems, DaySleep = Daytime sleepiness. *p < .05, **p < .01, ***p < .001

Model 3: χ^2 = 17.33 (*df* = 18), p = .50, RMSEA = .00, CFI = 1.00, TLI =1.01, SRMR = .08

Model 4: χ^2 = 8.36 (*df* = 12), p = .76, RMSEA = .00, CFI = 1.00, TLI = 1.05, SRMR = .04

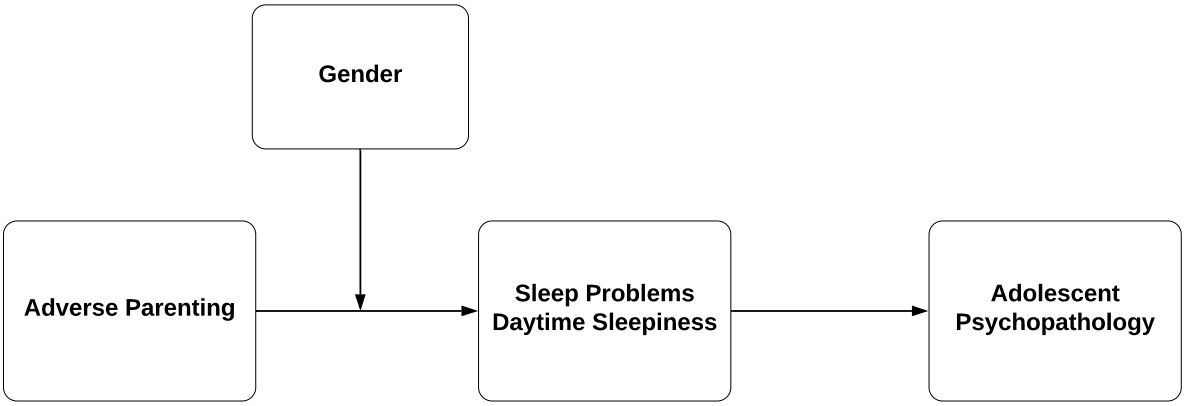

Figure 1. Conceptual figure illustrating the hypothesized model.

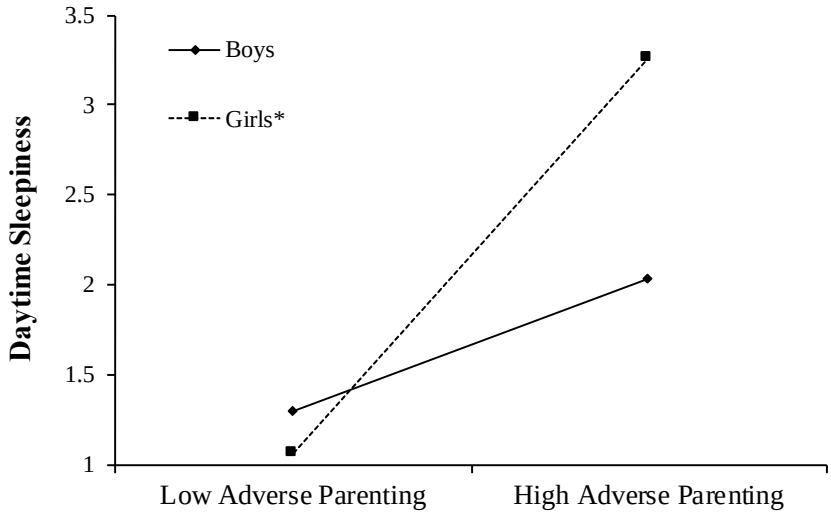

Figure 2. Gender moderates the association between adverse parenting and daytime sleepiness. Adverse parenting was tested at low (1 SD below the mean) and high (1 SD above the mean). *$p < .001$.