

Predictors and Consequences of Disruptions in Sleep among Children

by

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Abstract

This dissertation includes two studies. In the first study, children's emotional insecurity was examined as a process variable and as a moderator of effects using three waves of data spanning five years. Participants were 176 children at T1 ($M = 8.68$ years), 141 children at T2 ($M = 10.70$ years) and 113 children at T3 ($M = 13.60$ years) along with their parents. Parents reported on marital conflict, children reported on their emotional insecurity in the parental marital relationship, and children's sleep was measured via self-reports and actigraphy. Marital conflict predicted increased emotional insecurity about the marital relationship two years later (T2), which in turn predicted greater sleep problems three years later (T3). Moderation analyses indicated that increased emotional insecurity was a vulnerability factor for sleep problems in the context of greater marital conflict. Findings illustrate the importance of considering children's sleep within the family context and demonstrate the pivotal role of emotional security.

Using the same sample, the second paper examined the reciprocal relations between children's sleep and their internalizing and externalizing symptoms across the three waves of data. Sleep was measured subjectively via self-reports and objectively via actigraphy and children and parents reported on children's adjustment. Cross-lagged panel models were fit to examine whether sleep problems predicted changes in internalizing and externalizing symptoms over time. Examining reciprocal relations, we also assessed internalizing and externalizing symptoms as predictors of changes in sleep problems longitudinally. Reduced sleep amount and worse sleep quality predicted increases in depression, anxiety, and externalizing symptoms over time. To a lesser extent but supportive of reciprocal relations, more internalizing and externalizing symptoms predicted increases in sleep problems longitudinally. In sum, the

cyclical nature between sleep problems and adjustment difficulties are demonstrated among otherwise normally developing youth. Across both studies, important advances are made in identifying the predictors and consequences of children's sleep problems, which is an important facet of biological regulation that plays a key role in ensuring a healthy development.

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List of Abbreviations

EST Emotional Security Theory

Abstract Manuscript 1

To gain a better understanding of relations between marital conflict and children's sleep problems, children's emotional insecurity was examined as a process variable and a moderator of effects using three waves of data spanning five years. Participants were 176 children at T1 ($M = 8.68$ years), 141 children at T2 ($M = 10.70$ years) and 113 children at T3 ($M = 13.60$ years) along with their parents. Parents reported on marital conflict, children reported on their emotional insecurity in the parental marital relationship, and children's sleep was measured via self-reports and actigraphy. After controlling for autoregressive effects, marital conflict predicted increases in emotional insecurity two years later (T2), which in turn predicted an increase in sleep problems three years later (T3). Moderation analyses indicated that a higher level of emotional insecurity was a vulnerability factor for sleep problems in the context of greater marital conflict. Findings add to this emerging literature and illustrate the importance of considering sleep in the family context.

Longitudinal Relations between Marital Conflict and Children's Sleep: The Role of Emotional Insecurity

Sleep problems among school-aged children are highly prevalent and are related to adjustment (Alfano, Zakem, Costa, Taylor, & Weems, 2009; Arman et al., 2011) and cognitive problems (Buckhalt, El-Sheikh, Keller, & Kelly, 2009). Thus, identification of risk factors for sleep problems in normally developing children is important. Familial processes including parental marital conflict are pivotal for children's sleep (El-Sheikh & Kelly, 2011). Children who are exposed to marital conflict often experience sleep problems (El-Sheikh, Buckhalt, Mize & Acebo, 2006; Kelly & El-Sheikh, 2011; Spilsbury, 2009). However, the nature of the relation between marital conflict and children's sleep is just beginning to be understood. Examining intervening processes and moderators in the connection between parental marital conflict and children's sleep is critical to explicate why and for whom the association is evident. In the current study, we examined children's emotional insecurity about the marital relationship as a process variable and moderator in the association between marital conflict and children's sleep problems. In this context, a process variable would bridge the relation between conflict and children's sleep. Support for moderation effects would not speak to issues of causality, but rather would explicate for whom and under what conditions the relation between conflict and children's sleep problems is evident. Support for intervening or mediation effects does not negate the added possibility of moderation effects and indeed a variable may function as both (Baron & Kenny, 1986; Muller, Judd, & Yzerbyt, 2005).

We examined sleep problems that are common among normally developing children including a shorter average sleep duration and increased night wakings, compared to other participants. Short sleep duration (Pesonen et al., 2010) and restless sleep (Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009) are very prevalent in otherwise typically developing children,

and their assessment in community samples is thus warranted. Sleep problems were measured along a continuum versus dichotomizing children into groups.

Marital conflict (verbal/psychological and physical) is an important and highly prevalent familial stressor that can influence children's sleep. Verbal conflict/aggression refers to verbal threats, insults, and throwing objects (not at partner) whereas physical conflict/aggression is characterized by physical assault on a partner's body (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). The terms marital conflict and aggression are used interchangeably in this paper and refer to either psychological/verbal or physical aggression unless otherwise noted. Verbal conflict is quite common in marital relationships, with studies of community samples revealing that the majority of couples have experienced at least one recent incident of such aggression (Lawrence, Yoon, Langer, & Ro, 2009; Vickerman & Margolin, 2008). Moreover, about 49% of parents with at least one child have experienced some form of physical conflict, and 24% of these couples have experienced severe physical conflict such as burning, hitting, or using a gun or knife (Smith Slep & O'Leary, 2005).

Within the past five years, the association between marital conflict and children's sleep has received considerable attention (El-Sheikh et al., 2006; El-Sheikh, Buckhalt, Cummings, & Keller, 2007; Hall, Zubrick, Silburn, Parsons, & Kurinczuk, 2007). In his recent conceptual paper, Spilsbury (2009) proposed that the trauma and stress that accompany exposure to violent circumstances are likely to result in sleep problems, which in turn may put children at risk for a myriad of physical, emotional, and behavioral problems. Thus, given the pivotal role that sleep has for children's well-being across multiple domains, Spilsbury (2009) emphasized the importance of continued investigations of associations between family aggression and children's sleep. In cross-sectional studies, a higher level of parental marital conflict has been related to actigraphy-measured sleep parameters including a shorter sleep duration, reduced sleep efficiency (% of night spent asleep), and a higher level of activity during the sleep period (El-

Sheikh et al., 2006; El-Sheikh, Buckhalt, Cummings, et al., 2007). Similar findings have emerged with children's self-reported sleep problems and sleepiness (El-Sheikh et al., 2006; Kelly & El-Sheikh, 2011). Overall, the results are clear: high conflict homes are not conducive for children's sleep.

In addition to cross-sectional studies, longitudinal work has demonstrated that facets of the marital relationship including marital conflict may influence children's sleep over time (Gregory, Caspi, Moffitt, & Poulton, 2006; Hall et al., 2007). More marital instability (e.g., whether parents have seriously considered separation or divorce) when children were 9 months old predicted an increase in parent-reported child bedtime resistance (e.g., child struggles at bedtime) 9 months later, even after controlling for initial sleep problems (Mannering et al., 2011). Similarly, using the first two waves from the present study, Kelly and El-Sheikh (2011) found that exposure to parental marital conflict when children were 8 years old predicted an increase in sleep problems two years later, including self-reports of sleep/wake problems and daytime sleepiness, even after controlling for autoregressive effects. Although these studies are important and indicate that exposure to marital conflict predicts children's sleep problems, even years later, there is a continued need to explicate mechanisms of effects in the marital conflict – children's sleep association. In other words, why does exposure to marital conflict predict children's sleep problems?

Few studies have examined mechanisms of effects in the association between marital conflict and children's sleep. Using the first wave from the present study, El-Sheikh, Buckhalt, Cummings, et al. (2007) found children's emotional insecurity regarding their parents' marital relationship to be an intervening variable in the association between marital conflict and children's sleep problems. Increased marital conflict predicted greater emotional insecurity, which in turn predicted shorter sleep duration and poorer sleep quality as assessed by actigraphy. However, findings from El-Sheikh, Buckhalt, Cummings, et al.'s (2007) study were

cross-sectional, which limits inferences about directionality of effects. To address this gap, we sought to further examine emotional insecurity as a process variable in the longitudinal relation between marital conflict and children's sleep problems using three-waves of data spanning five years.

The primary thesis of The Emotional Security Theory (EST) is that marital conflict may undermine a child's sense of safety, stability, and security in the family (Cummings & Davies, 1996; 2010; Davies & Cummings, 1994), which predicts externalizing and internalizing symptoms. Whereas warm and supportive marital relationships promote children's emotional security, highly conflictual relationships can promote emotional insecurity. Emotional insecurity is characterized by (a) emotional reactivity, or intense feelings of anger, fear or sadness in response to parental marital conflict; (b) behavioral dysregulation, or inappropriate attempts to regulate or end parental marital conflict; and (c) destructive family representations, or appraisals of destructive consequences of marital conflict for the family.

Children who are emotionally insecure frequently feel that the family is unstable and unsafe and may question the future of their parents' relationship. Conversely, emotionally secure children feel comfortable, relaxed, and have little concern over the future of the marital relationship and the family. The elevated levels of anxiety, fear, and hyperarousal in response to marital conflict that are characteristic of emotional insecurity (Cummings & Davies, 2010) may be risk factors for sleep problems, given that sleep is difficult to achieve when an individual is vigilant or alert (Dahl, 1996; Dahl & Lewin, 2002).

Another objective of the current study was to examine emotional insecurity as a moderator of the association between marital conflict and children's sleep problems. While feelings of emotional insecurity such as sadness, fear, and anger are common in response to exposure to marital conflict, not all children respond uniformly. Rather, according to the EST, a child's reaction to marital conflict is in part influenced by the child's history of exposure to family

aggression (e.g., living in a high conflict home during early childhood) and by individual child characteristics (Cummings & Davies, 2002; 2010; Davies & Windle, 2001). Thus, not all children who are exposed to marital conflict may become emotionally insecure. The degree to which emotional arousal occurs in response to marital conflict may be pivotal in determining how conflict influences children's adaptation (Crockenberg & Langrock, 2001; El-Sheikh, 2005). In other words, and pertinent to the current study, emotional insecurity may interact with marital conflict and function as either a vulnerability or protective factor in the link between conflict and children's adaptation (Davies & Windle, 2001).

Supportive of children's emotional insecurity as a moderator of associations between marital conflict and child outcomes, El-Sheikh (2005) found that sadness and fear in response to simulated conflict moderated the relation between conflict and children's developmental outcomes. In comparison to children with less intense emotional reactions, higher levels of marital conflict were more strongly related to adjustment problems for children who responded to an audiotaped argument between a man and woman with higher levels of observed (i.e., coded by researchers) sadness and anger. In another example, Crockenberg and Langrock (2001) found that children's anger and fear in response to marital conflict moderated the association between exposure to conflict and internalizing symptoms. Specifically, elevated levels of self-reported anger and fear were vulnerability factors for internalizing symptoms whereas lower levels of these emotions served as protective factors. These findings illustrate an aggregation of risk perspective in relation to child and family characteristics (Appleyard, Egeland, van Dulmen, & Sroufe, 2005) and indicate that not all children are uniformly impacted by marital conflict.

Based on the aforementioned findings, it is plausible that emotional insecurity may moderate the relation between marital conflict and children's sleep outcomes. The increased arousal and vigilance that is characteristic of emotional insecurity may interact with marital

conflict and exacerbate children's sleep problems (Keller & El-Sheikh, 2011). In contrast, children who are more emotionally secure may be protected against sleep problems given their lower levels of vigilance and arousal, despite being exposed to marital conflict. Demonstrating moderation effects and aggregation of risk in a somewhat similar context, children's emotional insecurity in the child-parent relationship interacted with sleep to predict academic achievement (Keller, El-Sheikh, & Buckhalt, 2008). Specifically, children with both an insecure attachment and less optimal sleep (amount, quality) had the worst achievement outcomes.

The Current Study

Consistent with recent recommendations (Sadeh, 2008; 2011), we used both children's self-reports and actigraphy to assess sleep, which provide unique information on a wide range of sleep parameters. Using three waves of data spanning five years with a two year lag between T1 (age 8) and T2 (age 10) and nearly a three year lag between T2 and T3 (age 13), our primary objective was to examine whether change in children's emotional insecurity between T1 and T2 mediated or served as an intervening variable in the association between T1 marital conflict and change in the sleep parameters between T2 and T3. We hypothesized that a higher level of marital conflict at T1 would predict increases in children's emotional insecurity at T2, which in turn would predict increases in children's sleep problems at T3.

We also examined emotional insecurity as a moderator of relations between marital conflict and change in children's sleep over time (i.e., through controlling autoregressive effects for sleep). Consistent with an aggregation of risk perspective (Appleyard et al., 2005), we hypothesized that emotional insecurity about the marital relationship would function as a vulnerability factor and exacerbate sleep problems associated with marital conflict. Further, regarding the EST, Cummings and Davies (2010) stated a need to examine relations between marital conflict, emotional insecurity, and children's outcomes across different ages. Toward addressing this open scientific question and for a thorough examination of moderation effects,

we examined whether (a) T1 emotional insecurity moderated associations between T1 marital conflict and T2 sleep, and (b) whether T2 emotional insecurity moderated relations between T2 marital conflict and T3 sleep.

Method

Participants

Families participated in a three-wave study. At T1, 3rd grade children from a local public school system in the Southeastern United States were recruited. We contacted parents using home telephone numbers provided by schools and in addition, some interested families initiated contact with our research laboratory. Of those families who met our inclusion criteria (parents had to be married and living together, no child diagnosis of a chronic or acute physical illness, ADHD, learning disability, or history of a diagnosed sleep problem), 66% agreed to participate and were included in the study; 34% declined participation. A total of 78 boys and 98 girls ($N = 176$) with a mean age of 8.68 years ($SD = .36$) participated at T1, along with their parents. Based on mothers' reports on the Puberty Development Scale (1 = prepubertal, 2 = early pubertal, 3 = midpubertal, 4 = late pubertal, 5 = postpubertal; Petersen, Crockett, Richards, & Boxer, 1988), the mean puberty score was 1.25 ($SD = 0.23$) for boys and 1.45 ($SD = 0.34$) for girls, indicating that the majority of children were prepubertal.

Regarding the ethnic composition of the sample, 69% of children were European American (EA) and 31% were African American (AA); these percentages are representative of the community. Regarding annual family income, 11% earned between \$10,000 and \$20,000, 24% earned between \$20,000 and \$35,000, 29% earned between \$35,000 and \$50,000, 25% earned between \$50,000 and \$75,000, and 11% earned more than \$75,000; the mean family income was in the \$35,000 to \$50,000 range. For mothers' and fathers' education, 9% and 13% had partial high school education or less, 36% and 46% received a high school diploma, 42% and 30% had partial college education or received specified training, 11% and 9% received a

college diploma, and 2% and 2% earned a graduate degree or received professional training, respectively. We sought to recruit both EA and AA families across a wide range of income backgrounds through oversampling, which resulted in a significant moderate correlation between race/ethnicity and income ($r = -.37$; race/ethnicity was dummy coded such that AA = 1 and EA = 0). Preliminary analyses indicated that family income was related to mothers' ($r = .29$, $p < .001$) and fathers' education ($r = .35$, $p < .001$), and in addition mothers' and fathers' education were associated ($r = .32$, $p < .001$). Consequently, we standardized and mean composited family income, mothers' education, and fathers' education to create an overall socioeconomic status (SES) variable. Mention of an SES variable throughout the remainder of this paper is in reference to this composited variable.

Families participated in a second wave of data collection about two years after their initial participation (M time lag between T1 and T2 = 1 year and 361 days, $SD = 111$ days). Of the 176 participants at T1, 142 participated at T2 (62 boys and 80 girls; 81% of the original sample). Of the families that participated at T2, 2% of parents had divorced since T1. In cases where parents divorced, the families were still invited back for participation. At T2, children's average age was 10.70 years ($SD = 0.55$); 70% were EA and 30% were AA. At T2, the mean puberty score was 1.49 ($SD = 0.42$) for boys and 2.06 ($SD = 0.55$) for girls, corresponding with prepubertal status for boys and early pubertal status for girls (Petersen et al., 1988).

Families participated in a third wave of data collection nearly three years following T2 (M time lag between T2 and T3 = 2 years and 337 days, $SD = 119$ days). Of the 176 families that participated at T1, 113 participated at T3 (50 boys and 63 girls; 64% of original sample and 80% of those who participated at T2). Of the families that participated at T3, 5% of parents had divorced since T2. Reasons for attrition at T2 and T3 included having a busy schedule, moving out of the study area, disinterest in participating, and inability to be located. At T3, children's average age was 13.60 years ($SD = .76$); 70% were EA and 30% were AA. The mean puberty

score for boys was 2.27 ($SD = .65$) and for girls was 3.18 ($SD = .47$), which corresponds with early pubertal status for boys and midpubertal status for girls (Petersen et al., 1988).

Independent samples t -tests and chi-square analyses were used to examine potential selective attrition over study waves. In comparison to children who participated at T2, those who only participated at T1 came from lower SES households, $t(162) = -2.18, p < .05$. Additional analyses yielded no significant differences between retained and attrited participants from T1 to T2 in relation to gender, ethnicity/race, pubertal stage, child age, body mass index (BMI), marital conflict, emotional insecurity, or the sleep parameters. Further, we compared children who participated at T3 and those who discontinued participation after T2. No significant differences were found between retained and attrited participants on any study variable.

Procedure

At each wave of data collection, a research assistant instructed parents to place an actigraph on the child's non-dominant wrist at bed time. Parents were called each night to report child bed- and wake-times in order to validate the actigraphy measured sleep variables (Ambulatory Monitoring Inc., Ardsley, NY). Actigraphy data were collected during the regular school years at T1 and T2, and in the summer during T3. To avoid potential confounding variables, only data from medication-free nights were used in analyses.

Across all three waves, most families visited the research laboratory on the day following the final night of actigraphy data collection (T1 M days = 1.75, $SD = 8.30$ days; T2 $M = 13.19$, $SD = 44.74$; T3 $M = 1.15$, $SD = 5.18$). Parents and children completed several questionnaires; children were administered each questionnaire by a trained interviewer and parents completed questionnaires in separate rooms for privacy. Children's height and weight were measured and were used to calculate body mass index (BMI) based on criteria established by Must, Dallal, and Dietz (1991). Procedures and measures are identical for each of the three waves unless otherwise specified.

Measures

Marital conflict. At T1 and T2, marital conflict was measured via mothers' and fathers' reports on the Revised Conflict Tactics Scale (CTS2; Straus, 1995). The CTS2 measures the frequency and intensity of conflict tactics used between romantic partners and spouses, and has very good psychometric qualities (Straus et al., 1996). To reduce biases related to self-reports of marital conflict, mothers' and fathers' reports of conflict tactics perpetrated by their spouses against them were used in analyses (Ehrensaft & Vivian, 1996). The Psychological/Verbal Aggression (8 items) and Physical Aggression scales (12 items) were pertinent to this investigation. Because of IRB protocol regulations, four items regarding sexual aggression, severe physical aggression, and injury (e.g., choking one's partner and leaving bruises after a fight) were not administered. For each CTS2 item, parents rated how frequently their partner used the specified type of conflict tactic within the last year (e.g., my partner insulted or swore at me). A score of 1 indicates the specific type of conflict occurred once in the past year; 2 = 2 times; 3 = 3 - 5 times; 4 = 6 - 10 times; 5 = 11 - 20 times; 6 = more than 20 times. Overall, at each wave four variables were derived: Father-initiated verbal conflict (mother reported), father-initiated physical conflict (mother reported), mother-initiated verbal conflict (father reported), and mother-initiated physical conflict (father reported). Across T1 and T2, each of the four variables demonstrated good internal consistency and Cronbach's alphas ranged from .71 to .96.

Emotional insecurity. At T1 and T2, children reported on their emotional insecurity within the past year using the Security in the Interparental Subsystem Scales (SIS; Davies, Forman, Rasi, & Stevens, 2002). The Emotional Reactivity scale (e.g., feeling angry, sad, and scared in response to parental arguments), Behavioral Dysregulation scale (e.g., throwing things or yelling in response to parental disputes), and Destructive Family Representations scale (e.g., worry about what the parents will do next or ruminate about the future of the marital relationship) were pertinent to the current investigation and used in analyses. The SIS is based

on the EST (Cummings & Davies, 1996; Cummings & Davies, 2010), and consistent with past research (e.g., El-Sheikh, Buckhalt, Cummings, et al., 2007; Keller & El-Sheikh, 2011) we included the three aforementioned SIS scales in analyses because they represent the emotional, behavioral, and cognitive components of children's emotional insecurity. The SIS has well established psychometric properties (Davies et al., 2002). With the present sample, the three pertinent scales each had good internal consistency across T1 and T2 (alphas ranged from .73 to .90).

Subjective measures of children's sleep. At all three waves, children reported on their own sleep using the School Sleep Habits Survey (SHS; Wolfson & Carskadon, 1998). The SHS has good reliability and validity (Wolfson et al., 2003) and has been used in many studies of children's sleep (e.g., Amschler & McKenzie, 2005; Buckhalt et al., 2009; Keller & El-Sheikh, 2011). The 10-item Sleep/Wake Problems Scale was pertinent and examines the frequency of several sleep and wake problems including difficulty with falling asleep at bedtime and trouble falling back to sleep after a night waking. Items were rated for frequency from never (1) to every day/night (5); higher scores reflect more sleep related problems. Cronbach's alpha = .71 at T1, .79 at T2, and .74 at T3.

Actigraphy measures of children's sleep. At all three waves, actigraphs were placed on the child's non-dominant wrist at bedtime and removed when awake. Actigraphy is used frequently as a proxy for sleep duration and quality, and has demonstrated good reliability, especially when used for multiple consecutive nights (Acebo et al., 1999; Sadeh, Gruber, & Raviv, 2002). The actigraphs were Octagonal Basic Motionloggers (Ambulatory Monitoring Inc., Ardsley, NY), and being awake or asleep was determined with Sadeh's scoring algorithm (Sadeh, Sharkey, & Carskadon, 1994). Procedures for setting times for sleep onset and wake time followed a laboratory protocol developed at the E.P. Brown Sleep Laboratory at Brown University (Acebo & Carskadon, 2001); parents completed daily sleep logs to cross validate

sleep onset and wake time. The actigraphs and software package used in this study are reliable and valid based on comparisons with polysomnography derived sleep measures (Sadeh et al., 1994).

To tap different facets of sleep problems, Sadeh et al. (2000) recommended the assessment of multiple sleep parameters when using actigraphy. In this study, we examined: (a) Sleep Duration – the number of minutes from bedtime to wake time, and (b) Sleep Efficiency – percentage of epochs scored as sleep between sleep onset and offset. Parent diary information was used in the analyses of Actigraphy-based sleep onset time and wake time (Acebo & Carskadon, 2001). Actigraphy variables were generated by computing an average score across all available nights.

At T1, 75% of children had actigraphy data for all seven nights, 14% had data for six nights, 8% had data for five nights, and 3% had data for four nights or fewer ($M = 6.59$ days, $SD = .85$). For T2, 56% of participants had actigraphy data for all seven nights, 25% had data for six nights, 14% had data for five nights, and 5% had data for four nights or fewer ($M = 6.30$ days, $SD = .95$). At T3, 66% of children had actigraphy data for all seven nights, 20% had data for six nights, 6% had data for five nights, and 8% had data for four nights ($M = 6.33$ days, $SD = 1.03$). Reasons for missing data included the use of allergy medicine and exclusion of these data from analyses, forgetting to wear the actigraphs, and mechanical problems. Based on Acebo et al.'s (1999) recommendation, these rates of valid actigraphy data are considered very good.

Using intraclass correlations, we computed stability estimates for the sleep parameters over the week of actigraphy using all available data. Alphas were as follows: Sleep Duration (.78 at T1; .86 at T2; .73 at T3), Sleep Efficiency (.87 at T1; .92 at T2; .81 at T3). All reliability estimates surpassed Acebo et al.'s (1999) recommended stability level of .70. Based on these coefficients, each sleep parameter appears stable across the week.

Plan of Analysis

Structural equation path models were fit to investigate whether children's emotional insecurity served as a process variable (i.e., mediator, intervening variable) and moderator in the relation between marital conflict and sleep problems. Latent constructs were created for marital conflict and emotional insecurity. The latent variable of marital conflict was based on father-initiated verbal conflict, father-initiated physical conflict, mother-initiated verbal conflict, and mother-initiated physical conflict. These four observed variables loaded on the marital conflict latent construct at T1 ($r_s = .49$ to $.76$) and T2 ($r_s = .45$ to $.72$). The latent variable of emotional insecurity was based on emotional reactivity, behavioral dysregulation, and destructive family representations; these three observed variables loaded on the emotional insecurity latent construct at T1 ($r_s = .55$ to $.77$) and T2 ($r_s = .63$ to $.92$). Each of the three sleep variables (sleep/wake problems, duration, and efficiency) were treated as observed variables. To prevent outlier effects, values that exceeded 4SDs for the marital conflict, emotional insecurity, or sleep variables were set to missing (of note, the entire participant was not removed from analyses). In total, 16 values were set to missing. In addition, the following observed variables were substantially skewed based on skewness (± 2) and kurtosis statistics (± 2) and visual observation of histograms and were natural log transformed: father-initiated physical conflict at T1 and T2 and mother-initiated physical conflict at T1 and T2.

We examined whether changes in emotional insecurity between T1 and T2 (i.e., controlled for autoregressive effects) served as a mediator or intervening variable in the association between marital conflict at T1 and change in children's sleep from T2 and T3 (i.e., controlled for autoregressive effects). In both a mediation model and an intervening variable model, the independent variable (i.e., marital conflict) shares a significant relation with the process variable (i.e., emotional insecurity), which in turn is significantly associated with the outcome variable (i.e., sleep; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Thus,

mediating and intervening variables bridge the association between the independent and dependent variables. In a mediation model but not an intervening model, the relation between the predictor and outcome variable is statistically significant prior to the introduction of the process variable (MacKinnon et al., 2002). Our examination of mediating and intervening processes followed these well-established procedures (Baron & Kenny, 1986; MacKinnon et al., 2002). Further, to account for autoregressive effects, emotional insecurity at T1 was allowed to predict emotional insecurity at T2 and children's sleep at T2 was allowed to predict sleep at T3. Controlling for autoregressive effects helps reduce bias in parameter estimates and allows for conclusions about change in the predicted variable over time (Cole & Maxwell, 2003). Given the sample size and power considerations, each of the three sleep variables were examined in separate models resulting in three total models to examine mediation/intervening effects.

Next, we fit structural equation path models to examine whether emotional insecurity at T1 moderated relations between T1 marital conflict and T2 sleep (while controlling for T1 sleep). Similarly, in separate models we examined whether T2 emotional insecurity moderated relations between T2 marital conflict and children's sleep at T3 (while controlling for T2 sleep; see Figure 1 for an example). Similar to the mediation/intervening analyses each of the three different sleep parameters were examined in separate models, resulting in a total of six fitted models for moderation analyses (three models for the prediction of T2 sleep and three models for the prediction of T3 sleep). In the models, marital conflict at T1, emotional insecurity at T1, and the interaction term "T1 marital conflict x T1 emotional insecurity" were allowed to predict T2 sleep. Similarly, marital conflict at T2, emotional insecurity at T2, and the interaction term "T2 marital conflict x T2 emotional insecurity" were allowed to predict T3 sleep. Following Aiken and West (1991) criteria, interactions were plotted at high (+1 *SD*) and low (-1 *SD*) levels of the predictor and moderator. Preacher, Curran, and Bauer's (2006) interaction utility was used to plot

interactions using estimates obtained from the fitted models. Significant interactions indicate that the slopes are different from each other.

For a stringent examination of our research questions, we considered including control variables known to be associated with sleep. Specifically, because AAs and those from lower SES backgrounds (Buckhalt et al., 2009) often have more sleep problems, race/ethnicity and SES were considered as control variables. The prevalence of sleep problems have also differed among boys and girls (Sadeh et al., 2000), and thus child gender was considered as a covariate. Further, we considered controlling for BMI and puberty because children who have a higher BMI (Snell, Adam, & Duncan, 2007) and those further along in pubertal development (Carskadon, Vieira, & Acebo, 1993) may experience greater sleep problems. Similarly, although findings have not been uniform across studies, the prevalence of marital conflict has differed across race/ethnicity (some research has shown that conflict is more prevalent among AAs whereas other studies have found EAs to have more conflict) and SES backgrounds (for a review see Grych & Fincham, 2001), providing additional support for their consideration as covariates. Finally, because season of year and light exposure can influence sleep (Czeisler et al., 1986; Thorleifsdottir et al., 2002), we considered controlling for average daylight hours during the month of actigraphy data collection, which were calculated by estimating the average sunrise and sunset for that month recorded officially in the area (Edwards, 2007). We used $\Delta\chi^2$ tests to determine whether the control variables were needed in each model; for consistency across models, if the inclusion of a control variable resulted in a significant change in χ^2 in at least one model, the covariate was retained in each additional fitted model. The inclusion of race/ethnicity, SES, and child gender resulted in a significant change in χ^2 in at least one model, and thus only these covariates were retained in the fitted models.

Analyses were conducted using Amos 17. Full information maximum likelihood (FIML) estimation was used to handle missing data. FIML estimation is robust against violations of

normality and is one of the best methods for handling missing data (Acock, 2005). Exogenous variables were allowed to correlate. Residual variances among endogenous variables within the same wave were allowed to correlate. Models were considered an acceptable fit if they satisfied at least 2 of the 3 following criteria: $\chi^2/df < 3$, CFI $> .90$, and RMSEA $< .08$ (Arbuckle & Wothke, 1999; Browne & Cudeck, 1993). Each model satisfied these criteria.

Results

Preliminary Analyses

Means and standard deviations of study variables are presented in Table 1. A considerable amount of verbal and physical marital conflict was reported. At T1 18% of mothers and 20% of fathers reported that their partners used at least one form of physical aggression against them within the last year (similar prevalence rates at T2). Regarding sleep, children's sleep duration (i.e., minutes between bedtime and wake time) was on average 8.5 hours or less per night across all three waves, which is under the National Sleep Foundation's (2011) recent recommendations of roughly 10 to 11 hours for school aged children and 8.5 to 9.25 hours for 13 year old children. At all three waves sleep efficiency was below 90%, which is considered a characteristic of poor sleep quality (Sadeh et al., 2000).

To examine average changes in the main study constructs at the sample level, repeated measures ANOVA was used to assess whether the means among the marital conflict, emotional insecurity, and sleep variables differed across time (Table 1). Notably, of the marital conflict variables, father-initiated verbal conflict increased from T1 to T2. For emotional insecurity, emotional reactivity and behavioral dysregulation each decreased from T1 to T2. Further, self-reported sleep/wake problems at T3 were significantly higher in comparison to T1 and T2. Finally, sleep efficiency increased from T1 to T2.

Bivariate correlations among study variables are presented in Table 2. More marital conflict at T1 was related to greater emotional insecurity at T1 and T2. More marital conflict at

T2 was marginally related to greater emotional insecurity at T2 ($p < .10$). We found only one instance in which marital conflict was related to actigraphy measured sleep (i.e., more marital conflict at T1 was related to a shorter sleep duration at T1), however greater marital conflict was related to higher levels of self-reported sleep/wake problems in several instances both within and across waves. We found some evidence for relations between more emotional insecurity and actigraphy measured sleep problems (i.e., more emotional insecurity at T1 was marginally associated with a shorter sleep duration at T3 and more emotional insecurity at T2 was marginally related to reduced sleep efficiency at T2), but found several instances in which emotional insecurity was related to more sleep/wake problems within and across waves.

Marital Conflict and Children's Sleep over Time: The Intervening Role of Emotional Insecurity

The model fit to examine whether change in emotional insecurity at T2 mediated or intervened in the association between marital conflict at T1 and change in children's self-reported sleep/wake problems at T3 was a good fit to the data $\chi^2 = 93.09$, $p < .05$, $df = 69$; $\chi^2/df = 1.35$; CFI = .95; RMSEA = .05 ns . The autoregressive effect for emotional insecurity was significant from T1 to T2 ($B = .26$, $\beta = .27$, $p < .01$). Similarly, the autoregressive effect for sleep/wake problems was significant from T2 to T3 ($B = .70$, $\beta = .43$, $p < .001$). In total, with all variables in the model 17% of the variance was explained in T2 emotional insecurity and 34% was explained in T3 sleep/wake problems.

Greater marital conflict at T1 predicted increases in emotional insecurity at T2 (while controlling for emotional insecurity at t1; $B = .33$, $\beta = .20$, $p < .05$; $\Delta R^2 = 4\%$). Further, greater emotional insecurity at T2 predicted increases in sleep/wake problems at T3 (while controlling for sleep/wake problems at T2; $B = .20$, $\beta = .18$, $p < .05$; $\Delta R^2 = 4\%$). Because greater marital conflict at T1 was related to increased emotional insecurity at T2, which in turn predicted higher levels of sleep/wake problems at T3, we conducted additional analyses to examine whether

emotional insecurity at T2 mediated or intervened in the association between T1 marital conflict and changes in T3 sleep/wake problems. After temporarily removing T2 emotional insecurity from the model, the association between marital conflict at T1 and sleep/wake problems at T3 was not statistically significant, indicating that change in emotional insecurity at T2 served as an intervening variable versus a mediator in the relation between marital conflict at T1 and change in sleep/wake problems and T3.

We fit two additional models to examine whether emotional insecurity mediated or served as an intervening variable in the association between marital conflict and changes in either sleep duration or sleep efficiency. Marital conflict at T1 was not related to changes in sleep duration at T3 or sleep efficiency at T3. Further, emotional insecurity at T2 was not related to changes in either sleep duration or sleep efficiency at T3. Thus, we did not find evidence that emotional insecurity at T2 served as a mediator or intervening variable in the association between marital conflict at T1 and changes in either sleep duration or sleep efficiency at T3.¹

Marital Conflict and Children's Sleep over Time: Emotional Insecurity as a Moderator of Effects

Sleep/wake problems. We examined whether emotional insecurity moderated the association between marital conflict and children's sleep. We first fit a model to examine whether emotional insecurity at T1 moderated associations between marital conflict at T1 and children's sleep/wake problems at T2 (controlling for autoregressive effects). The model fit the data well: $\chi^2 = 41.39$, *ns*, *df* = 43; $\chi^2 / df = .96$; CFI = 1.00; RMSEA = .00*ns*. For the autoregressive effect, sleep/wake problems were moderately stable between T1 and T2 ($B = .33$, $\beta = .38$, $p < .001$). Regarding direct effects, emotional insecurity at T1 predicted an increase in sleep/wake problems at T2 ($B = .12$, $\beta = .17$, $p < .05$; $\Delta R^2 = 4\%$). No moderation effects were detected. In total, with all variables in the models 25% of the variance was explained in sleep/wake problems at T2.

Next, emotional insecurity at T2 was examined as a moderator of relations between marital conflict at T2 and sleep/wake problems at T3 (while controlling for sleep/wake problems at T2). The model was a good fit to the data: $\chi^2 = 77.54$, $p < .001$, $df = 43$; $\chi^2/df = 1.80$; CFI = .88; RMSEA = .07 ns (Figure 1). Sleep/wake problems was moderately stable over time from T2 to T3 ($B = .61$, $\beta = .35$, $p < .001$). For direct effects, greater marital conflict ($B = .31$, $\beta = .26$, $p < .05$; $\Delta R^2 = 5\%$) and more emotional insecurity at T2 ($B = .24$, $\beta = .20$, $p < .05$; $\Delta R^2 = 4\%$) were associated with an increase in sleep/wake problems at T3. In addition, emotional insecurity at T2 moderated the association between marital conflict at T2 and sleep/wake problems at T3 ($B = .11$, $\beta = .26$, $p < .001$; Figure 2A). When marital conflict at T2 was low, sleep/wake problems at T3 were rather low and similar for all children regardless of emotional insecurity. However, for children with higher levels of emotional insecurity, a positive association between T2 marital conflict and T3 sleep/wake problems was evident. Thus, emotional insecurity at T2 functioned as a vulnerability factor in the link between marital conflict at T2 and sleep/wake problems at T3. The interaction accounted for 9% of unique variance in sleep/wake problems at T3. In total, all of the variables simultaneously explained 50% of the variance in sleep/wake problems at T3.

Sleep duration. The model fit to examine whether emotional insecurity at T1 moderated the relation between marital conflict at T1 and sleep duration at T2 was a good fit to the data: $\chi^2 = 41.28$, ns , $df = 43$; $\chi^2/df = .96$; CFI = 1.00; RMSEA = .00 ns . Sleep duration was moderately stable over time from T1 to T2 ($B = .44$, $\beta = .46$, $p < .001$). For direct effects, neither marital conflict nor emotional insecurity at T1 were directly related to sleep duration at T2. However, emotional insecurity at T1 moderated the association between marital conflict at T1 and sleep duration at T2 ($B = -.73$, $\beta = -.20$, $p < .05$; Figure 2B). When marital conflict was low, sleep duration was similar for all children regardless of their emotional insecurity. However, when conflict was high, children who were more emotionally insecure had a shorter sleep duration than those with lower levels of insecurity. Note that neither slope was statistically different from

zero. The interaction effect accounted for 5% of unique variance in sleep duration at T2. All of the variables simultaneously explained 29% of the variance in sleep duration at T2.

The model fit to examine whether emotional insecurity at T2 moderated associations between T2 marital conflict and T3 sleep duration fit the data well: $\chi^2 = 59.60$, $p < .05$, $df = 43$; $\chi^2/df = 1.39$; CFI = .93; RMSEA = .05 ns . Sleep duration was moderately stable over time from T1 to T2 ($B = .54$, $\beta = .33$, $p < .001$). Neither marital conflict nor emotional insecurity at T2 were directly related to sleep duration at T3. No evidence of moderation was detected. All of the variables simultaneously explained 26% of the variance in sleep duration at T3.

Sleep efficiency. The model fit to examine emotional insecurity at T1 as a moderator of associations between marital conflict at T1 and children's sleep efficiency at T2 was a good fit to the data: $\chi^2 = 40.70$, ns , $df = 43$; $\chi^2/df = .95$; CFI = 1.00; RMSEA = .00 ns . Sleep efficiency was moderately stable from T1 to T2 ($B = .49$, $\beta = .56$, $p < .001$). For direct effects, greater marital conflict at T1 predicted a decreased change in sleep efficiency at T2 ($B = -.26$, $\beta = -.17$, $p < .05$; $\Delta R^2 = 4\%$). Further, emotional insecurity at T1 moderated associations between T1 marital conflict and T2 sleep efficiency ($B = -.10$, $\beta = -.14$, $p < .05$; Figure 2C). For children with higher levels of emotional insecurity, a significant negative association between marital conflict and sleep efficiency was observed; lower levels of sleep efficiency were observed in the context of higher but not lower marital conflict. However, children with lower levels of emotional insecurity tended to have high sleep efficiency regardless of marital conflict. Thus, a lower level of emotional insecurity functioned as a protective factor in this relation. The interaction term accounted for 4% of unique variance in sleep efficiency at T2. In total, all of the variables simultaneously explained 41% of the variance in sleep efficiency at T2.

The model fit to examine whether emotional insecurity at T2 moderated associations between T2 marital conflict and T3 sleep efficiency fit the data well: $\chi^2 = 62.20$, $p < .05$, $df = 43$; $\chi^2/df = 1.45$; CFI = .92; RMSEA = .05 ns . Regarding autoregressive effects, sleep efficiency was

significant from T2 to T3 ($B = .27$, $\beta = .22$, $p < .05$). Neither marital conflict nor emotional insecurity at T2 were directly related to sleep efficiency at T3. No evidence of moderation was detected. In total, all of the variables simultaneously explained 16% of the variance in sleep efficiency at T3.²

Discussion

To gain a better understanding of the longitudinal associations between marital conflict and children's sleep problems, we examined emotional insecurity as an intervening process and as a moderator using a community sample. In an attempt to conduct a rigorous assessment, we had both mothers and fathers report on verbal and physical marital conflict, assessed many parameters of children's sleep using both self-reports and actigraphy, utilized three waves of data spanning five years, and controlled for autoregressive effects. Supportive of intervening processes, marital conflict predicted increases in children's emotional insecurity two years later, which in turn predicted increases in sleep problems three years later. Emotional insecurity also moderated the relations between marital conflict and changes in sleep problems such that children who were more emotionally insecure were at greater risk for sleep problems within the context of higher levels of marital conflict. Collectively, despite stable autoregressive effects, findings demonstrate the important role of emotional insecurity in the association between marital conflict and children's sleep problems.

Our intervening variable model indicated that marital conflict and changes in self-reported sleep/wake problems are indirectly related through their shared association with change in emotional insecurity. Specifically, elevated levels of marital conflict at age 8 were related to increases in emotional insecurity between age 8 and 10, which in turn predicted increases in sleep/wake problems between age 10 and 13. These results build on past cross-sectional findings (El-Sheikh, Buckhalt, Cummings et al., 2007) and indicate that emotional insecurity may be a mechanism in the relation between marital conflict and changes in

children's sleep. Whereas mediation effects require that a significant association exists between the predictor (i.e., marital conflict) and outcome variable (i.e., sleep), intervening processes do not require such direct relations (MacKinnon et al., 2002). In our past research with children who participated in the first two waves of this study we found that marital conflict predicted increases in children's sleep/wake problems two years later (Kelly & El-Sheikh, 2011), thus the absence of a direct association between marital conflict at age 8 and changes in sleep/wake problems at age 13 in the current study was unexpected. However, the five year lag and the stable autoregressive effects for sleep/wake problems may explain why the association between marital conflict and sleep/wake problems was not statistically significant. However, and similar to findings reported in Kelly & El-Sheikh (2011) direct associations were observed between marital conflict and increases in children's sleep/wake problems over shorter increments of time (e.g., marital conflict at age 10 and sleep/wake problems at age 13).

Findings pertaining to emotional insecurity as a process variable in the relation between marital conflict and changes in children's sleep problems may be interpreted within the context of the EST, which suggests that the marital relationship undermines a child's sense of safety and security in the family (Cummings & Davies, 1996, 2010; Davies & Cummings, 1994). Repeated exposure to marital stress including verbal and physical aggression can often result in increased emotional insecurity, which includes feelings of instability about the parents' marital relationship and a lack of safety within the home environment. Children who are emotionally insecure often ruminate about the family's future and may be concerned for their mothers' or fathers' well-being. Consequently, these children often have increased levels of anxiety and fear (Cummings & Davies, 2010), all of which may interfere with the reduction of vigilance needed for obtaining good quality sleep (Dahl, 1996; Dahl & Lewin, 2002). Of note though, the moderation effects observed suggest that not all children exposed to higher levels of marital

conflict are emotionally insecure and that individual differences (or third variables) in this link are operative.

Incorporating biopsychosocial processes, exposure to family conflict may result in heightened sensitivity among the anterior insula and amygdala (McCrory et al., 2011), portions of the brain that play pivotal roles in detecting environmental threats and inducing vigilance (Pichon, de Gelder, & Grezes, 2011). In a recent study utilizing functional magnetic resonance imaging, the anterior insula and amygdala were more highly reactive in response to viewing pictures of angry faces among children from higher rather than lower conflict homes (McCrory et al., 2011). Findings from this body of literature suggest that repeated exposure to environmental danger including family conflict may recalibrate the neural responsiveness within the anterior insula and amygdala to be more sensitive to potential threats. Functionally, while the heightened salience to threat cues may be useful for escaping the occasional chaotic and dangerous threat within the home, the elevated neural activity may disrupt processes that require decreased levels of vigilance including sleep. In addition, the increased sensitivity of the neural network may remain heightened over time, thus potentially putting children at risk for sleep problems even when the threat of marital conflict is absent (e.g., when one parent is not home). Lastly, children's distress in response to marital conflict may be manifested on a physiological level including increased reactivity levels of the stress hormone cortisol (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008), which is an established risk factor for children's sleep problems (El-Sheikh, Buckhalt, Keller, & Granger, 2008; Raikkonen et al., 2010). Obviously, because these biopsychosocial processes were not examined we offer them as tentative explanations pending empirical tests.

Another main objective was to examine the moderating role of emotional insecurity in the relation between marital conflict and children's sleep problems. Supportive of an aggregation of risk hypothesis (Appleyard et al., 2005), analyses revealed that within the context of greater

marital conflict, children who were more emotionally insecure were at increased risk for more self-reported sleep/wake problems, a shorter sleep duration, and decreased sleep efficiency. In contrast, children who were less emotionally insecure tended to have better sleep across multiple parameters regardless of marital conflict. Collectively, this study makes an important advancement in the sleep and family literature (El-Sheikh & Dahl, 2007) by being one of the first to demonstrate that not all children are at equal risk for sleep problems within the context of marital conflict. Rather, emotional insecurity may play a pivotal role in determining the extent to which marital conflict predicts disruptions in both the quantity and quality of children's sleep.

The moderating role of emotional insecurity in the association between marital conflict and children's sleep may be interpreted using the EST (Cummings & Davies, 1996, 2010; Davies & Cummings, 1994). In addition to marital conflict, other factors play important roles in shaping a child's emotional insecurity about the marital system. For instance, the history of exposure to familial conflict (e.g., during early childhood), individual traits (e.g., temperament), family characteristics (e.g., quality of parent-child relationship), and physiological functioning may be pivotal in shaping children's emotional security about the marital system (Cummings & Davies, 2010; Davies & Windle, 2001). Thus, despite living in a high-conflict home, some children may not become emotionally insecure. The elevated feelings of vigilance, fear, and anger, the hallmarks of emotional insecurity, may explain why children who were more emotionally insecure experienced sleep problems within the context of higher levels of marital conflict. In contrast, children who were less emotionally insecure may have been able to maintain more emotion and physiological regulation (e.g., optimal respiratory sinus arrhythmia functioning) in response to family stress and thus their sleep was not affected by marital conflict. Obviously, these explanations are tentative and more research is needed to gain a more in-depth understanding of why emotional insecurity moderates the relation between marital conflict and children's sleep.

Regarding the EST, open scientific questions remain about age effects in terms of the impact that marital conflict and emotional insecurity have on children's outcomes. Cummings and Davies (2010) stated a need to examine relations between marital conflict, emotional insecurity and children's outcomes across different ages. Toward addressing a gap in the literature, we assessed whether marital conflict and emotional insecurity interacted at age 8 to predict sleep problems at age 10 and similarly, whether marital conflict and emotional insecurity interacted at age 10 to predict sleep problems at age 13. Within the context of higher levels of marital conflict, greater emotional insecurity was a vulnerability factor for sleep problems at age 10 and 13. Collectively, our findings are among the first to establish that marital conflict and emotional insecurity can interact to predict child outcomes during the school-aged to early adolescent years.

Findings from the current study have important implications. The average nightly sleep duration was far below the National Sleep Foundation's (2011) recent recommendations. Sleep plays a profound role in children's socio-emotional, behavioral, cognitive, and physical development, so there is a continued critical need for both researchers and clinicians to identify the sources of inadequate sleep. In addition, longitudinal associations were established between marital conflict and children's sleep problems using a community sample, thus even normative levels of conflict should not be overlooked. In other words, children do not need to live in severely violent homes to be impacted by marital aggression. Further, findings illustrate that greater emotional security about the marital system may reduce the likelihood for sleep problems within the context of marital conflict. A focus on developing coping tools and increasing emotional security among children who live in high conflict homes may substantially improve the likelihood of obtaining adequate sleep despite being in the context of marital conflict. Along this line, prevention efforts aimed at altering marital conflict tactics holds particular promise for reducing children's sleep problems.

Regarding study limitations and future directions, the use of a community sample may limit the generalizability of findings. Few studies have examined sleep among children from severely violent homes, yet given that normative types of marital conflict predicted sleep problems in the current study, the probability that children from highly violent homes would have more severe sleep problems is likely. Furthermore, regarding sleep assessments, actigraphy and questionnaires do not have the capability to assess important facets of sleep including sleep staging. A direction for future research is to examine relations between family functioning and polysomnography-derived measures of sleep. Examination of the impact that familial stressors, including marital conflict have on sleep architecture is an important step toward gaining a further understanding of sleep within the family context. Finally, although examining study questions over five years has many advantages, questions remain about the associations between marital conflict and children's sleep across shorter increments of time. For instance, an examination of relations between marital conflict and children's sleep from day-to-day could be complimentary to studies that have used longer time frames.

In sum, this study extends the literature by demonstrating the intervening role of emotional insecurity in the longitudinal relations between marital conflict and children's sleep problems. In addition, moderation findings indicated that emotional insecurity may be a vulnerability factor for sleep problems within the context of marital conflict. Findings add to the family and sleep literature and highlight the importance of considering the role of emotional insecurity.

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Footnotes

¹ We also fit the mediation/intervening models without controlling for autoregressive effects (for emotional insecurity and sleep). Results were nearly identical in nature to those reported while controlling for autoregressive effects.

² We also fit the moderation models without controlling for the autoregressive effects. Results were nearly identical in nature to those reported while controlling for autoregressive effects.

Table 1

Comparison of Means among Observed Study Variables over Time Using Repeated Measures ANOVA

Note. Emotional reactivity, behavioral dysregulation, and destructive family representations are indices of emotional insecurity. T1 =

| Variable name | T1 <i>M</i> (<i>SD</i>) Age 8 | T2 <i>M</i> (<i>SD</i>) Age 10 | T3 <i>M</i> (<i>SD</i>) Age 13 | <i>F</i> -Value |
|---|------------------------------------|-------------------------------------|-------------------------------------|-----------------|
| 1. Father-initiated Verbal Aggression | 6.37 (5.90) ¹ | 7.50 (8.35) | - | 5.04* |
| 2. Father-initiated Physical Aggression | .89 (2.78) | 1.23 (5.69) | - | .51 |
| 3. Mother-initiated Verbal Aggression | 6.10 (6.78) | 5.90 (7.19) | - | .74 |
| 4. Mother-initiated Physical Aggression | 1.13 (3.90) | .86 (4.27) | - | .77 |
| 5. Emotional Reactivity | 21.44 (7.01) ¹ | 16.45 (6.72) | - | 40.77*** |
| 6. Behavioral Dysregulation | 4.88 (1.68) ¹ | 4.10 (1.40) | - | 18.25*** |
| 7. Destructive Family Representations | 8.17 (3.44) | 7.41 (3.27) | - | 4.08* |
| 8. Sleep/Wake Problems ^a | 17.12 (4.92) ² | 15.93 (4.67) ³ | 20.13 (6.70) | 32.32*** |
| 9. Sleep Duration ^b | 8hr. 32min (37 min) | 8hr. 20min (38 min) | 8hr. 18min (57 min) | 2.51 |
| 10. Sleep Efficiency ^b | 86.60 (8.20) ¹ | 89.15 (9.31) | 87.85 (8.20) | 4.44* |

data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3. Marital conflict and emotional insecurity were not assessed at T3.

^aChild-reported sleep measure. ^bActigraphy-derived sleep measure.

¹. Statistically significant difference between means at T1 and T2. ². Significant difference between T1 and T3. ³. Significant difference between T2 and T3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2

Correlations among Study Variables

| | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. | 11. | 12. | 13. |
|---|-------|-------|-------|-------|------|------|-------|------|------|------|------|------|-----|
| 1. Marital Conflict T1 | - | | | | | | | | | | | | |
| 2. Emotional Insecurity T1 | .20* | - | | | | | | | | | | | |
| 3. Sleep/Wake Problems ^a T1 | .06 | .29* | - | | | | | | | | | | |
| 4. Sleep Duration ^b T1 | -.17* | -.03 | -.09 | - | | | | | | | | | |
| 5. Sleep Efficiency ^b T1 | -.01 | -.09 | -.06 | .19* | - | | | | | | | | |
| 6. Marital Conflict T2 | .98* | .08 | .03 | -.15* | -.02 | - | | | | | | | |
| 7. Emotional Insecurity T2 | .27* | .35* | .26* | -.12 | -.07 | .16† | - | | | | | | |
| 8. Sleep/Wake Problems ^a T2 | .26* | .27* | .38* | -.01 | -.08 | .23* | .42* | - | | | | | |
| 9. Sleep Duration ^b T2 | -.09 | -.06 | -.18* | .49* | .05 | -.03 | -.04 | -.07 | - | | | | |
| 10. Sleep Efficiency ^b T2 | -.13 | -.01 | -.09 | .14 | .56* | -.01 | -.14† | -.05 | -.02 | - | | | |
| 11. Sleep/Wake Problems ^a T3 | .22* | .24* | .22* | .05 | .01 | .26* | .32* | .52* | .01 | -.06 | - | | |
| 12. Sleep Duration ^b T3 | -.05 | -.19† | .07 | .28* | .14 | .11 | -.02 | .01 | .40* | .12 | -.01 | - | |
| 13. Sleep Efficiency ^b T3 | -.03 | -.06 | -.02 | .01 | .13 | -.01 | -.07 | -.13 | -.07 | .22* | .02 | .19† | - |

Note.

Marital conflict and emotional insecurity are latent constructs. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3.

^aChild-reported sleep measure. ^bActigraphy-derived sleep measure.

† $p < .10$. * $p < .05$.

Figure 1

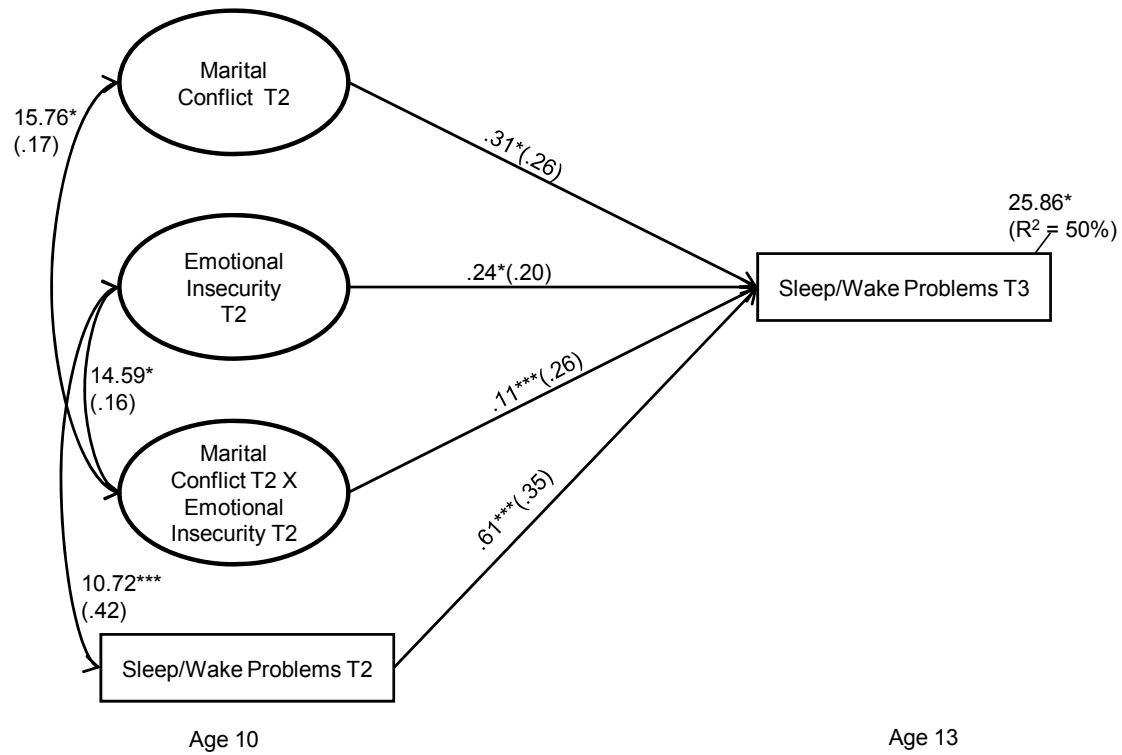
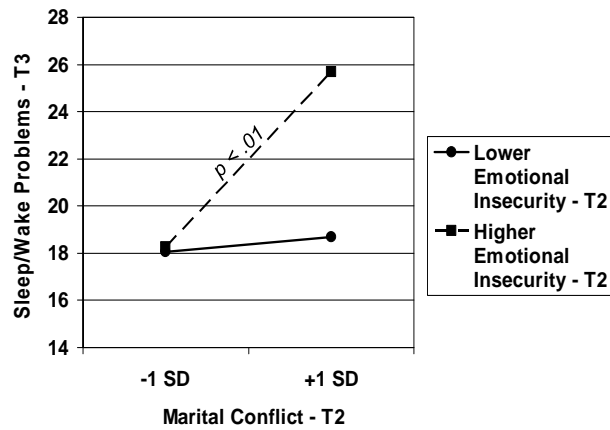


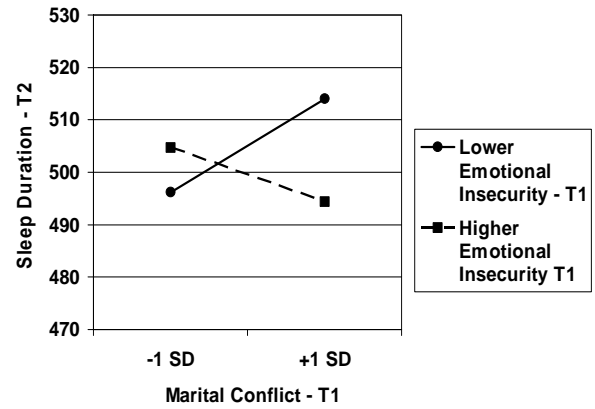
Figure 1. Examination of emotional insecurity at T2 as a moderator of associations between marital conflict at T2 and children's sleep/wake problems at T3. Unstandardized and standardized coefficients (in parentheses) are provided. Child gender, race/ethnicity, and SES are controlled in analyses. T2 = data collected at Time 2; T3 = data collected at Time 3. Model Fit: $\chi^2 = 77.54$, $df = 43$, $p < .001$; $\chi^2/df = 1.80$; CFI = .88; RMSEA = .07 ns . * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2.

A.



B.



C.

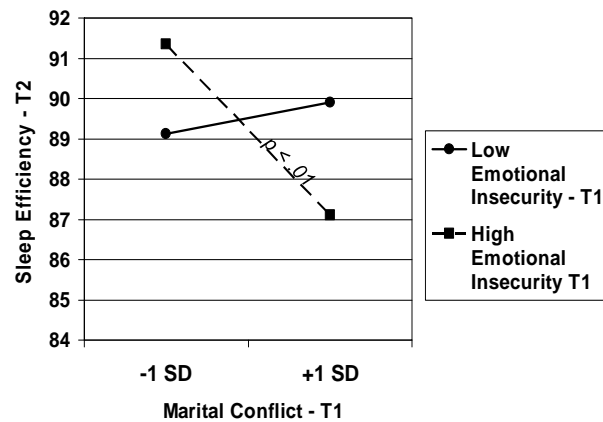


Figure 2. Emotional insecurity as a moderator of relations between marital conflict and children's sleep. For slopes that differ from zero, the p value is presented next to the slope. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3. On average, children were 8 years old at T1, 10 years old at T2, and 13 years old at T3.

Abstract Manuscript 2

We examined reciprocal relations between children's sleep and their internalizing and externalizing symptoms using three waves of data spanning five years. Participants included 176 children at T1 ($M = 8.68$ years), 141 children at T2 ($M = 10.70$ years) and 113 children at T3 ($M = 13.60$ years). Children's sleep was measured subjectively via self-reports and objectively via actigraphy. Children and parents reported on children's adjustment. Cross-lagged panel models were fit to examine whether sleep problems predicted changes in internalizing and externalizing symptoms over time. Examining reciprocal relations, we also assessed internalizing and externalizing symptoms as predictors of changes in sleep problems longitudinally. Reduced sleep amount and worse sleep quality predicted increases in depression, anxiety, and externalizing symptoms over time. To a lesser extent but supportive of reciprocal relations, more internalizing and externalizing symptoms predicted increases in sleep problems longitudinally. In sum, the cyclical nature between sleep problems and adjustment difficulties are demonstrated among otherwise normally developing youth.

Reciprocal Relations between Children's Sleep and their Adjustment over Time

Sleep problems among otherwise normally developing school-aged children are highly common (National Sleep Foundation, 2004) and are related to adjustment problems (Alfano, Zakem, Costa, Taylor, & Weems, 2010; Arman et al., 2011). Although researchers have shed much needed light on the association between children's sleep problems and their adjustment, several important gaps remain. Not much is known about the direction of effects between sleep problems and children's adjustment and thus, questions remain about potential reciprocal relations (i.e., whether sleep predicts increases in adjustment and/or vice versa). Among the few longitudinal studies that have examined these relations, the majority have focused on sleep as a predictor of change in adjustment (El-Sheikh, Kelly, Buckhalt, & Hinnant, 2010, conducted with the first two study waves of the present sample; Gregory & O'Connor, 2002) and not the reverse direction (i.e., adjustment as a predictor of sleep problems). In an attempt to further clarify this association, the main objective of this study is to examine reciprocal relations between children's sleep problems and their behavioral and emotional adjustment over time. Further, few studies have investigated these associations with objective sleep measures, multiple informants of children's adjustment, or multiple-wave designs. This investigation builds on the existing literature by examining sleep problems both subjectively via child reports and objectively via actigraphy in a large community sample utilizing three waves of data spanning five years and utilizing a multi-informant approach.

We examined normative sleep problems that are common among otherwise typically developing youth including short sleep duration (Pesonen et al., 2010), nighttime awakenings (Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009), and erratic sleep schedules (Hale, Berger, LeBourgeois, & Brooks-Gunn, 2009; Henderson, Barry, Bader, & Jordan, 2011). These sleep problems are related to children's developmental outcomes (El-Sheikh et al., 2010), which

highlights the importance of their examination in sleep and child development research. We examined sleep parameters along a continuum (versus dichotomizing children into groups).

Acquisition of adaptive socioemotional behaviors is an important developmental task with immediate and long-term consequences and indications are that sleep plays an important role in this development. In community samples, sleep problems have been concurrently associated with children's internalizing behaviors including depression symptoms (Chorney, Detweiler, Morris, & Kuhn, 2008; El-Sheikh, Buckhalt, Cummings, & Keller, 2007, based on the first study wave with the present sample) and anxiety (Alfano, Pina, Zerr, & Villalta, 2010; Gregory & Eley, 2005) as well as with externalizing symptoms including aggression, conduct problems, and delinquency (Chervin, Dillon, Archbold, & Ruzicka, 2003; Pesonen et al., 2010; Sadeh, Gruber, Raviv, 2002). In a recent review article, children who reported greater levels of sleep problems also reported more depression and anxiety symptoms (Alfano, et al., 2010). Similarly, shorter sleep duration, as measured by actigraphy, has been associated with higher levels of externalizing symptoms including aggression and conduct problems (Aronen, Paavonen, Fjallberg, Soininen, & Torronen, 2000; Holley, Hill, & Stevenson, 2011). Stein, Mendelsohn, Obermeyer, Amromin, and Benca (2001) found that parent reports of children's symptoms of insomnia were correlated with higher levels of anxiety, depression, and externalizing symptoms. Overall, these studies have demonstrated that sleep problems are related to many adjustment problems in community samples; however the cross-sectional designs limit inferences about the directionality of effects. Just as children's sleep problems may predict adjustment problems, adjustment may have preceded sleep problems.

In community samples, a growing body of literature has demonstrated children's sleep problems to predict longitudinally increases in adjustment problems (e.g., El-Sheikh et al., 2010; El-Sheikh, Bub, Kelly, & Buckhalt, under review; Fredriksen, Rhodes, Reddy, & Way, 2004; Goodnight, Bates, Staples, Pettit, & Dodge, 2007; Gregory et al., 2005; Wong, Brower, &

Zucker, 2009). Utilizing the first two waves of the present sample, El-Sheikh and colleagues (2010) found that more self-reported sleep/wake problems at age eight were associated with increased levels of internalizing and externalizing symptoms two years later, even after controlling for autoregressive effects or earlier adjustment. In another study, a general measure of parent-reported child sleep problems (e.g., experiences nightmares, sleeps less than most children, talks in sleep, overtired) in childhood predicted an increase in depression/anxiety symptoms and aggression during adolescence, while controlling for initial sleep problems (Gregory & O'Connor, 2002). Further, shorter sleep duration (as measured by one self-report item) predicted an increase in children's depression symptoms three years later (Fredricksen et al., 2004). In a three-wave study that spanned three years with school-aged children, El-Sheikh et al. (under review; unique sample from the current study) found that initial levels and increases in self-reported sleep/wake problems over time predicted elevated levels of depression symptoms, anxiety, and externalizing symptoms in the final wave.

Theory to guide research in this domain is in a developing stage. Dahl (1996) proposed that the prefrontal cortex (PFC) plays an important role in the relation between children's sleep problems and their behavioral and emotional regulation. Sleep problems can disrupt processes mediated in the PFC, including executive functioning needed for controlling cognition and emotion (Jones & Harrison, 2000; Muzur, Pace-Schott, & Hobson, 2002), which in turn may put children at risk for both internalizing and externalizing problems (Eisenberg et al., 2001). Further, sleep plays a pivotal role in emotional memory formation and even slight sleep problems may impair the ability to recall emotionally related experiences (Yoo, Hu, Gujar, Jolesz, & Walker, 2004). However, research from cognitive neuroscience has indicated that negative memories are more resistant to the effects of sleep loss compared to positive or neutrally themed memories (Phelps, 2004; Walker & van der Helm, 2009). The retention of negative information and loss of positive or even neutrally themed information from sleep

deprivation may be risk factors for adjustment difficulties (Walker & van der Helm, 2009). Taken together, the consequences of sleep problems on cognitive and emotional regulation in addition to emotional memory formation are thought to jeopardize children's development.

Supportive of effects in the reverse direction, conceptual evidence has indicated that children's emotional and behavioral adjustment predict increases in sleep problems over time. The emotional arousal that is often characteristic of depression, anxiety, and externalizing symptoms may make sleep difficult to attain (Dahl, 1996; Dahl & Lewin, 2002). For instance, symptoms of depression, such as a change in affect, ruminating thought processes, and increased emotional distress may interfere with the relaxation and reduction of vigilance needed to attain sleep (Dahl, 1996; Dahl & Lewin, 2002; Patten, Choi, Gillin, & Pierce, 2000). Similarly, feelings of fear, worry, and alertness, the hallmarks of anxiety, may prevent individuals from being able to reduce arousal in order to sleep (Dahl, 1996; Dahl & Lewin, 2002). From a biopsychosocial perspective, children who exhibit internalizing symptoms frequently have elevated cortisol levels at night (Forbes et al., 2006), which is a risk factor for sleep problems (El-Sheikh, Buckhalt, Keller, & Granger, 2008; Raikkonen et al., 2010). In addition, research has shown that children who exhibit externalizing symptoms have a higher skin conductance level, a marker of sympathetic nervous system activity (El-Sheikh, 2005; Hubbard et al., 2002, 2004). Although there is no consensus in the literature on the relations between sympathetic nervous system (SNS) activity and sleep problems (El-Sheikh & Arsiwalla, 2011; Holmes, Burgess, & Dawson, 2002), effects of greater SNS activity include increased sensory acuity to facilitate assessment and engagement with the environment, increased blood pressure and heart rate to facilitate movement, and increased perspiration (Beauchaine, 2001; Boucsein, 1992), all of which may interfere with the reduction of vigilance needed to sleep (Dahl, 1996; Dahl & Lewin, 2002).

The majority of studies that have examined the impact of adjustment problems on sleep have utilized samples characterized by clinical levels of internalizing problems including depression (Cousins et al., 2011; Ivanenko, Crabtree, & Gozal, 2005; Forbes et al., 2008; Williamson et al., 1995), anxiety (Alfano, Beidel, Turner, & Lewin, 2006; Alfano, Ginsburg, & Kingery, 2007; Alfano et al., 2010; Hudson, Gradisar, Gamble, Schniering, & Rebelo, 2009) and externalizing problems (Dahl, 2006; Ireland & Culpin, 2006; Sneddon, 2007). For example, Bertocci et al. (2005) found that children and adolescents with major depression disorder self-reported more sleep problems including number of awakenings, minutes awake, and more trouble waking compared to healthy controls. In another study, children with clinically-diagnosed anxiety disorders reported more sleep problems including a shorter sleep duration compared to children without anxiety disorders (Hudson et al., 2009). Finally, mothers of clinically referred children with externalizing problems reported more child sleep problems including frequent night wakings and a shorter sleep duration than did mothers of children without behavior problems (Sneddon, 2007). Taken together, these studies indicate clinical levels of emotional or behavioral problems are risk factors or correlates of a wide range of sleep problems in children.

Very few studies have used to community samples to examine whether less severe emotional and behavioral problems predict increases in sleep problems. Among the few existing studies, findings have been mixed. Patten et al. (2000) found that children who had elevated levels of depression symptoms were more likely to report sleep problems (measured with one item: "During the past 12 months how often have you had trouble going to sleep or staying asleep") four years later. However, Gregory and O'Connor (2002) examined whether depression/anxiety symptoms during childhood predicted self-reported sleep problems during adolescence and found no such evidence. Further, Johnson, Roth, and Breslau (2006) found that retrospective reports of anxiety were a risk factor for current self-reported sleep problems among children, but retrospective reports of depression were not related to current sleep

problems. In one of the few studies to examine longitudinally the reciprocal relations between children's sleep problems and their adjustment, Gregory, Rijsdijk, Lau, Dahl, and Eley (2009) found that parent-reported child sleep problems at age 8 predicted an increase in self-reported depression symptoms two years later but no evidence was found for the reverse direction (i.e., depression symptoms as predictors of increases in sleep problems). In a similar study, the reciprocal relations were examined between sleep problems and daytime affect across 4-day intervals among normally developing youth ages 8 to 16, but no such significant associations were found for either direction of effects (Cousins et al., 2011).

Overall, the aforementioned findings have been inconsistent, which limits conclusions about bidirectional associations. In addition, mono-reporter bias for adjustment and sleep measures, small sample sizes, a lack of objective measures of sleep (e.g., actigraphy), and fewer samples of typically developing school-aged children may limit the generalizability of some of these findings. Lastly, no study to our knowledge has used a community sample to investigate whether externalizing symptoms predict later sleep problems, and thus a gap exists in the literature regarding whether the presence of behavior problems contributes to increases in sleep problems among otherwise typically developing youth.

The Current Study

Addressing a notable gap in the literature, the main objective of the current study was to examine longitudinally the reciprocal relations between children's sleep problems and their emotional and behavioral adjustment. For a more thorough elucidation of relations, we examined a range of sleep parameters: quantity, quality, and variability in sleep schedule. Likewise, we assessed several adjustment variables including symptoms of depression, anxiety, and externalizing behaviors. We used cross-lagged panel models, which allow for the investigation of reciprocal relations between two variables over time and the determination of which variable is a more robust predictor (i.e., whether sleep problems are more robust

predictors of adjustment problems or vice versa). Specifically, sleep at each time point was allowed to predict adjustment at the following time point. Similarly, adjustment at each wave was allowed to predict sleep at the following time point.

In our assessment of children's sleep and consistent with recent recommendations (Sadeh, 2008; 2011), we used both children's self-reports and actigraphy. While actigraphy has the capability to assess important information regarding actual sleep amount (e.g., minutes spent asleep) and quality (e.g. night awakenings), subjective reports of sleep problems are useful due to individual differences in the amount and quality of needed sleep for optimal functioning (e.g., some children require 9 hours of sleep whereas others require 10 hours). In addition, subjective reports can yield information on different facets of sleep, which actigraphy cannot provide such as daytime sleepiness or trouble waking in the morning (Sadeh, 2011).

To address our research questions, we used three waves of data spanning five years with a two year lag between T1 (age eight) and T2 (age ten) and a three year lag between T2 and T3 (age thirteen). We hypothesized that children's sleep problems would predict increases in depression, anxiety, and externalizing symptoms over time. Because of developmental effects, sleep problems may have a more robust influence on adjustment as children enter the adolescent years (Chorney et al., 2008; Gregory & O'Connor, 2002). Consequently, to build on this small body of literature we examined whether sleep problems at age ten were more robust predictors of later adjustment compared to sleep problems at age eight. Supportive of reciprocal relations, we also expected that depression, anxiety, and externalizing symptoms would predict increases in sleep problems. Given the novelty of our research question and the dearth of research that has examined reciprocal relations between children's sleep and their adjustment, we had no expectations regarding which parameter (adjustment or sleep) would be a more robust predictor of the other. Lastly, internalizing and externalizing symptoms frequently co-occur and are highly comorbid (Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003). For a more

stringent examination of research questions and consistent with recent recommendations (Coulombe, Reid, Boyle, & Racine, 2011), we simultaneously examined depression, anxiety, and externalizing symptoms to better ascertain the unique association that children's sleep shares with each adjustment variable. Because few studies have simultaneously examined depression, anxiety, and externalizing symptoms, we had no hypotheses regarding whether sleep problems would more robustly influence one adjustment variable over the others. Similarly, we had no expectations for whether one adjustment variable would be more strongly predictive of sleep problems compared to the other adjustment variables.

Method

Participants

This study consisted of three waves. Of note, examinations of relations between sleep and adjustment using the first two waves of data have been reported in prior papers (El-Sheikh et al., 2007; 2010). At T1, 3rd grade children were recruited from public schools in the Southeastern United States. We contacted parents using home telephone numbers provided by local schools. In a few cases, some interested families initiated contact with our research laboratory. Inclusion criteria were based on mothers' reports and included the child living in a two-parent home, and having no history or diagnosis of a sleep problem, attention deficit hyperactivity disorder, chronic or acute physical illness, or a learning disability. Of those families who were contacted and satisfied our inclusion criteria, 66% agreed to participate and were included in the study; 34% declined participation.

T1 participants included 176 children (78 boys, 98 girls) with an average age of 8.68 years ($SD = .36$), along with their parents. Mothers' reports on the Puberty Development Scale (1 = prepubertal, 2 = early pubertal, 3 = midpubertal, 4 = late pubertal, 5 = postpubertal; Petersen, Crockett, Richards, & Boxer, 1988), indicated that on average both boys ($M = 1.25$, $SD = .23$) and girls ($M = 1.45$, $SD = .34$) were prepubertal.

Regarding the ethnic composition of the sample, 69% of children were European American (EA) and 31% were African American (AA); these percentages are representative of the community. Regarding annual family income, 11% earned between \$10,000 and \$20,000; 24% earned between \$20,000 and \$35,000; 29% earned between \$35,000 and \$50,000; 25% earned between \$50,000 and \$75,000; and 11% earned more than \$75,000. The mean family income was in the \$35,000 to \$50,000 range. For mothers' and fathers' education, 9% and 13% had partial high school education or less, 36% and 46% received a high school diploma, 42% and 30% had partial college education or received specialized training, 11% and 9% received a college diploma, and 2% and 2% earned a graduate degree or received professional training, respectively. An objective was to recruit both EA and AA families across a wide range of income backgrounds through oversampling, however race/ethnicity and income were still moderately correlated ($r = -.37$; race/ethnicity was dummy coded such that AA = 1 and EA = 0). Preliminary analyses indicated that family income was related to mothers' ($r = .29, p < .001$) and fathers' education ($r = .35, p < .001$), and in addition mothers' and fathers' education were associated ($r = .32, p < .001$). Consequently, we standardized and mean composited family income, mothers' education, and fathers' education to create an overall socioeconomic status (SES) variable. Mention of an SES variable throughout the remainder of this paper is in reference to this composited variable.

Approximately 2 years later (M time lag between T1 and T2 = 1 year and 361 days; $SD = 111$ days), 142 of the children returned for a second wave of data collection (T2; 81% of the original sample). At T2, 62 boys and 80 girls (70% EA, 30% AA) participated. Children's average age was 10.70 years ($SD = .55$) at T2. Average puberty status at T2 was 2.06 ($SD = 0.55$) for girls and 1.49 ($SD = 0.42$) for boys, which indicates an early pubertal status for girls and prepubertal status for boys (Petersen et al., 1988).

Children participated in a third wave of data collection (*M* time lag between T2 and T3 = 2 years and 337 days, *SD* = 119 days). Of the 176 families that participated at T1, 113 participated (50 boys and 63 girls) at T3 (80% of those who participated at T2; 64% of original sample). At T3, children's average age was 13.60 years (*SD* = .76); 70% were EA and 30% were AA. At T3, the mean puberty score for boys was 2.27 (*SD* = .65) and for girls was 3.18 (*SD* = .47), which corresponds with early pubertal status for boys and midpubertal status for girls (Petersen et al., 1988). Reasons for attrition at T2 and T3 included having a busy schedule, moving out of the study area, disinterest in continued participation, and inability to be located.

Independent samples *t*-tests and chi-square analyses were used to examine potential selective attrition over study waves. First, comparisons were made between individuals who participated at T1 and T2 versus those who only participated at T1. In comparison to children who participated at T1 and T2, those who only participated at T1 came from lower SES households, $t(162) = -2.18, p < .05$; there were no significant differences based on gender, race/ethnicity, pubertal status, child age, body mass index (BMI), sleep, or children's emotional or behavioral adjustment. Second, we compared children who participated at T3 and those who discontinued participation after T2; no significant differences between retained and attrited participants were found.

Procedure

At each wave of data collection, a research assistant instructed parents to place an actigraph (Ambulatory Monitoring Inc., Ardsley, NY) on the child's non-dominant wrist prior to bedtime for seven consecutive nights. To validate the actigraphy measured sleep variables, parents were called each night to report child bed- and wake-times. Actigraphy data were collected during the regular school years at T1 and T2, and in the summer during T3. To reduce confounds, only data from medication-free nights were used in analyses.

In most cases, families came to our university based research laboratory on the day following the last night of actigraphy sleep assessment (T1 M days = ± 1.75 , $SD = \pm 8.30$ days; T2 $M = \pm 13.19$, $SD = \pm 44.74$; T3 $M = \pm 1.15$, $SD = \pm 5.18$). During the visit, both children and parents completed several questionnaires. Children were administered each questionnaire by a trained research assistant. For privacy, parents completed questionnaires in separate rooms. In order to calculate BMI (based on criteria established by Must, Dallal, & Dietz, 1991), children's height and weight were measured. We took into account BMI given that a higher BMI has been linked to greater sleep problems among children (Snell, Adam, & Duncan, 2007). Procedures and measures are identical for each of the three waves unless otherwise specified.

Measures

Children's self-reports of sleep. Children reported on their own sleep using the Sleep Habits Survey (SHS; Wolfson & Carskadon, 1998). The SHS has demonstrated adequate reliability and validity (Wolfson et al., 2003) and has been used in many studies to examine the sleep of elementary school-aged children (e.g., Amschler & McKenzie, 2005; Buckhalt, El-Sheikh, Keller, & Kelly, 2009; El-Sheikh & Buckhalt, 2005). The 10-item Sleep–Wake Problems scale was pertinent and assessed trouble with falling asleep at bedtime and falling back to sleep after waking during the night as well as difficulties waking up; higher scores reflect more problems. Likert type response choices for each item ranged from (1) never to (5) everyday/night. Cronbach's alpha = .71 at T1, .79 at T2, and .74 at T3.

Actigraphy measures of children's sleep. Actigraphs were used to record children's activity between bedtime and wake time. Actigraphy is frequently used as a proxy for sleep duration and quality, and has demonstrated reliability, especially when used for multiple consecutive nights (Acebo et al., 1999; Sadeh et al., 2002). The actigraphs were Octagonal Basic Motionloggers (Ambulatory Monitoring Inc., Ardsley, NY), and being awake or asleep was determined with Sadeh's scoring algorithm (Sadeh, Sharkey, & Carskadon, 1994). Procedures

for setting times for sleep onset and wake time followed a laboratory protocol developed at the E.P. Brown Sleep Laboratory at Brown University (Acebo & Carskadon, 2001); parents completed daily sleep logs to cross validate sleep times. The actigraphs and software package used in this study are reliable and valid based on comparisons with polysomnography derived sleep measures (Sadeh et al., 1994).

The assessment of multiple sleep parameters is recommended when using actigraphy to tap a wide range of sleep problems (Sadeh et al., 2000). In this study, the following sleep variables were assessed: (a) Sleep Minutes – the number of minutes scored as sleep during the sleep period (sleep onset time to wake time); (b) Sleep Efficiency – percentage of epochs scored as sleep between sleep onset and offset; (c) Sleep % - the percent of minutes that were scored as sleep during the total time in bed; (d) Long Wake Episodes – number of wake episodes during the night that last at least five minutes; and (e) Minutes Awake After Sleep Onset – the number of minutes scored as awake between sleep onset and offset. Each of these sleep variables were created by computing the average across all available nights. Further, (f) Variability in Sleep Onset was computed using the mean-centered coefficient of variance statistic (Snedecor & Cochran, 1967) and represents the variability in sleep onset time across the week of actigraphy.

At T1, 75% of children had actigraphy data for all seven nights, 14% had data for six nights, 8% had data for five nights, and 3% had data for four nights or fewer ($M = 6.59$ days, $SD = .85$). For T2, 56% of participants had actigraphy data for all seven nights, 25% had data for six nights, 14% had data for five nights, and 5% had data for four nights or fewer ($M = 6.30$ days, $SD = .95$). At T3, 66% of children had actigraphy data for all seven nights, 20% had data for six nights, 6% had data for five nights, and 8% had data for four nights or fewer ($M = 6.33$ days, $SD = 1.03$). Reasons for missing data included the use of allergy medicine (only medication free

nights were used in analyses) and exclusion of these nights from analyses, forgetting to wear the actigraph, and mechanical actigraphy problems.

Using intraclass correlations, we computed stability estimates for the various sleep parameters using all available data (i.e., maximum number of nights with valid actigraphy data). Alphas were as follows: Sleep Minutes ($\alpha = .78$ at T1; $.86$ at T2; $.79$ at T3); Sleep Efficiency ($\alpha = .87$ at T1; $.92$ at T2; $.81$ at T3); Sleep % ($\alpha = .86$ at T1; $.92$ at T2; $.90$ at T3); Long Wake Episodes ($\alpha = .85$ at T1; $.91$ at T2; $.85$ at T3); Minutes Awake After Sleep Onset ($\alpha = .88$ at T1; $.91$ at T2; $.87$ at T3); and Variability in Sleep Onset ($\alpha = .83$ at T1; $.79$ at T2; $.81$ at T3). All reliability estimates surpassed Acebo et al.'s (1999) recommended stability level of $.70$. Based on these coefficients, each sleep parameter appears stable across the week and was composited for analyses (i.e., across all available nights).

Internalizing symptoms. Children completed the Children's Depression Inventory (CDI; Kovacs, 1985); one item regarding suicidal ideation was excluded. For each item, children chose the statement that best described their feelings during the past two weeks (e.g., "I am sad once in a while," "I am sad many times," "I am sad all the time"). The CDI is a well-established measure and has demonstrated test-retest reliability and discriminant validity. For this study, $\alpha = .89$ at T1, $.77$ at T2, and $.81$ at T3. Scores of 20 or above on the CDI indicate potential clinical levels of depression (Kovacs, 1992). In our sample, three children at T1, one child at T2, and one child at T3 had scores that surpassed the clinical cutoff.

Children reported on general levels of anxiety via the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978). The RCMAS has demonstrated test-retest reliability (Wisniewski, Mulick, Genshaft, & Coury, 1987) and concurrent validity (James, Reynolds, & Dunbar, 1994). Children rated whether statements are true about them (yes/no). Cronbach's alphas = $.90$ at T1, $.89$ at T2, and $.82$ at T3. Although the RCMAS does not provide a clinical cutoff, researchers have recommended that children who fall beyond 2 *SDs* from the

mean should receive a follow up (Reynolds & Richmond, 1985). In our sample, four children at T1, four children at T2, and seven children at T3 met this criterion.

Externalizing symptoms. Mothers and fathers completed the widely used 280-item Personality Inventory for Children (PIC; Wirt, Lachar, Klinedinst, & Seat, 1990). The Externalizing scale, which measures symptoms including aggression, impulsivity, disruptive behavior, delinquency, and noncompliance, was used. The PIC has demonstrated very good psychometric properties (Wirt et al., 1990). Cronbach's alphas ranged from .89 to .96 across waves and across mother and father reports. Because the PIC *T* scores are age and gender corrected, raw scores were used in longitudinal analyses. Based on mother reports, 17 children at T1, nine at T2, and six at T3 surpassed the clinical cutoff *T* score of 65 (Lachar & Gruber, 1995). Similarly, based on father reports, 16 children at T1, seven at T2, and three at T3 surpassed the clinical cutoff.

Plan of Analysis

Cross-lagged panel models were fit to examine whether children's sleep predicts changes in emotional and behavioral development over time and conversely, whether children's emotional and behavioral problems predict sleep problems longitudinally. The sleep variables included reported sleep/wake problems (observed variable). In addition, the following actigraphy-based variables were examined: sleep minutes (observed variable), poor sleep quality (latent construct composed of sleep efficiency, sleep %, long wake episodes, and minutes awake after sleep onset), and variability in sleep onset (observed variable). The adjustment variables used in analyses were: depression symptoms (observed variable), anxiety symptoms (observed variable), and externalizing symptoms (latent construct composed of mothers and fathers reports of externalizing symptoms). Children's depression symptoms, anxiety symptoms, and externalizing symptoms were examined simultaneously in each model. Each of the four sleep variables/constructs were examined in separate models resulting in a

total of four cross-lagged panel models. To reduce outlier effects, data points that exceeded 4SDs among either the sleep or adjustment variables were removed and set to missing (of note, the entire participant was not removed from analyses); 12 data points were set to missing. Of note, models were fit before outliers were removed as well; no major differences were detected before or after outlier removal. In addition, the following observed variables were skewed based on skewness (± 2) and kurtosis statistics (± 2) in addition to visual observation and were natural log transformed: T1 sleep %, T3 depression symptoms, mother reported externalizing symptoms at all three waves, and father reported externalizing symptoms at T1.

The two latent constructs of poor sleep quality and externalizing symptoms showed excellent measurement properties across all three waves. The latent variable of sleep quality was based on actigraphy-derived sleep efficiency, sleep %, long wake episodes, and minutes awake after sleep onset. These four observed variables loaded well on the poor sleep quality latent construct at T1 ($r_s = .80$ to $.95$), T2 ($r_s = .68$ to $.99$), and T3 ($r_s = .93$ to $.99$); higher scores reflect poorer sleep quality. The latent variable of externalizing symptoms was based on mothers and fathers reports of externalizing symptoms; these two observed variables loaded well on the latent construct of externalizing symptoms at T1 ($r_s = .82$ to $.98$), T2 ($r_s = .79$ to $.96$), and T3 ($r_s = .92$ to $.99$).

For each cross-lagged panel model, there were four variables at each time point, one for sleep, one for depression symptoms, one for anxiety symptoms and one for externalizing symptoms. Each of the four variables at one time point was allowed to predict the same variable at the next time point (see Figure 1 for an example). These estimates provide information about the stability of each construct over the three waves and result in controlling for the autoregressive effect. Controlling for autoregressive effects helps reduce bias in parameter estimates and allows for conclusions about change in the predicted variable (Cole & Maxwell, 2003). Further, in each model sleep was allowed to predict each adjustment variable at the

following wave. Similarly, for the assessment of reciprocal relations, the adjustment variables at each time point were allowed to predict sleep at the following wave (see Figure 1). These estimates illustrate the cross-lagged cascade effects of these constructs over the three waves of data. In the models, residual variances among exogenous variables within the same wave were allowed to correlate. The adjustment and sleep variables were allowed to covary at T1. Models were considered an acceptable fit if they satisfied at least two of the three following criteria: $\chi^2/df < 3$, CFI $> .90$, and RMSEA $< .08$ (Arbuckle & Wothke, 1999; Browne & Cudeck, 1993). Each of the four models satisfied these criteria.

To address whether sleep problems were more strongly related to adjustment as children became older, $\Delta\chi^2$ tests were conducted in the cross-lagged panels models to assess whether sleep problems at T2 (age 10) were more robust predictors of subsequent adjustment compared to the association between sleep problems at T1 and subsequent adjustment (age 8; the magnitude of effects among statistically significant associations were compared). Similarly, $\Delta\chi^2$ tests were used to determine whether sleep problems were more robustly associated with one adjustment variable compared to the others (e.g., does sleep more strongly predict depression versus anxiety symptoms?). Finally, $\Delta\chi^2$ tests were used to compare the magnitude of effects to assess whether sleep more strongly predicted adjustment or vice versa.

For a stringent examination of our research questions in the models, we considered including control variables known to be associated with sleep. Specifically, because the prevalence of sleep problems may differ among boys and girls (Sadeh et al., 2000), gender was considered as a control variable. Further, AAs (compared to EAs) and those from lower SES backgrounds (compared to their higher SES counterparts) have been shown to have more sleep problems (Buckhalt, El-Sheikh, Keller, & Kelly, 2009). Thus race/ethnicity and SES were considered as control variables. We also considered controlling for body mass index (BMI) and

puberty because children who have a higher BMI (Snell et al., 2007) or are further along in pubertal development (Carskadon, Vieira, & Acebo, 1993) often experience greater sleep problems. Similarly, although findings have not been uniform across studies, children's internalizing and externalizing symptoms have been shown to differ across race/ethnicity (McLaughlin, Hilt, & Nolen-Hoeksema, 2007), SES (Keegan, 2000), and gender (Leadbeater, Kuperminc, Blatt, & Hertzog, 1999), providing additional support for their consideration as covariates. Finally, because season of year and light exposure can influence sleep (Czeisler et al., 1986; Thorleifsdottir et al., 2002), we considered controlling for average daylight hours during the month of actigraphy data collection, which were calculated by estimating the average sunrise and sunset for that month recorded officially in the area (Edwards, 2007). $\Delta\chi^2$ tests were used to determine whether the control variables were needed in each of the four fitted models. For consistency, if the inclusion of a control variable resulted in a significant change in χ^2 in at least one model, the covariate was retained in all fitted models. The inclusion of child gender, race/ethnicity, and SES (at T1) resulted in a significant change in χ^2 in at least one model and thus only these covariates were retained in the fitted models. To control for their effects, the covariates were allowed to predict each of the sleep and adjustment variables at each wave.

Analyses were conducted using Amos 17. Full information maximum likelihood (FIML) estimation was used to handle missing data. FIML estimation is robust against violations of normality and is one of the best methods for handling missing data (Acock, 2005).

Results

Preliminary Analyses

Means and standard deviations of study variables are presented in Table 1. Notably, on average, children received less than 7.5 hours of sleep per night during all three waves. Average time in bed per night (i.e., minutes between bedtime and wake time) during each study

wave was less than 8.5 hours which is under the National Sleep Foundation's (2011) recent recommendations of 10 to 11 hours for school-aged children and 8.5 to 9.25 hours for 13-year-old children. At all three waves sleep efficiency was below 90%, which is considered a characteristic of poor sleep quality (Sadeh et al., 2000).

Repeated measures ANOVA was used to test whether the means of the sleep and adjustment variables differed across the three waves (Table 1). Notably, on average sleep minutes did not change across time. However, among the sleep quality parameters, sleep efficiency and sleep % increased from T1 to T2 and minutes awake after sleep onset decreased from T1 to T2. Regarding the adjustment variables, anxiety decreased from T1 to T2 and from T2 to T3. In addition, father-reported externalizing symptoms were significantly higher at T1 compared to T3. Further, bivariate correlations among study variables are provided in Table 2. Overall, many of the sleep parameters were statistically correlated with the adjustment variables within and across waves, such that more sleep problems were related to greater adjustment problems and vice versa.

Examination of Reciprocal Relations between Children's Sleep Problems and their Adjustment Symptoms over Time

Sleep/wake problems. The cross-lagged panel model used to examine the reciprocal relations between children's sleep/wake problems and depression, anxiety, and externalizing symptoms over time was a good fit to the data, $\chi^2 = 106.24$, $df = 71$, $p < .01$; $\chi^2/df = 1.50$; CFI = .96; RMSEA = .05 ns (Figure 1). All autoregressive effects were significant. At T2 and T3, respectively, the following amount of variance was explained in each model: 21% and 27% for sleep/wake problems, 15% and 21% for depression symptoms, 14% and 35% for anxiety symptoms, and 67% and 70% for externalizing symptoms.

As shown in Figure 1, greater sleep/wake problems at T1 predicted an increase in T2 depression symptoms and T2 anxiety symptoms, accounting for 3% and 4% of unique variance, respectively. Similarly, more sleep/wake problems at T2 was related to an increase in T3 depression symptoms and T3 anxiety symptoms, accounting for 6% and 11% of unique variance, respectively. Regarding reciprocal relations, greater externalizing symptoms at T1 predicted increased sleep/wake problems at T2 and explained 3% of unique variance.

We conducted $\Delta\chi^2$ tests to examine whether the magnitude of associations between T1 sleep/wake problems and subsequent depression and anxiety symptoms were different than those observed between T2 sleep/wake problems and subsequent depression and anxiety symptoms. In addition, $\Delta\chi^2$ tests were used to determine whether sleep/wake problems was a more robust predictor of subsequent depression symptoms compared to anxiety symptoms. No differences were found, indicating that T2 sleep/wake problems was not a more robust predictor of subsequent depression or anxiety symptoms compared to relations between T1 sleep/wake problems and subsequent T2 pertinent outcomes. Finally, no evidence was found for sleep/wake problems as a more robust predictor of subsequent depression versus anxiety symptoms.

Sleep minutes. The cross-lagged panel model used to examine the reciprocal relations between sleep minutes and depression, anxiety, and externalizing symptoms over time was a good fit to the data, $\chi^2 = 112.01$, $df = 71$, $p < .001$; $\chi^2/df = 1.58$; CFI = .94; RMSEA = .06 ns (Figure 2). In addition to autoregressive effects for the adjustment variables already presented in Figure 1, the autoregressive effect for sleep minutes was significant from T1 to T2 and from T2 to T3. At T2 and T3, respectively, the following amount of variance was explained for each variable: 37% and 19% for sleep minutes, 8% and 22% for depression symptoms, 8% and 27% for anxiety symptoms, and 68% and 71% for externalizing symptoms.

As shown in Figure 2, fewer sleep minutes at T2 predicted an increase in children's depression symptoms, anxiety symptoms, and externalizing symptoms at T3 and explained 8%, 4%, and 2% of unique variance, respectively. Further, elevated levels of depression symptoms at T1 predicted a decrease in sleep minutes at T2 and explained 2% of unique variance.

$\Delta\chi^2$ tests were used to determine whether the magnitude of associations differed between T2 sleep minutes and subsequent depression, anxiety, and externalizing symptoms. No differences were found, indicating that the magnitude of associations between sleep minutes and child outcomes was similar across depression, anxiety and externalizing behaviors.

Poor sleep quality. The cross-lagged panel model used to examine the reciprocal relations between children's sleep quality and all adjustment outcomes was a good fit to the data, $\chi^2 = 535.1$, $df = 235$, $p < .001$; $\chi^2/df = 2.27$; CFI = .93; RMSEA = .09 $p < .001$. As shown in Figure 3, examination of autoregressive effects indicated that poor sleep quality was moderately stable over time from T1 to T2 and from T2 to T3. At T2 and T3, respectively, the following amount of variance was explained in each model: 31% and 17% for poor sleep quality, 9% and 24% for depression symptoms, 9% and 27% for anxiety symptoms, and 69% and 74% for externalizing symptoms.

Poorer sleep quality at T2 predicted an increase in depression symptoms, anxiety symptoms and externalizing symptoms at T3 and explained 9%, 5%, and 4% of unique variance, respectively. Regarding reciprocal relations, more depression symptoms at T1 predicted poorer sleep quality at T2 and accounted for 2% of unique variance. Greater symptoms of anxiety at T2 predicted poorer sleep quality at T3 and accounted for 4% of unique variance.

$\Delta\chi^2$ tests were used to determine whether the magnitude of associations differed between T2 poor sleep quality and subsequent depression, anxiety, and externalizing

symptoms; no significant differences were found. We conducted one additional $\Delta\chi^2$ test to examine whether the relation between T2 poor sleep quality and T3 anxiety symptoms was more robust than the association between T2 anxiety symptoms and T3 sleep quality; no significant differences were found.

Variability in sleep onset. The cross-lagged panel model used to examine the reciprocal relations between variability in sleep onset time and all adjustment outcomes was a good fit to the data, $\chi^2 = 115.33$, $df = 71$, $p < .01$; $\chi^2/df = 1.62$; CFI = .93; RMSEA = .06 ns (model not presented in a Figure for brevity). The autoregressive effect for variability in sleep onset was significant from T2 to T3 ($B = .28$, $\beta = .22$, $p < .05$) but not from T1 to T2. At T2 and T3, respectively, the following amount of variance was explained in each model: 3% and 17% for variability in sleep onset, 8% and 13% for depression symptoms, 8% and 22% for anxiety symptoms, and 69% and 67% for externalizing symptoms.

Externalizing symptoms at T2 predicted an increase in variability in sleep onset at T3 ($B = .001$, $\beta = .35$, $p < .01$) and explained 12% of unique variance. No other relations involving variability in sleep schedule were significant.

Discussion

Toward gaining a better understanding of the directionality of effects between an important biological regulatory system and children's adjustment, the reciprocal relations between sleep and emotional and behavioral problems were examined over time. In an attempt to conduct a rigorous assessment we measured sleep using self-reports and actigraphy, had multiple informants report on children's adjustment, and used a three wave longitudinal design that spanned five years. Among a community sample of otherwise normally developing children, sleep problems predicted increases in emotional and behavioral adjustment longitudinally, despite significant autoregressive effects. To a lesser extent but supportive of reciprocal

relations, children's depression, anxiety, and externalizing symptoms predicted increases in sleep problems over time, while controlling for autoregressive effects. This study serves as one of the first to examine the reciprocal relations between children's sleep and adjustment using a community sample and suggests that associations between sleep problems and emotional and behavioral problems may be cyclical.

Results build on a growing body of literature that has examined children's sleep problems as predictors of adjustment longitudinally (e.g., El-Sheikh et al., 2010; El-Sheikh et al., under review; Fredricksen et al., 2004; Goodnight et al., 2007; Gregory et al., 2005; Wong et al., 2009). Greater sleep/wake problems, fewer sleep minutes, and poorer sleep quality predicted increases in depression and anxiety symptoms over time. Fewer sleep minutes and poorer sleep quality (but not self-reported sleep/wake problems) also predicted greater externalizing symptoms longitudinally. While we have not examined potential mechanisms of effects, plausible explanations exist that may explain why sleep problems predict increases in both internalizing and externalizing symptoms. Sleep serves an important role in brain development including the PFC, which is responsible for daytime cognitive performance and emotional regulation (Dahl, 1996). Sleep problems can compromise processes mediated in the PFC including the executive function that is needed for emotion regulation (Jones & Harrison, 2000; Muzur et al., 2002), which in turn may put children at risk for adjustment problems (Eisenberg et al., 2001).

Furthermore, researchers from cognitive neuroscience have begun to make exciting advances in understanding the influence that sleep has in processing, consolidating, and buffering daily emotional experiences (Walker & van der Helm, 2009). Specifically, negative memories have been shown to be more resistant to the impact that sleep problems have on retention loss, compared to neutral or positively themed memories (Phelps, 2004). Although additional studies are needed, one may begin to hypothesize that the retention of negative

information and loss of positive information from sleep problems may have an adverse impact on internalizing and externalizing symptoms. In addition, sleep problems may influence the ability to accurately interpret emotional experiences in the environment. In a recent study, Soffer-Dudek, Sadeh, Dahl, and Rosenblat-Stein (2011) tested school-aged children on their ability to accurately identify emotional expressions on presented faces. Greater night wakings were associated with a reduced ability to accurately interpret emotional expressions. This work suggests that adequate sleep is necessary for accurately identifying emotional information in the environment, which in turn may be a precursor for adjustment problems (Crick & Dodge, 1994). Lastly, to offer an additional explanation as to why sleep problems may specifically predict externalizing symptoms, researchers have suggested that sleep problems lead to increased irritability, which in turn increases the likelihood that an individual will respond to aversive situations in an aggressive manner (Clinkinbeard, Simi, Evans, & Anderson, 2010). Overall however, continued work is needed to further test these potential mechanisms of effects in the link between sleep problems and children's emotional and behavioral development.

Consistent with research that found sleep to be a more robust predictor of maladjustment when children entered adolescence as compared to younger ages (Chorney et al., 2008; Gregory & O'Connor, 2002), we found that actigraphy measured sleep at age 10 (T2) predicted increases in adjustment problems at age 13 (T3), but no such relations were found between actigraphy measured sleep at age 8 (T1) and adjustment at age 10 (T2). Plausible explanations exist. For example, although much debate surrounds how much children and teens should sleep (Lumeng, 2010; Olds Blunden, Petkov, & Forchino, 2010), some evidence indicates that the amount of needed sleep increases as children enter adolescence (Carskadon, Harvey, Duke, Anders, & Dement, 1980; Dahl & Lewin, 2002). However, in our sample sleep amount did not change from age 8 (*M* sleep amount = 7 hr. 21 min) to age 10 (*M* sleep amount = 7 hr. 25 min) and several markers of sleep quality actually improved from ages 8 to 10 (e.g.,

sleep efficiency, minutes awake after sleep onset). The disparity between sleep needed and received may have been greater at age ten, which marks the early stages of preadolescence and thus, children may have been more prone to the negative consequences of inadequate sleep at age 10 compared to age 8. Further, the autoregressive effects were highly stable across waves for sleep quantity and sleep quality, potentially indicating consistency in sleep problems over time. Therefore, another potential explanation for stronger findings between ages 10 and 13 than between ages 8 and 10 is that the additive effect of sleep problems at age 8 and age 10 may have been a “tipping point” for later emotional and behavioral problems. Indeed, compared to transient sleep loss, longer periods of inadequate sleep may stunt brain development and cause greater neuronal damage resulting in increased adjustment difficulties (Jan et al., 2010). Utilizing longitudinal growth modeling techniques with an independent sample, El-Sheikh et al. (under review) demonstrated that children who experienced sleep problems persistently over two years were at increased risk for adjustment problems, compared to children who had more temporary sleep problems. Overall, we view our explanations as tentative. Continued investigations are needed to further determine whether the relations between sleep and adjustment become more evident as children develop. More work is also needed to examine how persistent versus transient sleep problems differentially influence children’s development.

For a better understanding of the unique impact that sleep has on children’s depression, anxiety, and externalizing symptoms, we examined each adjustment variable simultaneously in the cross-lagged models. Although internalizing and externalizing symptoms often co-occur (Keiley et al., 2003), the simultaneous inclusion of each adjustment variable removes shared variance and allows us to determine whether sleep is a more robust predictor of internalizing or externalizing symptoms. The magnitude of statistically significant associations did not differ between sleep and each adjustment variable (i.e., the beta weights), however we found more

instances in which sleep problems predicted depression (4 out of 8 tested paths; 50%) and anxiety symptoms (4 out of 8 tested paths; 50%) in comparison to externalizing symptoms (2 out of 8 tested paths; 25%). Thus, sleep problems may be more likely to predict internalizing symptoms compared to externalizing symptoms. Although replication of findings are certainly needed across future studies, one may begin to question whether different mechanisms bridge the associations between sleep problems and internalizing symptoms versus externalizing symptoms.

To a lesser extent but supportive of reciprocal relations, depression (2 out of 8 tested pathways; 25%), anxiety (1 out of 8 tested pathways; 13%), and externalizing symptoms (2 out of 8 tested pathways; 25%) predicted increases in sleep problems over time. Specifically, depression symptoms predicted fewer sleep minutes and poorer sleep quality, more anxiety predicted poorer sleep quality, and externalizing symptoms were related to increases in self-reported sleep/wake problems and more variability in sleep onset time. While there are some accounts that children's adjustment difficulties impact sleep quantity and quality, most of these studies have used clinical samples of children with internalizing (e.g., Alfano et al., 2010; Forbes et al., 2008) and externalizing problems (Sneddon, 2007). The current study builds on a very small body of research that has utilized community samples and longitudinal designs (Patten et al., 2000), and demonstrates that depression and anxiety symptoms predict greater sleep problems over time. In addition, this study is one of the first to our knowledge to establish longitudinal relations between externalizing symptoms and increases in children's sleep problems using a community sample. Despite finding more support for the opposite direction (i.e., sleep as a predictor of later adjustment) continued investigations are important to gain a more in-depth understanding of why adjustment difficulties predict later sleep problems.

Using cognitive-behavioral, biological, and contextual frameworks, there are many possibilities that may explain why emotional and behavioral difficulties predicted increases in

sleep problems over time. Symptoms of depression and anxiety include ruminating thought processes, emotional distress, and feelings of fear, all of which may lead to fragmented and restless sleep (Dahl, 1996; Dahl & Lewin, 2002; Patten et al., 2000). From a biological perspective, children who have higher levels of internalizing symptoms also have increased levels of cortisol (Forbes et al., 2006), which is a risk factor for sleep problems (El-Sheikh et al., 2008; Raikonen et al., 2010). Specifically, cortisol promotes wakefulness and thus, increased levels can interfere with falling asleep and may contribute to night wakings and greater difficulty falling back asleep. Similarly, children who exhibit externalizing symptoms often have increased SNS activity. Although findings have been mixed (El-Sheikh & Arsiwalla, 2011; Holmes et al., 2002), increased SNS activity may interfere with the reduction of vigilance needed for falling and staying asleep (Dahl, 1996; Dahl & Lewin, 2002).

Further, higher levels of externalizing symptoms predicted greater variability in sleep onset time across the week of actigraphy. From a contextual perspective, children who are high on externalizing symptoms may be less compliant to household rules such as scheduled bed times, and thus sleep onset time may fluctuate across nights (Bartels et al., 2003; Goodnight et al., 2007). Obviously, these potential explanations about processes and mechanisms of effects accounting for the association between children's adjustment difficulties and sleep problems are tentative until explicated empirically. Nonetheless, direct associations between children's emotional and behavioral adjustment difficulties and later sleep problems are established, marking an important step in the sleep and adjustment literature.

Findings from the current study have important implications for parents, teachers, and clinicians. The use of a community sample indicates that even slight sleep problems can influence children's adjustment. For instance, one hour of less sleep per night or occasional night awakenings should not be overlooked, given their shared associations with emotional and behavioral difficulties. Further, the co-occurrence and cyclical nature of children's sleep

problems and adjustment problems may potentially influence facets of daytime functioning that could have long term ramifications, including poor school performance and troubled peer relationships.

Our results indicated that the mean scores for child reported anxiety and parent-reports of child externalizing symptoms decreased across study waves. Plausible explanations exist. The RCMAS, which was used to assess children's anxiety symptoms, consists of many items that assess fear (i.e., I am afraid of a lot of things). Research studies have repeatedly found that the prevalence and intensity of fear decreases as children become older (see Gullone, 2000 for a review). Thus, symptoms of anxiety may have decreased in the current study due to the expected decrease in fear and sense of threat. For externalizing symptoms, parents often become less aware of children's daytime activities including noncompliant and delinquent types of behaviors (Bongers, Koot, van der Ende, & Verhulst, 2004).

Regarding study limitations and future directions, the combination of actigraphy and self-reports of children's sleep combine to measure multiple facets of sleep problems, however they do not assess sleep stages. Polysomnography, the gold standard measure of sleep has the capability to assess sleep staging, and thus may be useful to determine whether disruptions in sleep architecture are related to emotional and behavioral difficulties among community samples. A study feature was an examination of associations between sleep and adjustment among children ages 8 to 13. Assessments over other developmental periods may yield a different pattern of effects, and it is imperative for future research to examine developmental trajectories of child adaptation and sleep at various ages. Moreover, while this study illustrated the reciprocal relations between sleep and adjustment across three waves spanning five years, few studies have assessed the reciprocal relations between sleep and adjustment day-to-day (Cousins et al., 2011). For instance, sleep problems during one night may lead to emotional and behavioral problems the following day, which in turn may result in increased sleep problems the

next night. Thus, the cyclical nature between sleep and adjustment problems may operate within smaller timeframes. An examination of the reciprocal relations between sleep and adjustment from day-to-day could be complementary to studies across longer time frames. Also, the reciprocal relations between sleep and adjustment may differ among clinical versus community samples. Finally, reliance of one measure to assess each adjustment variable may be a potential limitation.

In sum, the primary contribution is the demonstration of reciprocal relations between sleep problems and emotional and behavioral adjustment in a community sample of children. Results add to a growing body of literature that has found sleep to play an important role in children's development and builds substantially on the much less studied topic of emotional and behavioral problems as antecedents of sleep problems. Overall, findings demonstrate the cyclical relationship between sleep and adjustment problems.

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Table 1

Comparison of Means among Observed Study Variables over Time Using Repeated Measures ANOVA

| Variable Name | T1 Mean (SD) Age 8 | T2 Mean (SD) Age 10 | T3 Mean (SD) Age 13 | F-value |
|---|-----------------------------|---------------------------|------------------------|----------|
| 1. Sleep/Wake Problems ^a | 17.12 (4.92) ² | 15.93 (4.67) ³ | 20.13 (6.70) | 32.32*** |
| 2. Sleep Minutes ^b | 441 min (59 min) | 445 min (48 min) | 434 min (67 min) | .39 |
| 3. Sleep Efficiency ^b | 86.60 (8.20) ¹ | 89.15 (9.31) | 87.85 (8.20) | 4.44* |
| 4. Sleep % ^b | 86.15 (8.92) ¹ | 89.38 (6.79) | 87.31 (8.22) | 4.86* |
| 5. Long Wake Episodes ^b | 3.99 (2.28) | 3.15 (2.25) | 3.51 (2.31) | 2.70 |
| 6. Minutes Awake After Sleep Onset ^b | 67.65 (37.20) ¹ | 53.45 (34.17) | 63.01 (38.71) | 4.35* |
| 7. Variability in Sleep Onset ^b | .04 (.02) | .08 (.15) | .04 (.02) | .17 |
| 8. Depression Symptoms | 5.77 (5.19) | 4.90 (4.57) | 4.64 (4.66) | .71 |
| 9. Anxiety Symptoms | 11.50 (6.96) ^{1,2} | 8.21 (6.25) ³ | 6.40 (5.11) | 14.50*** |
| 10. Externalizing Symptoms (Mother Report) | 50.71 (10.84) | 48.02 (9.30) | 48.14 (8.53) | 6.51 |
| 11. Externalizing Symptoms (Father Report) | 50.14 (10.71) ² | 48.26 (8.09) | 47.03 (8.73) | 8.59*** |

Note. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3.

^a Child-reported sleep measure. ^b Actigraphy-derived.

¹. Statistically significant difference between means at T1 and T2. ². Significant difference between T1 and T3. ³. Significant difference between T2 and T3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2

Correlations among Study Variables

| | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. | 11. | 12. | 13. | 14. |
|--|-------|-------------------|------------------|------|-------|------------------|------|------|-------------------|-------------------|------------------|------------------|------------------|------------------|
| 1. Sleep/Wake Problems ^a T1 | - | | | | | | | | | | | | | |
| 2. Sleep Minutes ^b T1 | -.12 | - | | | | | | | | | | | | |
| 3. Poor Sleep Quality ^b T1 | .08 | -.81* | - | | | | | | | | | | | |
| 4. Variability in Sleep Onset ^b T1 | .02 | -.12 | .03 | - | | | | | | | | | | |
| 5. Depression Symptoms T1 | .39* | -.24* | .21* | .00 | - | | | | | | | | | |
| 6. Anxiety Symptoms T1 | .39* | -.03 | -.03 | .06 | .50* | - | | | | | | | | |
| 7. Externalizing Symptoms T1 | .07 | -.16* | .14 | .06 | .18* | .14 [†] | - | | | | | | | |
| 8. Sleep/Wake Problems ^a T2 | .40* | -.09 | .03 | .09 | .23* | .25* | .25* | - | | | | | | |
| 9. Sleep Minutes ^b T2 | -.17* | .57* | -.46* | -.13 | -.27* | -.02 | -.07 | -.12 | - | | | | | |
| 10. Poor Sleep Quality ^b T2 | .08 | -.46* | .55* | .08 | .26* | .07 | .02 | .08 | -.65* | - | | | | |
| 11. Variability in Sleep Onset ^b T2 | -.07 | -.07 | .06 | .06 | .01 | .13 | .06 | -.01 | -.11 | -.04 | - | | | |
| 12. Depression Symptoms T2 | .17* | -.14 | .07 | .03 | .34* | .30* | .14 | .34* | -.16 [†] | .06 | .15 [†] | - | | |
| 13. Anxiety Symptoms T2 | .27* | -.06 | .06 | -.02 | .33* | .36* | .17* | .58* | -.02 | .04 | .08 | .49* | - | |
| 14. Externalizing Symptoms T2 | .03 | -.14 [†] | .15* | .06 | .18* | .11 | .92* | .26* | -.09 | .10 | .03 | .13 | .18* | - |
| 15. Sleep/Wake Problems ^a T3 | .21* | .01 | .02 | -.05 | .14 | .27* | .21* | .51* | -.07 | .09 | .06 | .19 [†] | .30* | .27* |
| 16. Sleep Minutes ^b T3 | .04 | .26* | -.20* | -.07 | -.13 | -.10 | -.07 | -.07 | .32* | -.20 [†] | -.05 | -.06 | -.14 | -.02 |
| 17. Poor Sleep Quality ^b T3 | .03 | -.07 | .16 | .03 | -.01 | .01 | .09 | .11 | -.11 | .23* | -.17 | .05 | .19 [†] | .08 |
| 18. Variability in Sleep Onset ^b T3 | -.01 | -.20 [†] | .19 [†] | .15 | .15 | .24* | .31* | .10 | -.16 | .17 | .23* | .14 | .09 | .24* |
| 19. Depression Symptoms T3 | .08 | -.18 [†] | .14 | -.08 | .33* | .20* | .16 | .27* | -.29* | .32* | -.03 | .41* | .37* | .19 [†] |
| 20. Anxiety Symptoms T3 | .26* | -.02 | .12 | .07 | .21* | .30* | .15 | .48* | -.19 [†] | .25* | -.06 | .34* | .50* | .21* |
| 21. Externalizing Symptoms T3 | .03 | -.19* | .21* | .00 | .24* | .06 | .82* | .24* | -.15 [†] | .20* | .04 | .13 | .19* | .91* |

[†] p < .10; *p < .05.

Table 2 (Cont.)

| | 15. | 16. | 17. | 18. | 19. | 20. | 21. |
|--|------|-------------------|------------------|------------------|------|------|-----|
| 15. Sleep/Wake Problems ^a T3 | - | | | | | | |
| 16. Sleep Minutes ^b T3 | -.03 | - | | | | | |
| 17. Poor Sleep Quality ^b T3 | -.03 | -.62* | - | | | | |
| 18. Variability in Sleep Onset ^b T3 | .12 | -.08 | .03 | - | | | |
| 19. Depression Symptoms T3 | .46* | -.20 [†] | .21* | .29* | - | | |
| 20. Anxiety Symptoms T3 | .54* | -.14 | .20 [†] | .25* | .64* | - | |
| 21. Externalizing Symptoms T3 | .30* | .01 | .01 | .20 [†] | .33* | .24* | - |

Figure 1.

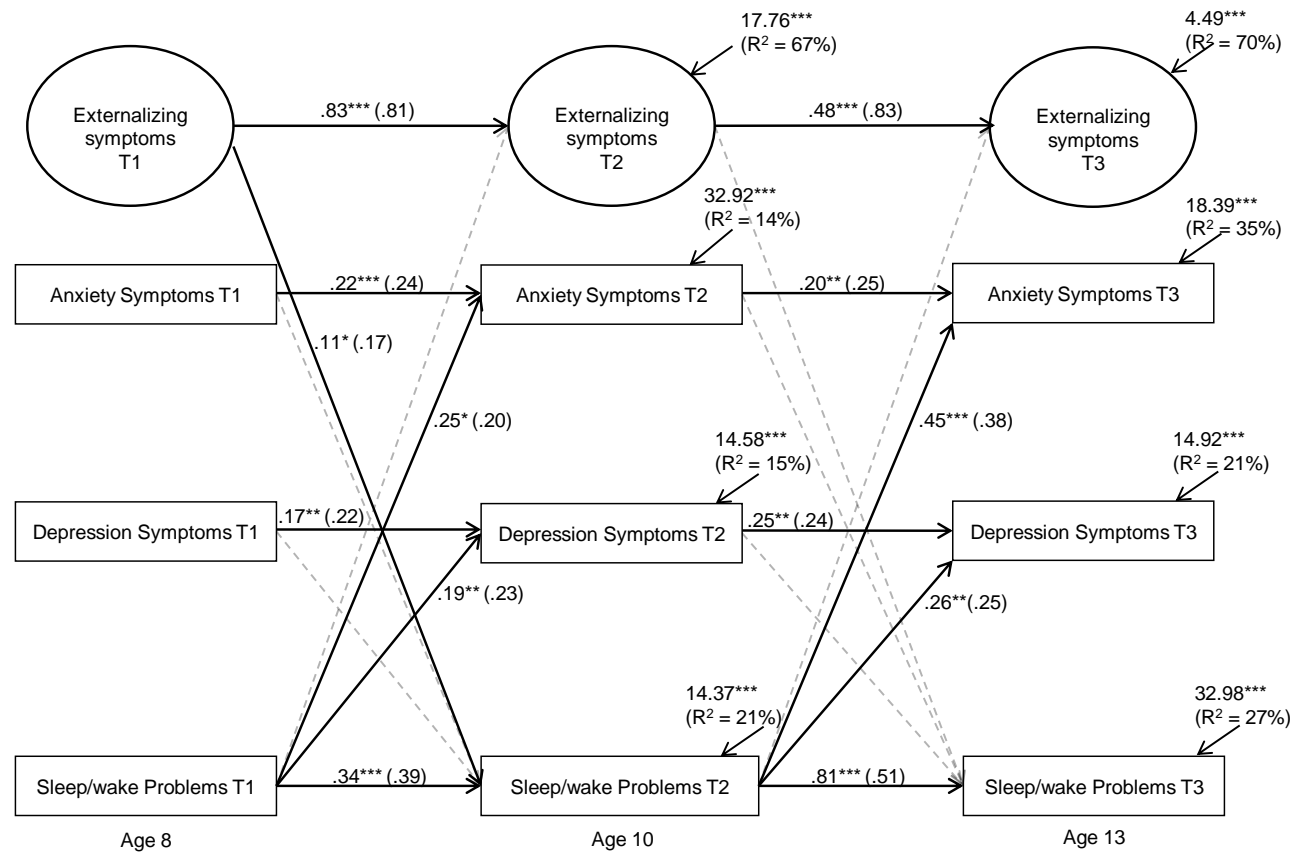


Figure 1. Examination of reciprocal relations between children's reports of sleep/wake problems and emotional and behavioral adjustment. Unstandardized and standardized coefficients (in parentheses) are provided. Child gender, race/ethnicity, and T1 SES are controlled in analyses. Residual variances among endogenous variables within each wave were allowed to correlate. Model fit: $\chi^2 = 106.24$, $df = 71$, $p < .01$; $\chi^2/df = 1.50$; CFI = .96; RMSEA = .05 ns . Statistically significant lines are solid whereas non-significant lines are dotted. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2.

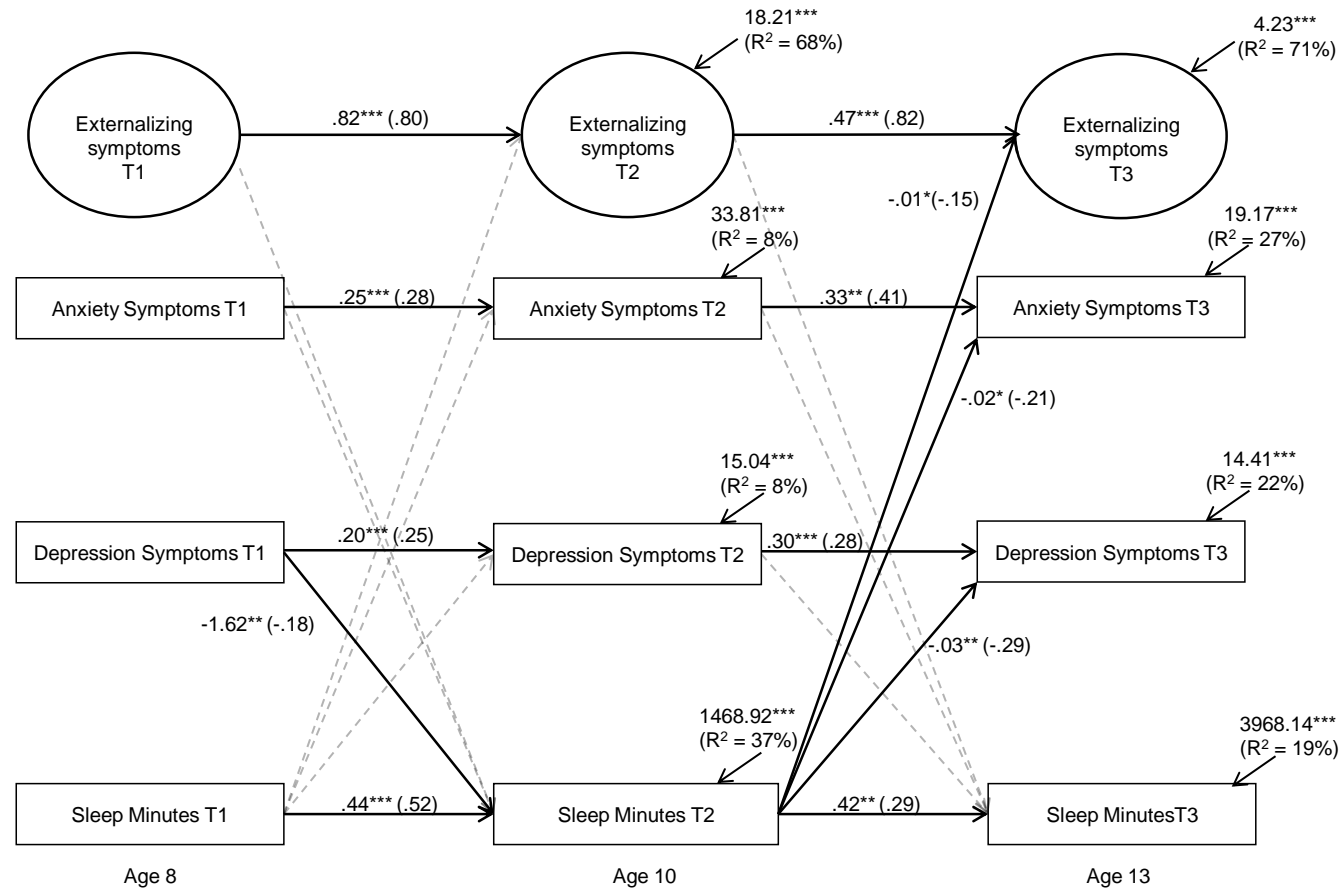


Figure 2. Examination of reciprocal relations between children's sleep minutes and emotional and behavioral adjustment. Unstandardized and standardized coefficients (in parentheses) are provided. Child gender, race/ethnicity, and T1 SES are controlled in analyses. Residual variances among endogenous variables within each wave were allowed to correlate. Model fit: $\chi^2 = 112.01$, $df = 71$, $p < .001$; $\chi^2/df = 1.58$; CFI = .94; RMSEA = .06 ns . Statistically significant lines are solid whereas non-significant lines are dotted. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 3.

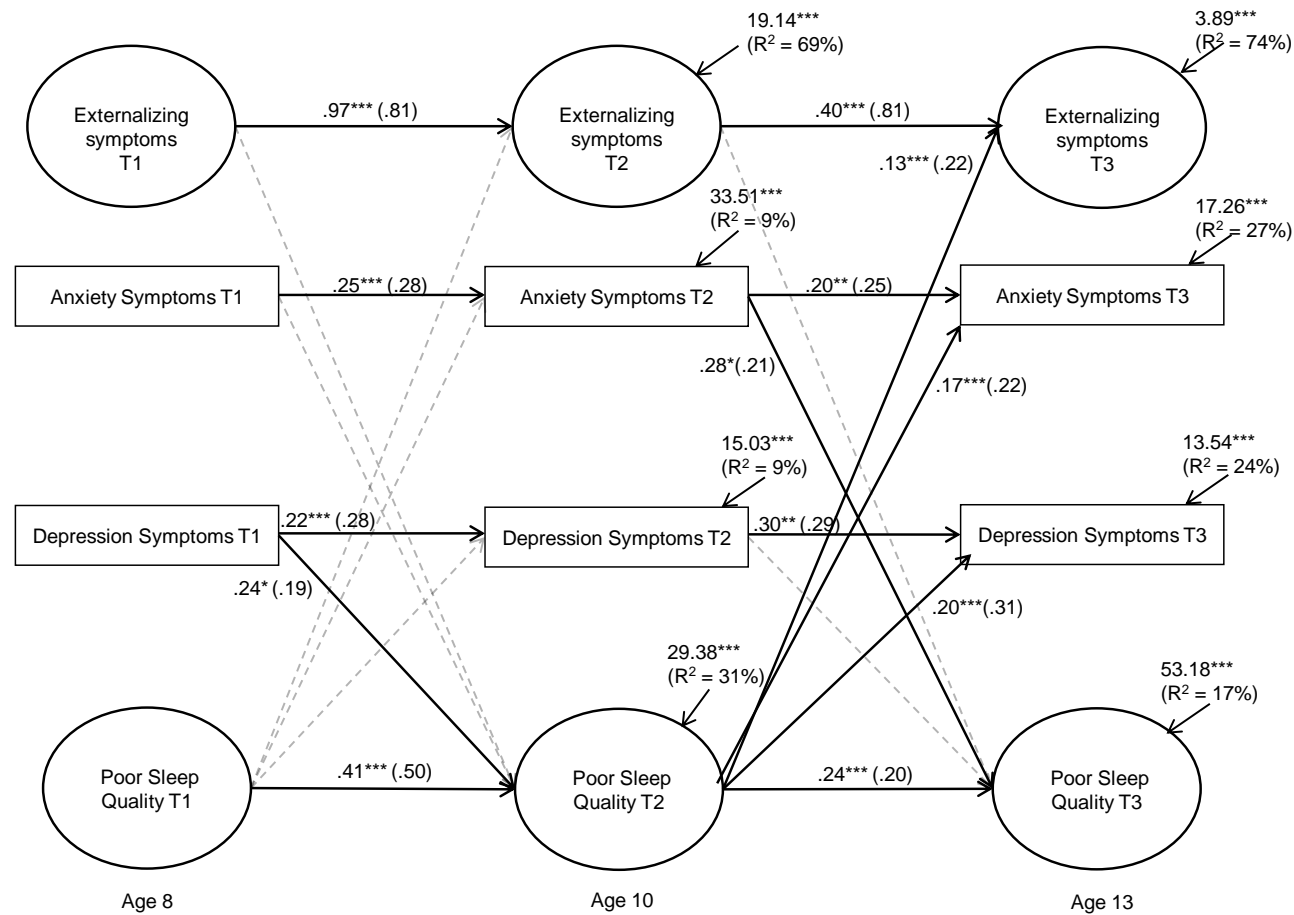


Figure 3. Examination of reciprocal relations between children's poor sleep quality and emotional and behavioral adjustment. Unstandardized and standardized coefficients (in parentheses) are provided. Child gender, race/ethnicity, and T1 SES are controlled in analyses. Residual variances among endogenous variables within each wave were allowed to correlate. Model fit: $\chi^2 = 535.1$, $df = 235$, $p < .001$; $\chi^2/df = 2.27$; CFI = .93; RMSEA = .09 $p < .001$. Statistically significant lines are solid whereas non-significant lines are dotted. T1 = data collected at Time 1; T2 = data collected at Time 2; T3 = data collected at Time 3. * $p < .05$. ** $p < .01$. *** $p < .001$.