

## Longitudinal Associations Between Marital Instability and Child Sleep Problems Across Infancy and Toddlerhood in Adoptive Families

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This study examined the longitudinal association between marital instability and child sleep problems at ages 9 and 18 months in 357 families with a genetically unrelated infant adopted at birth. This design eliminates shared genes as an explanation for similarities between parent and child. Structural equation modeling indicated that T1 marital instability predicted T2 child sleep problems, but T1 child sleep problems did not predict T2 marital instability. This result was replicated when models were estimated separately for mothers and fathers. Thus, even after controlling for stability in sleep problems and marital instability and eliminating shared genetic influences on associations using a longitudinal adoption design, marital instability prospectively predicts early childhood sleep patterns.

An extensive body of research indicates that marital problems (e.g., conflict, discord, instability, and dissatisfaction) can have pervasive influences on fam-

ily functioning and negative effects on child social, emotional, and behavioral health (Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006; Davies, Harold, Goeke-Morey, & Cummings, 2002; El-Sheikh, 2005; Emery, 1982; Grych & Fincham, 2001; Grych, Harold, & Miles, 2003; Harold, Aitken, & Shelton, 2007; Rhoades, 2008). Similarly, in accordance with the view that the family context comprises interacting relational systems (Cummings & Davies, 2002), child behavioral dysregulation has been shown to predict increased interparental discord (Schermerhorn, Cummings, DeCarlo, & Davies, 2007), and reciprocal relations have been identified between marital

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This project was supported by grant R01 HD042608, NICHD, NIDA, and the Office of the Director, NIH, U.S. PHS (PI Years 1–5: David Reiss, MD; PI Years 6–10: Leslie Leve, PhD). The writing of this manuscript was partially supported by P30 DA023920, NIDA, NIH, U.S. PHS. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health & Human Development or the National Institutes of Health. We thank the adoptive families who participated in this study, the adoption agency staff members who helped with the recruitment of study participants, Matthew Rabel and Michelle Baumann for editorial assistance, Samuel Simmens and Sally Guyer for data management assistance, and Xiaojia Ge and John Reid for their scientific contributions to the Early Growth and Development Study.

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DOI: 10.1111/j.1467-8624.2011.01594.x

conflict and child behavior problems (Jenkins, Simpson, Dunn, Rasbash, & O'Connor, 2005).

Investigators have recently focused attention on neurobiological systems underlying the association between aspects of the marital relationship and child adjustment (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; Davies, Woitach, Winter, & Cummings, 2008; El-Sheikh, 2005; Sadeh, Keinan, & Daon, 2004). Consistent with transactional models that emphasize the importance of the family context in the development and regulation of sleep (Sadeh & Anders, 1993), disturbances in child sleep patterns are believed to be a marker of the impact of family stress on neurobiological functioning (El-Sheikh, Buckhalt, Cummings, & Keller, 2007; Sadeh, Gruber, & Raviv, 2002). Studies of nonclinical populations indicate that sleep problems (i.e., difficulty initiating or maintaining sleep) that emerge during early childhood tend to persist later in development (Richman, Stevenson, & Graham, 1982; Zuckerman, Stevenson, & Bailey, 1987). For example, Zuckerman and colleagues found that 41% of infants in a community-based sample who had sleep problems at age 8 months still had sleep problems at age 3 years (Zuckerman et al., 1987). Thus, early identification of the precursors, correlates, and outcomes of child sleep problems is particularly important given this continuity and the numerous longitudinal studies indicating that childhood sleep problems are associated with a range of adjustment, executive attention, and academic difficulties later in development (El-Sheikh et al., 2007; Gregory & O'Connor, 2002; Meijer, Habekoth, & van den Wittenboer, 2000). The mechanisms whereby these linkages occur are unclear, though there is some evidence that sleep problems may reflect underlying sensitization of stress response systems (Dahl, 1996; Sadeh, 1996; Sadeh et al., 2004). Moreover, it is unclear how early in development associations between the marital relationship and child sleep problems emerge.

*Associations Between Marital Instability and Child Sleep Problems: An Artifact of Passive Gene-Environment Correlation?*

Previous research examining associations between child sleep and the marital relationship has uniformly been conducted with biological families; indeed, this is the case for the vast majority of studies linking marital and parenting factors to child functioning (Davies et al., 2002; Rhoades, 2008). However, in research involving biologically related family members, it is difficult to ascertain

whether associations between parent behaviors and child outcomes represent environmental effects or shared genetic influences (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). That is, associations between a characteristic of the parent and a characteristic of the child may result from an underlying shared genetic characteristic that simultaneously influences both the trait in the parent and the trait in the child (e.g., passive gene-environment correlation), even if the parent and child traits have quite different behavioral manifestations. For example, adolescents of divorced parents are at increased risk for delinquent behavior (D'Onofrio et al., 2005). Whereas this association could be mediated by negative parenting or the presence of a chaotic home environment, associations between parental divorce and child delinquency could also be explained by a shared genetic predisposition for negative emotionality (Krueger, 1999; McGue & Lykken, 1992). In support of the latter explanation, genetically informed studies have suggested significant passive gene-environment correlations on associations between parental divorce and child internalizing symptoms (D'Onofrio et al., 2007) and between parental divorce, academic achievement, and social adjustment (O'Connor, Caspi, DeFries, & Plomin, 2000). In addition, independent evidence has indicated that individual differences in marital quality (Spotts et al., 2004), propensity to divorce (D'Onofrio et al., 2005; McGue & Lykken, 1992; Waldman, 2007), and sleep patterns (Ambrosius et al., 2008; Watson, Goldberg, Arguelles, & Buchwald, 2006) are heritable. Taken together, these findings underscore the need for genetically informed research designs to specify the mechanisms underlying associations between parent and child characteristics.

Research designs that include only biological families cannot disentangle whether associations between aspects of the couple relationship and child adjustment are genetic or environmental in origin precisely because shared genes influence the behavior of both parents and children, and by extension, shape the family environment. However, in a recent study, Burt and colleagues were able to separate the potential influence of passive gene-environment correlation from environmental effects by examining the timing of parental divorce and risk for adolescent delinquency in a sample of adoptive and biological families (Burt, Barnes, McGue, & Iacono, 2008). Using this approach, Burt et al. (2008) concluded that direct exposure to parental divorce accounted for the association between divorce and delinquency for both adopted

and nonadopted adolescents (rather than associations being mediated through shared genes). Thus, designs that permit the separation of passive gene-environment correlation from environmental effects may have important implications for understanding the mechanisms underlying associations between parent and child characteristics.

To date, no study has examined associations between the marital relationship and child sleep problems where the research design employed can unambiguously eliminate the confounding role of passive gene-environment influences on the observed pattern of association. By using a sample of adopted children and their parents, the influence of shared genes on parent and child characteristics is eliminated in examining the association between marital instability and child sleep problems. For example, where significant correlations are found among unrelated family members (in which passive gene-environment correlation is absent), the primacy of environmental mechanisms underlying any such association is apparent.

#### *The Importance of Sleep During Early Childhood*

The 1st year of life is a transitional period in the emergence and consolidation of nocturnal sleep patterns. Parental intervention during this period is often necessary to help regulate young infants' sleep patterns, particularly in helping children return to sleep (Anders & Keener, 1985; Burnham, Goodlin-Jones, Gaylor, & Anders, 2002). During the 2nd year, nighttime awakenings decrease, but difficulties initiating sleep tend to remain stable or increase (Gaylor, Burnham, Goodlin-Jones, & Anders, 2005; Lam, Hiscock, & Wake, 2003). Thus, infancy and toddlerhood are arguably important times during which to focus on the directionality of effects between child sleep problems and aspects of the marital relationship.

Although sleep is an aspect of child functioning that can be affected by family stress (Bell & Belsky, 2008), very little is known about the specific association between child sleep patterns and marital instability. To address this gap, the present study employed a prospective, longitudinal design in which marital instability and child sleep problems were measured when children were 9 and 18 months of age. The present study extends prior work in three ways. First, associations between marital instability and child sleep problems were examined during infancy, a developmental period that is central in the establishment of sleep routines. Second, a longitudinal design was employed to

examine the reciprocal and cross-lagged relations between marital instability and child sleep problems across infancy and toddlerhood in order to assess the directionality of effects between these two variables. Third, an adoption sample was used in order to eliminate potential effects of shared genes between children and parents on associations between marital instability and child sleep problems. Although caution is always warranted when interpreting nonexperimental data, the use of a longitudinal design in conjunction with a genetically informed approach improves the capacity to offer causal inferences.

#### *Associations Between Aspects of Marital Relationship Quality and Child Sleep Problems*

To date, the evidence linking marital relationship quality and child sleep problems has been sparse and inconclusive with respect to the direction of effects. Furthermore, researchers have not directly examined reciprocal associations between aspects of the marital relationship and sleep problems during early childhood. In the majority of prior work, researchers have examined changes in the marital relationship during the transition to parenthood rather than child sleep patterns per se (Belsky & Rovine, 1990; Wilkie & Ames, 1986) or have examined the relation between child sleep problems and marital satisfaction for parents of children with chronic illnesses or diagnosed sleep disorders (Cottrell & Khan, 2005; Lam et al., 2003).

The dearth of research examining the influence of child sleep problems on aspects of the marital relationship is somewhat surprising given evidence that child sleep problems are associated with parent reports of increased fatigue, depression, and parenting stress (Boergers, Hart, Owens, Streisand, & Spirito, 2007; Meltzer & Mindell, 2007; Wake et al., 2006). However, recent longitudinal studies demonstrating child effects in domains related to the marital relationship, including parent depression (Gross, Shaw, Moilanen, Dishion, & Wilson, 2008), parent negativity (Larsson, Viding, Rijdsdijk, & Plomin, 2008), and parenting practices (e.g., monitoring, reinforcement, and harsh punishment; Burke, Pardini, & Loeber, 2008; Hipwell et al., 2008) suggest the possibility that child sleep problems may also have a direct influence on aspects of the marital relationship. This hypothesis is tested in the present study.

In contrast to the lack of direct evaluations of child sleep effects on aspects of the marital relationship, there is a modest body of evidence linking

various indices of family stress with child sleep problems (Bell & Belsky, 2008; Moore, 1989; Sadeh et al., 2004). Specifically, increases in marital conflict have been found to predict disruptions in child sleep quantity and quality, perhaps by way of sensitizing physiological stress systems (El-Sheikh, Buckhalt, Mize, & Acebo, 2006). In a separate study, El-Sheikh et al. (2007) found that sleep disruptions mediated the negative relation between marital insecurity and children's academic achievement. The work of El-Sheikh and colleagues, although methodologically rigorous in the use of actigraphs to measure child sleep patterns, is cross-sectional in nature. This precludes interpretations about the direction of effects. In addition, this research was conducted during middle childhood rather than during infancy, when levels of child sleep problems and parenting stress are likely to be relatively high (Belsky & Rovine, 1990; Cowan et al., 1985; Sadeh & Anders, 1993). Thus, although several studies suggest a relation between marital distress and child sleep difficulty, few of these studies have been conducted during infancy, a developmental period during which nighttime sleep becomes more regulated. Studying relation between marital instability and child sleep during infancy therefore affords an opportunity to build upon past research examining parent effects on child sleep quality and explore potential child effects on marital instability. Given the lack of prospective longitudinal research in this area, we examined two competing hypotheses regarding marital instability and child sleep problems using cross-lagged panel correlation (CLPC) analyses. Controlling for initial levels of each variable, CLPC allows examination of the bidirectional and reciprocal interplay between marital instability and child sleep problems across time. Specifically, we examined whether variation in marital instability predicted variation in child sleep problems or vice versa.

In the context of examining reciprocal effects between marital instability and child sleep problems, it is important to consider other child and parent characteristics that might influence associations. First, previous studies have found associations between child sex and marital instability. For example, the presence of at least one male child was negatively related to marital instability and propensity to divorce in two studies (Katzev, Warner, & Acock, 1994; Morgan, Lye, & Condran, 1988). Second, several studies have observed associations between infant difficult temperament, parent reports of infant sleep problems, and measures of sleep-wake state organization (Carey, 1974; Keener,

Zeanah, & Anders, 1988; Richman, 1981; Sadeh, Lavie, & Scher, 1994; Schaefer, 1990). Some evidence also suggests that perceptions of infant sleep problems and infant temperament may be more closely linked for fathers than for mothers (Keener et al., 1988). Third, parent psychopathology, including elevated anxiety, has been associated with child bedtime resistance and daytime sleepiness in prior research (Moore, David, Murray, Child, & Arkwright, 2005; Seifer, Sameroff, Dickstein, Hayden, & Schiller, 1996). Given the transactional nature of family processes, it is plausible that parent anxiety could be related to both child sleep problems and marital instability. Thus, we examined the potential influence of child sex, infant difficult temperament, and parent anxiety in the present set of analyses. In addition, in light of evidence that the transition to parenthood is associated with changes in the marital relationship (Belsky & Rovine, 1990; Cowan et al., 1985; Kurdek, 1993), we examined the potential influence of child birth order (i.e., first- or laterborn status) on the association between marital instability and child sleep problems.

## Method

### *Participants*

The data for the present study were derived from interviews with 357 adoptive mothers (AM) and fathers (AF) participating in the Early Growth and Development Study (EGDS), an ongoing multisite study of adoptive families and birth parents (Leve et al., 2007). Adoptive families were recruited between 2003 and 2006, beginning with the recruitment of adoption agencies located in the Northwest, Mid-Atlantic, and Southwest regions of the United States. Each adoption agency appointed a liaison to identify families who had completed an adoption plan through their agency and met the following eligibility criteria: (a) the adoption placement was domestic, (b) the infant was placed within 3 months postpartum, (c) the infant was placed with a nonrelative adoptive family, (d) the infant had no known major medical conditions such as extreme prematurity or extensive medical surgeries, and (e) the parents were able to read or understand English at the eighth-grade level. Study participants were representative of the adoptive populations that completed adoption plans at the participating agencies during the same time period (Leve et al., 2007).

The children were 9 months old ( $M = 8.9$  months,  $SD = 1.1$  months) during the Time 1 (T1) assessment and 18 months old ( $M = 18.1$  months,

$SD = 1.3$  months) during the Time 2 (T2) assessment. Forty-two percent of the children were female ( $n = 151$ ); 63% of the children were the adoptive parents' first child. The mean infant age at adoption placement was 7 days ( $SD = 13$  days,  $Mdn = 2$  days, mode = 2 days). The adoptive families were typically college-educated, middle-class families. The mean ages of AM and AF at T1 were 38 ( $SD = 5.4$  years) and 39 ( $SD = 5.9$  years) years, respectively, and the couples had been married an average of 11.9 years ( $SD = 5.1$  years). The infants and adoptive parents were predominantly White (64% infants, 91% mothers, 88% fathers). There were also a significant number of multiethnic participants (21% infants, 4% mothers, 3% fathers) and African American participants (11% infants, 4% mothers, 6% fathers). Ninety-five percent of the families ( $n = 338$ ) had data at both time points and were included in the present analyses following listwise deletion protocols. The remaining 19 families only had data at one time point and were excluded from analyses. There were no significant demographic differences between families who had data at T1 only and families who had data at T1 and T2. Mean ratings of marital instability and child sleep problems did not differ between families who had data at T1 only and families who had data at T1 and T2. Additional AM and AF demographic information is presented in Leve, Neiderhiser, Scaramella, and Reiss (2008).

In-home assessments with the adoptive families were conducted at T1 and T2. The assessments were 2½ hr long, and the participants were paid for their time. During both assessments, the adoptive parents independently completed computer-assisted interview questions and questionnaires that had been mailed to them prior to the assessments. Additional details on the EGDS study recruitment procedures, sample, and assessment methods can be found in Leve et al. (2007).

### Measures

#### Marital Instability

At T1 and T2, the five-item Marital Instability Index (Booth, Johnson, & Edwards, 1983) was used to assess marital instability. For each item, AMs and AFs independently reported on the frequency of their own behavior using a 4-point scale: 1 (*not in the last year*) to 4 (*within the last 3 months*). Sample items include: "Have you or your partner seriously suggested the idea of divorce?" and "Has the thought of separating or getting a divorce crossed

your mind?" Items were aggregated and a sum score computed for each parent; higher scores indicated greater marital instability (T1 AM  $\alpha = .88$ , T1 AF  $\alpha = .79$ ; T2 AM  $\alpha = .77$ , T2 AF  $\alpha = .77$ ).

Marital instability scores were fairly low (means ranged from 5.53 to 5.79), although there was adequate variation (standard deviations ranged from 1.63 to 2.22, sum scores ranged from 5 to 20 at T1 and from 5 to 17 at T2). AM- and AF-reported marital instability were correlated at T1 ( $r = .54$ ) and T2 ( $r = .47$ ), and the T1 and T2 ratings were correlated ( $r = .57$  for AMs and  $r = .44$  for AFs). Because the marital instability scores for the AMs and AFs were positively skewed at both time points, scores were log-10 transformed prior to analysis (skews ranged from 4.29 to 4.92 at T1 and from 3.50 to 3.84 at T2).

#### Child Sleep Problems

At T1 and T2, AMs and AFs independently completed the Sleep Habits Questionnaire (SHQ; Seifer, Dickstein, Spirito, & Owens-Stively, 1996). Versions of the SHQ have been used to assess normative and clinical sleep problems in young infants, toddlers, and older children (Goodlin-Jones, Sitnick, Tang, Liu, & Anders, 2008; Kelmanson, 2004; Owens, Spirito, & McGuinn, 2000; Seifer, Dickstein et al., 1996; Warren et al., 2003). We used the six-item Bedtime Resistance subscale from this questionnaire, which assesses the child's difficulty initiating or maintaining sleep (e.g., "Child needs parent in room to fall asleep," "Child struggles at bedtime"). Parents were asked to think about their child's sleep during the past week and rate the frequency of each behavior on the following 3-point scale: *rarely* (0–1), *sometimes* (2–4), or *usually* (5 or more). The items were aggregated and a mean score computed for each parent; higher scores indicated greater bedtime resistance (T1 AM  $\alpha = .74$ , T1 AF  $\alpha = .71$ ; T2 AM  $\alpha = .81$ , T2 AF  $\alpha = .78$ ). AM- and AF-reported Bedtime Resistance scores were correlated at T1 ( $r = .80$ ) and T2 ( $r = .86$ ), and the T1 and T2 ratings were also correlated ( $r = .68$  for AMs and  $r = .61$  for AFs). Scores were normally distributed at both time points (T1 AM:  $M = 1.32$ ,  $SD = 0.40$ ; T1 AF:  $M = 1.33$ ,  $SD = 0.39$ ; T2 AM:  $M = 1.25$ ,  $SD = 0.42$ ; T2 AF:  $M = 1.28$ ,  $SD = 0.43$ ; the range for mothers' scores across T1 and T2 was 1 to 2.83 and the range for fathers' scores was 1 to 3.00).

#### Covariates

As reviewed earlier, several child and parent characteristics unrelated to the present study's

hypotheses have been identified as having associations with marital instability and child sleep problems (Belsky & Rovine, 1990; Cowan et al., 1985; Katzev et al., 1994; Kurdek, 1993; Moore et al., 2005; Sadeh et al., 1994; Schaefer, 1990; Seifer, Dickstein et al., 1996) and were therefore controlled for in the analyses.

*Child firstborn status.* Child birth order (0 = *firstborn* or 1 = *laterborn*) was measured via parent-report at T1.

*Adoptive parent anxiety.* At T1 and T2, AMs and AFs completed the 21-item Beck Anxiety Inventory (BAI; Beck & Steer, 1993). The adoptive parents rated the extent to which they had been bothered by each specific symptom (i.e., numbness, sweating) over the past week on the following scale: 1 (*not at all*) to 4 (*severely*). Anxiety symptoms were calculated as the sum of the 21 items (T1 AM  $\alpha = .75$ , T1 AF  $\alpha = .73$ ; T2 AM  $\alpha = .78$ , T2 AF  $\alpha = .81$ ).

*Infant difficult temperament.* At T1 and T2, each adoptive parent completed the seven-item Fussy-Difficult subscale of the Infant Characteristics Questionnaire (ICQ; Bates, Freeland, & Lounsbury, 1979), which assesses parental perception of infant negative emotionality (e.g., "How difficult is your baby to soothe?"). Higher scores indicated more difficult temperament (T1 AM  $\alpha = .84$ , T1 AF  $\alpha = .85$ ; T2 AM  $\alpha = .83$ , T2 AF  $\alpha = .85$ ).

### Statistical Analysis

As a preliminary step in our analyses, we performed a series of ordinary least squares regressions to partial out the effects of the covariates on the Marital Instability Index and the Bedtime Resistance subscale at T1 and T2. Residualized T1 and T2 scores were created separately for AMs and AFs by partialling out the following: birth order, AM-reported anxiety (T1, T2), AF-reported anxiety (T1, T2), AM-reported child difficult temperament (T1, T2), and AF-reported child difficult temperament (T1, T2). The variance-covariance matrices derived from these preliminary analyses were used in all subsequent statistical analyses. Listwise deletion procedures were employed in all analyses (listwise  $n = 338$ ). In addition, subgroup models tested separately by child sex replicated the pattern of effects observed for the combined sample; therefore, we report the combined models.

Primary statistical analyses proceeded in two stages. First, we specified cross-lagged and reciprocal-effects models using structural equation modeling (SEM; LISREL 8.50; Jöreskog & Sörbom, 1996) with maximum likelihood estimation to assess the

link between marital instability and child bedtime resistance across time. These models utilized latent measures of AM-/AF-reported marital instability (T1,  $r = .54$ ,  $p < .01$ ; T2,  $r = .47$ ,  $p < .01$ ) and utilized composite measures of AM-/AF-reported child bedtime resistance (T1,  $r = .80$ ,  $p < .01$ ; T2,  $r = .86$ ,  $p < .01$ ) to measure child sleep problems at T1 and T2. Second, to examine further the pattern of effects derived from these analyses and to separate potential rater effects, we conducted cross-lagged and reciprocal-effects analyses separately for mothers and fathers. Third, we additionally examined the pattern of association between mothers' and fathers' respective reports of marital instability and sleep problems separately by child sex. The use of a mother-father composite rating for child bedtime resistance and a latent variable approach to measuring marital instability helps ensure that any found associations are not merely the result of shared method variance.

## Results

Descriptive statistics for the raw variables and correlations for the residualized variables used in the SEM analyses are presented in Table 1. As is evidenced by the correlations, AM-reported marital instability at T1 was associated with child bedtime resistance concurrently,  $r = .13$ ,  $p < .05$ , and longitudinally,  $r = .18$ ,  $p < .01$ . AF-reported marital instability at T1 was not associated with child bedtime resistance concurrently,  $r = .06$ ,  $p > .10$ , but was associated with child bedtime resistance longitudinally,  $r = .12$ ,  $p = .05$ . Bedtime resistance at T1 was associated with AM-,  $r = .12$ ,  $p < .05$ , but not with AF-,  $r = .09$ ,  $p > .10$ , reported marital instability at T2. The concurrent association between T2 marital instability and T2 bedtime resistance was significant for both AMs,  $r = .23$ ,  $p < .01$ , and AFs,  $r = .18$ ,  $p < .01$ . Longitudinal correlations between each respective construct were moderate to strong in magnitude for AM- and AF-reported marital instability,  $r = .47$ ,  $p < .01$ , and  $r = .45$ ,  $p < .01$ , respectively, and for bedtime resistance,  $r = .65$ ,  $p < .01$ .

### *Establishing the Directionality of Pathways Between Marital Instability and Child Bedtime Resistance: CLPC Analyses*

Cross-lagged and panel correlation and synchronous-effects analyses examined whether a relation existed between marital instability and child

Table 1  
Intercorrelations Among Variables<sup>a</sup> Used in the SEM Analyses

Variable	1	2	3	4	5	6
1. AM marital instability (T1)	—					
2. AF marital instability (T1)	.54**	—				
3. Child bedtime resistance <sup>b</sup> (T1)	.13*	.06	—			
4. AM marital instability (T2)	.47**	.31**	.12*	—		
5. AF marital instability (T2)	.46**	.45**	.09	.51**	—	
6. Child bedtime resistance <sup>b</sup> (T2)	.18**	.12*	.65**	.23**	.18**	—
<i>M</i>	5.73	5.53	1.32	5.79	5.71	1.26
<i>SD</i>	2.22	1.63	0.38	2.01	1.87	0.41

Note. SEM = structural equation modeling; AM = adoptive mother; AF = adoptive father; T1 = Time 1; T2 = Time 2.

<sup>a</sup>The scores shown are residualized scores from the variance-covariance matrix that partialled out the effects of birth order, AM-reported anxiety (T1, T2), AF-reported anxiety (T1, T2), AM-reported child difficult temperament (T1, T2), and AF-reported child difficult temperament (T1, T2). Raw means and standard deviations are reported for ease of interpretation.

<sup>b</sup>The child Bedtime Resistance scores shown here are composite scores created from AM- and AF-rated bedtime resistance.

\* $p < .05$ . \*\* $p < .01$ .

bedtime resistance, or vice versa, across time and whether the effects between these constructs were reciprocal or unidirectional. Cross-lagged panel analysis permits the examination of the longitudinal relation between marital instability and child bedtime resistance while controlling for stability in each construct across time points. A significant cross-lagged effect would reflect a relation beyond that accounted for by stability of constructs and the magnitude of their association at T1 and T2 (Fincham, Beach, Harold, & Osbourne, 1997). Bidirectional, synchronous or reciprocal-effects analysis represents the specification of a nonrecursive model, such that each theoretical construct at T2 simultaneously predicts the other, controlling for initial levels of each construct and their correlation at T1. For a synchronous-effects model to be identified, several conditions need to be satisfied. The present model satisfies these conditions in that earlier measures of marital instability and child sleep problems serve as instrumental variables and are thereby uncorrelated with the disturbance terms in both T2 equations, and both cross-lagged effects are constrained to zero (see Fincham et al., 1997). A significant path between T2 marital instability and sleep disturbance, or vice versa, represents a contemporaneous influence of one variable on the other while simultaneously controlling for the opposite direction of effects, stability of each construct across time, and the correlation between each construct at T1.

As shown in Figure 1 (Panel a), T1 marital instability predicted increased T2 child bedtime resistance,  $\beta = .10$ ,  $p < .05$ . However, T1 child bedtime resistance did not predict T2 marital instability,  $\beta = .06$ ,  $p > .10$ . Stability coefficients were moderate

to strong for both marital instability ( $\beta = .71$ ,  $p < .01$ ) and child bedtime resistance ( $\beta = .64$ ,  $p < .01$ ) across the 9-month study period.

As shown in Figure 1 (Panel b), we also tested reciprocal effects at T2 while controlling for stability in these constructs across time. Once again, marital instability predicted child bedtime resistance,  $\beta = .14$ ,  $p < .05$ , but child bedtime resistance did not predict marital instability,  $\beta = .09$ ,  $p > .10$ . Stability coefficients were equivalent to those reported for the cross-lagged model,  $\beta_s = .70$  and  $.63$ ,  $p < .01$ , for each construct respectively. Subgroup analyses separating for child sex revealed no significant differences between boys and girls in the pattern of effects observed. Indicator loadings across both models were acceptable ( $\lambda$ s ranged from .55 to .99) and the goodness-of-fit statistics provided by each model were excellent: cross-lagged model,  $df = 3$ ,  $\chi^2 = 0.99$ ,  $p = .80$ , root mean square error of approximation (RMSEA) = .00, goodness-of-fit index (GFI) = 1.00, adjusted goodness-of-fit (AGFI) = .99, and reciprocal-effects model:  $df = 4$ ,  $\chi^2 = 0.99$ ,  $p = .91$ , RMSEA = .00, GFI = 1.00, AGFI = 1.00.

#### *Marital Instability and Child Sleep Problems: Mother- Versus Father-Driven Effects?*

In order to examine further the pattern of effects observed between parent-reported marital instability and children's bedtime resistance, we conducted a series of additional model tests separately for mothers and fathers. Two aspects of our previous model tests should be highlighted in order to preclude alternative explanations as to the pattern of effects observed. First, indicator loadings for each

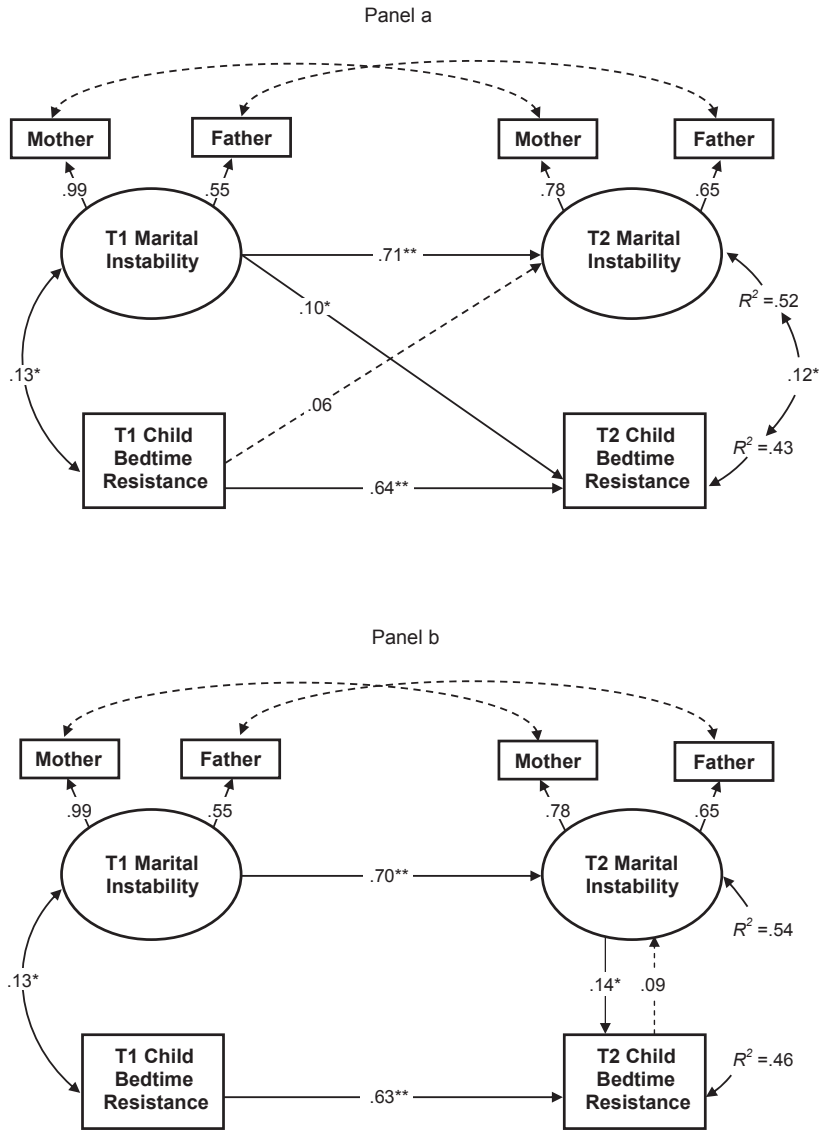


Figure 1. Maximum likelihood estimation of the relation between marital instability and child bedtime resistance: Panel a (cross-lagged results) and Panel b (reciprocal effects).

Note. Panel a:  $df = 3$ ,  $\chi^2 = 0.99$ ,  $p = .80$ , GFI = 1.00, AGFI = .99. Panel b:  $df = 4$ ,  $\chi^2 = 0.99$ ,  $p = .91$ , GFI = 1.00, AGFI = 1.00. GFI = goodness-of-fit index; AGFI = adjusted goodness-of-fit index; T1 = Time 1; T2 = Time 2.

\* $p < .05$ . \*\* $p < .01$ .

latent construct of marital instability are larger in magnitude for mother reports ( $\lambda_s = .99$  and  $.78$ ) compared to father reports ( $\lambda_s = .55$  and  $.65$ ), suggesting that the direction of effects between marital instability and child sleep problems may be distinct by parent sex such that combined effects are driven more by mothers than fathers. Second, the stability coefficients linking marital instability are larger in magnitude ( $\beta_s = .71$  and  $.70$ ) compared to those for child bedtime resistance ( $\beta_s = .64$  and  $.63$ ). This may artifactually assist the direction of effects observed in that a larger proportion of variance

remains eligible to be accounted for in T2 child sleep problems compared to T2 marital instability after initial levels of each variable are controlled within each respective model. We therefore examined cross-lagged and reciprocal-effects models separately for AMs and AFs to test whether the effects of T1 marital instability on child sleep problems at T2 were replicated (see Figure 2, Panel a and Panel b).

The pattern of effects observed in our original model tests were observed when analyses were conducted separately for AM- and AF-reported

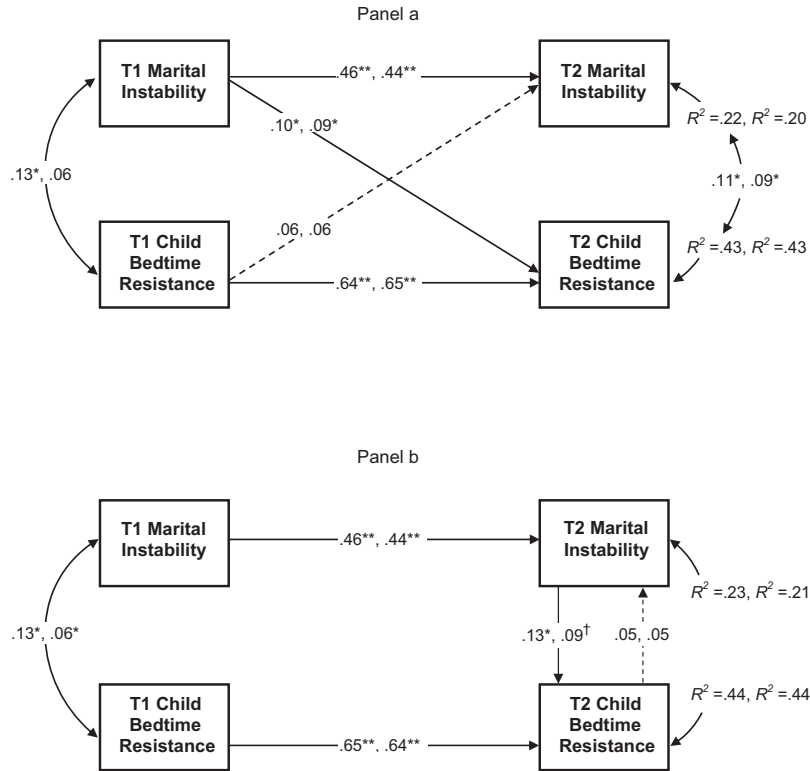


Figure 2. Maximum likelihood estimation of the relation between marital instability and child bedtime resistance shown separately for adoptive mothers (AM) and adoptive fathers (AF): Panel a (cross-lagged results) and Panel a (reciprocal effects). Note. Path coefficients and statistics for AMs (left) and AFs (right) are separated by commas. For AMs:  $df = 1$ ,  $\chi^2 = 1.53$ ,  $p = .22$ , GFI = 1.00, AGFI = .98. For AFs:  $df = 1$ ,  $\chi^2 = 1.84$ ,  $p = .20$ , GFI = 1.00, AGFI = .98. GFI = goodness-of-fit index; AGFI = adjusted goodness-of-fit index; T1 = Time 1; T2 = Time 2. <sup>†</sup> $p < .10$ . \* $p < .05$ . \*\* $p < .01$ .

marital instability, respectively. That is, marital instability significantly predicted child sleep problems both longitudinally (Panel a) and concurrently (Panel b), but child sleep problems did not predict marital instability in either model. Importantly, this direction of effects was retained even when the stability between T1 and T2 bedtime resistance was larger in magnitude compared to T1 and T2 marital instability (opposite pattern to that observed in the full SEM analyses), thereby precluding artifactual statistical influences (i.e., due to there being less variance to be accounted for in T2 bedtime resistance than in T2 marital instability) as a possible alternative explanation for the direction of effects observed between marital instability and child sleep problems, but not vice versa, in the present study.

### Discussion

The present investigation extends earlier transactional approaches to examining the reciprocal influences between marital relationship quality

and child adjustment (Cummings & Davies, 2002; Harold, Shelton, Goeke-Morey, & Cummings, 2004). Utilizing a prospective adoption design, we examined the relation between marital instability and child sleep problems at ages 9 and 18 months. Marital instability and child sleep problems showed a high degree of stability over time ( $r$ s ranged from .45 to .65). After controlling for this stability, and for associations between parent anxiety, birth order, and child difficult temperament, marital instability significantly predicted child sleep problems concurrently and across time. The reverse association from child sleep problems to marital instability was not evident. Furthermore, this pattern of association was replicated when the models were examined for mothers and fathers separately. Taken together, this pattern of results suggests that marital instability represents a risk factor in the emergence of child sleep problems early in development. Although children in the present study demonstrated normative variability in sleep problems (e.g., on average, parents rated each item on the Bedtime Resistance subscale as

occurring rarely [0–1 times] or sometimes [2–4 times] each week), prior research indicates that variations within the normal range of sleep problems predict later maladjustment (Bell & Belsky, 2008; Zuckerman et al., 1987).

Our findings indicate that the effects of marital instability on child sleep problems emerge earlier in development than has been demonstrated previously. Other researchers have focused on the impact of marital problems on older children's functioning (Davies et al., 2002; El-Sheikh et al., 2006). Despite evidence that parents of young infants report declines in marital satisfaction (Meijer & van den Wittenboer, 2007), we did not find that child sleep problems predicted marital instability once continuities and covariates were considered. This could be related to the early childhood period examined. In the second half of the 1st year of life, infants begin to establish more predictable nocturnal sleep patterns, remaining asleep for longer periods and demonstrating increased self-soothing abilities after waking (Burnham et al., 2002). Thus, although this sample was composed of very young children, by conducting our first assessment at 9 months of age, we may have missed the time during which infant sleep affects the marital relationship. However, given that previous studies have not directly examined the relation between marital instability and child sleep difficulties, this explanation is speculative. Furthermore, it is possible that other factors such as individual parental adjustment (Gross et al., 2008) or parental conflict (El-Sheikh et al., 2006) have a greater influence on the marital relationship during this developmental period.

The finding that marital instability predicted child sleep problems at ages 9 and 18 months is consistent with the broader context of research highlighting the importance of parent effects early in development when children are the most dependent on parents for their welfare (Bornstein, 2002). A range of factors, including parental well-being, parenting hassles, social support, and socioeconomic factors that can compromise care-giving quality have been associated with adjustment during early childhood (Crnic & Greenberg, 1987; Duncan, Brooks-Gunn, & Klebanov, 1994; Trentacosta et al., 2008). Although not a specific focus of this article, there is also evidence for parenting-mediated effects on child sleep problems (Bell & Belsky, 2008; Johnson & McMahon, 2008; Sadeh, Flint-Ofir, Tirosh, & Tikotzky, 2007). In the present investigation we limited our focus to studying the effects of marital instability on child sleep problems exclusive

of other potential mechanisms due to the lack of research on marital instability and child sleep problems in very early childhood and evidence for direct associations between marital relations and child adjustment in other domains (Crockenberg, Leerkes, & Lekka, 2007; Rhoades, 2008). However, examination of parenting-mediated effects is essential in future studies. For example, negative parenting has been shown to influence sleep problems in older children (Bell & Belsky, 2008). Given that behavioral control/discipline issues are not a central focus during the infancy period, additional theory is needed to guide decisions around which aspects of parenting to consider as mediators of child sleep problems during this developmental period.

Several factors should be taken into account when interpreting and generalizing the present findings. First, a noteworthy limitation was the use of parent reports to assess child sleep problems (although we attempted to minimize shared method bias by using a composite mother–father construct of child bedtime resistance and a latent variable approach to measuring marital instability). Although observational measures such as actigraphy and videosomnography have been used with infants and toddlers (Burnham et al., 2002; Sadeh, Lavie, Scher, Tirosh, & Epstein, 1991), behavioral sleep data were not collected in the present study. Second, the reliance on parent reports to assess both the predictor and outcome variables raises the possibility that the observed associations could be partly attributable to shared method variance. To control for this possibility, the SEM analyses examined marital instability as a latent variable and used an aggregate mother–father report of child sleep problems. Furthermore, despite observing both high levels of stability across time and high levels of interparent agreement for marital instability and child sleep problems, we found cross-lagged predictions from marital instability to child sleep problems. In addition, to rule out the possibility that one parent's report of marital instability was driving the observed pattern of associations, we examined the relationship between T1 marital instability and T2 child sleep problems for each parent separately. These secondary analyses confirmed the results obtained in the SEM analyses and increased our confidence that the pattern of relations observed is meaningful. Third, caution is warranted in generalizing the findings from the present study, given that the sample consisted of predominately White, well-educated, middle-class families. Although comparable in demographic

characteristics to adoptive families from the only other large-scale prospective adoption study (i.e., The Colorado Adoption Project; DeFries, Plomin, & Fulker, 1994), the families in the present study likely differed in some respects from biological families (Ceballo, Lansford, Abbey, & Stewart, 2004; Ferguson, Lynskey, & Horwood, 1995). For example, whereas family size and levels of anxiety and depression are similar for adoptive and biological families (Leve et al., 2007), adoptive parents have higher socioeconomic status (Kreider, 2003; Leve et al., 2008) and lower levels of antisocial behavior (Cloninger, Sigvardsson, Bohman, & von Knorring, 1982). Furthermore, evidence of racial/ethnic and cultural differences in how parents manage and perceive their children's sleep (Liu, Liu, Owens, & Kaplan, 2005; Milan, Snow, & Belay, 2007) highlights the importance of examining marital instability and child sleep problems in more diverse samples.

Despite these limitations in the nature of the sample, the use of a prospective adoption sample was also a primary strength of the present study. Because the parents and children were not biologically related, we were able to rule out the possibility that the associations observed between marital instability and child sleep disturbances in the present study were attributable to shared genetic influences on behavior. Our findings do not permit an examination of the degree to which the association between marital instability and child sleep problems is overestimated in nongenetically informed studies, nor do they permit insight into other forms of gene-environment interplay, such as effects of the child's genetically influenced behavior on caregiver behavior (i.e., evocative gene-environment correlation), which would require a twin sample or sample of biologically reared children. However, this is the first study of which we are aware to rule out the role of shared genes on associations between marital instability and child sleep problems. Furthermore, the current study adds to growing evidence that environmental influences on child development previously observed in between-family research designs (e.g., parental divorce and child delinquency) replicate when examined via more conservative genetically informed designs (e.g., Burt et al., 2008; Roisman & Fraley, 2008).

The present findings also suggest several avenues for further investigation. First, as noted above, given associations between child sleep and parenting (Bell & Belsky, 2008) and between the marital relationship and parenting (Shelton &

Harold, 2008), future research should examine the extent to which parenting behaviors mediate or moderate associations between the marital relationship and child sleep. Second, future studies may want to move beyond self-report approaches and incorporate behavioral and neurophysiological measures to more directly assess how marital instability affects children's neurobiological functioning over time. For example, the relation between normative variations in child sleep and marital instability might be mediated by the impact of family stress on the neurobiological mechanisms that underlie sleep regulation (see Van Goozen, Fairchild, Snoek, & Harold, 2007), although additional research is needed to further test this hypothesis. Exposure to early life stress has been shown to impact the functioning of neurobiological stress- and threat-response systems (i.e., amygdala and HPA axis; Heim & Nemeroff, 2001; LeDoux & Phelps, 2000). Given that these systems are particularly vulnerable to the effects of adverse environments early in development (e.g., Sánchez, Ladd, & Plotsky, 2001), heightened physiological arousal associated with activation of these systems during infancy and toddlerhood could be related to increased sleep disruption (Dahl, 1996). Third, through the collection of DNA or the measurement of phenotypes related to sleep in birth parents, future research could extend the current study to examine specific genetic influences on the associations between child sleep problems and marital instability. Such studies might yield insight into additional mechanisms whereby genetic influences might affect family systems. For example, as noted above, genetic influences in the child might evoke or elicit specific kinds of reactions from caregivers (e.g., Ge et al., 1996) and, as such, be identified as a target for preventive intervention.

The results from the present study add to an expansive body of research highlighting the deleterious effects of marital distress on children (Cummings & Davies, 2002; Harold et al., 2007). This study contributes to the field by examining the direction of effects between marital instability and child sleep problems, a variable with known adverse effects on a variety of socioemotional and behavioral outcomes during later childhood (El-Sheikh et al., 2007; Goodnight, Bates, Staples, Pettit, & Dodge, 2007). By studying these effects within the context of a prospective longitudinal adoption design that could eliminate the potentially confounding presence of shared genetic influences, we were able to more precisely attribute both

directionality and environmental causality on the association between marital instability and sleep problems in early childhood.

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