VI. MARITAL CONFLICT, VAGAL REGULATION, AND CHILDREN’S SLEEP: A LONGITUDINAL INVESTIGATION

Mona El-Sheikh, J. Benjamin Hinnant, and Stephen A. Erath

ABSTRACT We examined longitudinal relations between adult interpartner conflict (referred to as marital conflict) and children’s subsequent sleep minutes and quality assessed objectively via actigraphy, and tested parasympathetic nervous system (PNS) activity indexed through respiratory sinus arrhythmia reactivity (RSA-R) and initial sleep as moderators of predictive associations. At Wave 1 (W1), children (85 boys, 75 girls) with a mean age of 9.43 years ($SD = .69$) reported on marital conflict, and their sleep was assessed with actigraphs for seven nights. Sleep minutes, sleep efficiency, sleep activity, and number of long wake episodes were derived. RSA-R was measured in response to a lab challenge. Sleep parameters were assessed again 1 year later at Wave 2 (W2; mean age = 10.39; $SD = .64$). Analyses consistently revealed 3-way interactions among W1 marital conflict, sleep, and RSA-R as predictors of W2 sleep parameters. Sleep was stable among children with more sleep minutes and better sleep quality at W1 or low exposure to marital conflict at W1. Illustrating conditional risk, marital conflict predicted increased sleep problems (reduced sleep minutes, worse sleep quality) at W2 among children with poorer sleep at W1 in conjunction with less apt physiological regulation (i.e., lower levels of RSA-R or less vagal withdrawal) at W1. Findings build on the scant literature and underscore the importance of simultaneous consideration of bioregulatory systems (PNS and initial sleep in this study) in conjunction with family processes in the prediction of children’s later sleep parameters.

Sleep problems in childhood are common, with estimates ranging from 20% to 40% (Mindell & Owens, 2010). Understanding factors that influence children’s sleep and individual differences that can exacerbate sleep problems is critical. According to Dahl’s (1996) model, sleep involves diminished attentiveness and responsiveness, and it is most adaptive for sleep

Corresponding author: Mona El-Sheikh, Human Development and Family Studies, 203 Spidle Hall, Auburn University, Auburn, AL 36849-5214, USA, email: elshemm@auburn.edu

This research was supported by National Institute of Health R01-HL093246.
to occur at times and places that are relatively free of threat. Paired with the emotional security model (Cummings & Davies, 2010), which posits that feelings of emotional security derive from family relationships that children perceive as safe, caretaker conflicts are expected to produce psychological distress, interfere with children’s sense of security, and undermine sleep.

Longitudinal relations between adult interpartner conflict in either the marital or cohabitating relationship (referred to as marital conflict) and children’s normative sleep problems, including fewer sleep minutes and poor sleep quality indicated by nighttime awakenings assessed objectively with actigraphy, were examined. Two bioregulatory systems with potential protective or vulnerability functions for future sleep problems were considered in the context of marital conflict: (1) individual differences in parasympathetic nervous system (PNS) activity indexed by respiratory sinus arrhythmia reactivity (RSA-R) to a lab challenge, and (2) prior sleep minutes and quality.

Marital Conflict and Children’s Sleep

Marital conflict may involve physical or psychological forms (Cummings & Davies, 2010). Physical conflict refers to bodily assault, and psychological conflict involves verbal aggression, demeaning behaviors, and/or actions. Although cross-sectional associations between marital conflict and children’s sleep have been documented (El-Sheikh, Buckhalt, Cummings, & Keller, 2007), few studies have examined whether marital conflict predicts sleep problems over time (for a review, see El-Sheikh & Kelly, 2011). Kelly and El-Sheikh (2011) found that marital conflict predicted child-reported sleepiness and sleep/wake problems 2 years later, controlling for earlier levels of the respective sleep parameter. Consistent with cumulative risk (Evans & English, 2002) and biopsychosocial (El-Sheikh & Erath, 2011) models of development, it is possible that marital conflict predicts sleep problems among a subset of children, depending on the functioning of stress-sensitive, bioregulatory systems that leave them more or less susceptible to marital conflict. The study of physiological stress response systems has facilitated understanding of developmental psychopathology (Cicchetti & Gunnar, 2008) and such investigations are equally important for understanding sleep from a developmental perspective.

Bioregulatory Moderators: PNS Activity and Sleep

The PNS is activated to serve “rest and digest” functions or is withdrawn to facilitate engaged responses to stress. Heart rate variability mediated through activity of the ventral vagal complex is commonly used to index PNS activity in children and is operationalized via RSA (Berntson, Cacioppo, & Grossman, 90
RSA activity is somewhat analogous to the function of brakes on a vehicle; higher resting levels of RSA indicate that the “brakes” are on, which promotes lower heart rate and “rest and digest” functions but may not be adaptive for coping with stress when the body needs to mobilize resources. Greater RSA-R (i.e., greater reductions in RSA from baseline to stress or challenge; greater vagal withdrawal or suppression) is analogous to letting off the “brake” and corresponds with increased heart rate and blood flow and reflects adaptive physiological engagement with environmental stress (Porges, 2007). Consistent with this conceptualization, positive associations between RSA-R and attentional, emotional, and behavioral regulation have been documented (Porges, 2007). Greater RSA-R also operates as a protective factor against internalizing and externalizing behaviors in the context of marital conflict (for a review, see El-Sheikh & Erath, 2011). Conversely, lower levels of RSA-R (suppression) or RSA augmentation (i.e., greater increases in RSA from baseline to stress or challenge) function as vulnerability factors in the association between marital conflict and adjustment problems (El-Sheikh & Erath, 2011).

Better sleep may also operate as a protective factor in the context of marital conflict. Longer and better quality sleep can promote and poor sleep can undermine executive functioning and the capacity to regulate emotions (Jones & Harrison, 2001; Muzur, Pace-Schott, & Hobson, 2002), accurate interpretations of emotional experiences (Soffer-Dudek, Sadeh, Dahl, & Rosenblat-Stein, 2011), as well as consolidation of emotional memory (Walker & van der Helm, 2009). Consistent with the protective function of better sleep, the association between parental psychological control and child-reported internalizing symptoms was not observed among children with higher sleep quality and higher socioeconomic status (El-Sheikh, Hinnant, Kelly, & Erath, 2010). Furthermore, less optimal sleep can function as a vulnerability factor and exacerbate negative outcomes especially for children exposed to socioeconomic adversity (Buckhalt, 2011). Similarly, sleep may enhance or compromise children’s capacity to cope with the negative thoughts and feelings associated with marital conflict, and moderate child outcomes, including later sleep duration and quality.

**Aims and Hypotheses**

There is moderate stability in the associations between sleep parameters across times and contexts in childhood (El-Sheikh, Kelly, Buckhalt, & Hinnant, 2010) and there is recognition that trait-like biological factors underlie this stability (Jenni, Molinari, Caflisch, & Largo, 2007). At the same time, environmental factors such as exposure to marital conflict may affect biological regulation, including sleep. Thus, the current study investigates predictors of sleep at the level of the *immediate context* through marital conflict.
and at the level of the child context through RSA-R and prior sleep (i.e., within individual) (see El-Sheikh & Sadeh, Figure 1, Chapter I, in this volume). Late childhood may be a particularly important developmental period in which to study interactions between environmental and bioregulatory systems because of the rapid physiological changes occurring at the pubertal transition and increased sensitivity to social stress. Furthermore, in comparison to the literature with infants and adolescents, investigations of sleep and child development are fewer with elementary school-aged children. As discussed by El-Sheikh and Sadeh in Chapter I of this monograph, examination of prior sleep in the prediction of later sleep parameters, in terms of direct effects and interactions with environmental stressors (marital conflict in this study) and other facets of biological regulation (PNS activity), could lead to a more nuanced understanding of sleep problems within a biopsychosocial framework.

We assessed longitudinal relations between marital conflict at Wave 1 (W1) and children’s sleep problems 1 year later (Wave 2; W2), and we hypothesized that marital conflict would predict increased sleep problems over time. Our reference to sleep problems do not indicate clinically significant disorders, but rather fewer sleep minutes and worse sleep quality along a continuum. Consistent with best practices, multiple measures of

![Graphs showing the interaction between Wave 1 marital conflict, sleep, and RSA-R predicting Wave 2 sleep minutes (a), efficiency (b), activity (c), and long wake episodes (d).](image)

FIGURE 1.—Interaction between Wave 1 marital conflict, sleep, and RSA-R (respiratory sinus arrhythmia reactivity) predicting Wave 2 sleep minutes (a), efficiency (b), activity (c), and long wake episodes (d).
objective (actigraphy-based) sleep parameters and a multi-informant and multi-method approach were utilized in this study. We expected that these predictive associations would be stronger among children with bioregulatory vulnerabilities, including initial sleep problems and lower levels of RSA-R (i.e., less vagal withdrawal) in response to stress at W1. Conversely, better initial sleep or higher levels of RSA-R (i.e., greater vagal withdrawal) were expected to function as protective factors, attenuating predictive associations between marital conflict and subsequent sleep problems. We conceptualize vulnerability and protection on a continuum (Rutter, 1993). These moderating effects were examined through two-way interactions with either RSA-R or W1 sleep as moderators. Further, three-way interactions among W1 marital conflict, sleep, and RSA-R in the prediction of W2 sleep were conducted with the expectation that children with multiple risk factors (worse sleep at W1 in conjunction with lower levels of RSA-R) would be most at risk for more sleep insufficiency and worse sleep quality at W2.

METHOD

Participants

The sample was drawn from the first and second waves of a study examining biopsychosocial influences on children’s health; third and fourth graders and their primary caregivers were recruited from schools in semi-rural towns in the Southeastern United States. Children (85 boys, 75 girls; W1 mean age = 9.43 years; SD = .69; W2 mean age = 10.39 years; SD = .64) were between 8 years and 10 years at recruitment (W1), had no diagnosed learning disability or sleep disorder based on mothers’ report, resided in two-parent homes, and had sleep outcome data at W2; data collection occurred in 2009–2011. Children’s ethnicity was 73% European American and 27% African American. Familial income ranges reported by mothers at W1 were: 12% less than $10,000; 8% between $10,000 and $20,000; 18% between $20,000 and $35,000; 24% between $35,000 and $50,000; 22% between $50,000 and $75,000 and 16% reported incomes greater than $75,000. Primary caregivers were biological parents (68%; n = 122); one biological parent and a partner (31%; n = 55); and adoptive parents (1%; n = 2).

Procedures

We refer to the child’s primary caregivers as parents (mothers and fathers) and to interpartner conflict as marital conflict. Informed consent and assent were obtained. Parents placed an actigraph on the child’s non-dominant wrist before bed time for seven consecutive evenings; actigraphic
assessments were validated through daily parent reports of bed and wake times. Sleep data were collected during the regular school year; nights with medication use for acute illnesses were excluded from analyses. Then, children and their parents visited our laboratory ($M=3.05$ days, $SD=8.10$ between sleep and physiological assessments). RSA was collected while children were seated and quiet and in response to a laboratory stressor: 3 minutes adaptation, 3 minutes baseline, and a 3 minutes star-tracing task (Lafayette Instrument Company, Mirror Tracer). This task is frustrating and consistently elicits RSA withdrawal (El-Sheikh, Hinnant, & Erath, 2011).

Measures

Marital Conflict

Children’s reports on the Conflict Tactics Scale-2 (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) were used to measure the frequency of caregiver marital conflict. Children are considered valid informants of the marital conflict to which they are exposed (Grych, Seid, & Fincham, 1992). The established Psychological/Verbal Aggression and Physical Aggression scales of the CTS2 were pertinent to the present study. Children rated how frequently the mother and the father used the specified type of conflict tactic within the past year. A score of 0 = did not happen in the past year; 1 = once; 2 = 2 times; 3 = 3—5 times; 4 = 6—10 times; 5 = 11—20 times; 6 = > 20 times. The following four conflict scales were examined: Father-initiated verbal and physical, and Mother-initiated verbal and physical; $\alpha = .67–.81$. Based on children’s reports, 12% of mothers and 15% of fathers engaged in physical aggression against the partner. In total, 16% of children reported physical aggression perpetrated by either the mother or father. Average verbal aggression was 3.79 ($SD = 4.12$) for mother-initiated conflict and 4.36 ($SD = 4.68$) for father-initiated conflict; overall average was 4.01 ($SD = 4.40$), indicative of a wide range in the sample. In the context of scores given above, a score of 4 indicates six to ten acts of verbal conflict within the past year. Children reported that 23% of mothers and 34% of fathers engaged in verbal aggression against the partner. In total, 46% of children reported verbal aggression perpetrated by either the mother or father.

RSA

Data were collected with the MW1000A acquisition system (Mindware Technologies Inc., Gahanna, OH) and analyzed with the Mindware analysis system. A Mindware BioNex 8-slot chassis was used to collect ECG data. Cardiovascular responses were recorded via the ECG activity amplifier module and disposable snap pediatric ECG electrodes using a standard lead II configuration. Respiration was calculated through spectral analysis of thoracic impedance (Ernst, Litvack, Lozano, Cacioppo, & Berntson, 1999). Data were scored in 1-minute intervals using Mindware software (HRV 3.0.1). Cardiovascular data were inspected for artifacts and missing or misplaced
R-peaks based on improbable inter-beat intervals; these were edited manually by a trained researcher. RSA was calculated as the natural log of the high frequency power (.15–.40 HZ), is expressed in milliseconds, and is a validated method for isolating parasympathetic vagal influence on the heart (Berntson et al., 1997). RSA-R was computed by subtracting RSA during the baseline from RSA during the star-tracing task. A negative reactivity score indicates RSA withdrawal.

Actigraphy

Octagonal Basic MotionLogger (Ambulatory Monitoring Inc., Ardsley, NY) actigraphs measured activity between bedtime and wake time in 1-minute epochs using zero crossing mode. Data were downloaded and transformed into sleep variables using the Octagonal Motionlogger Interface with ACTme software and the analysis software package (Action-W User’s Guide, 2002). Procedures for setting times for sleep onset followed a lab protocol created at the E. P. Bradley Hospital Sleep Laboratory at Brown University (Acebo & Carskadon, 2001). Data were scored using the Sadeh algorithm (Sadeh, Sharkey, & Carskadon, 1994).

When using actigraphy, the assessment of multiple sleep variables is recommended to tap a wide range of sleep parameters (Sadeh, Raviv, & Gruber, 2000). We derived: (a) Sleep Minutes-total number of minutes between sleep onset and wake time that were scored as sleep; (b) Sleep Efficiency-percentage of actual sleep during the sleep period (sleep onset to offset); (c) Sleep Activity-percentage of epochs during the night with activity; and (d) Long Wake Episodes-number of wake episodes lasting for longer than 5 minutes. Children had an average of 6.20 days ($SD = 1.68$) of valid actigraphy data. Intraclass correlations indicated good night-to-night stability over the week for the various actigraphy-based sleep parameters ($\alpha s = .86–.95$).

Demographic/Child Characteristics

W1 child sex, ethnicity, illness/disorder as reported by mothers ($n = 6$; e.g., migraines, controlled seizures, autism spectrum, sickle cell), and caregiver-child relationship status/family structure (i.e., biological parents) were dichotomized. Children’s height and weight were measured, and body mass index (BMI) was calculated [703 × (weight/height$^2$); http://www.cdc.gov]. Children in the sample had a mean BMI of 19.03 ($SD = 4.64$). Mothers’ reports on the Puberty Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) revealed that the mean puberty score was 1.44 ($SD = .29$) for boys and 1.71 ($SD = .45$) for girls, indicating that most children were prepubertal.

Plan of Analysis

Potential confounds related to children’s sleep included sex, age, ethnicity, BMI, severe illness/disorder, pubertal status, family income, and
whether the child resided with/reported on biological parents or other caregivers. Only BMI and the presence of an illness were related to W2 sleep in our models and were retained as covariates. At W1 and W2, a few outlier data points (≥4 SDs; n = 1–3) were removed for sleep minutes, sleep efficiency, sleep activity, and long wake episodes; one RSA-R outlier was removed at W1. The sources of the outliers (sleep actigraphy and RSA) suggested possible equipment or sensor failure. Sensitivity analyses indicated that inclusion of these questionable data points that were removed did not alter the results presented in the paper. All marital conflict variables were skewed (kurtosis statistics ≥2) and were log transformed (Pedhazur, 1997).

Regression analyses in AMOS took advantage of Full Information Maximum Likelihood estimation. The marital conflict variable was derived from a latent construct of child-reported: father-initiated verbal conflict, father-initiated physical conflict, mother-initiated verbal conflict, and mother-initiated physical conflict, which was exported as a factor score. Fit for the measurement model of marital conflict was acceptable; Root Mean Squared Error of Approximation or RMSEA = .08. Observed RSA measures were: (1) baseline RSA and (2) RSA-R; because RSA-R is influenced by baseline levels of RSA, the latter was controlled in analyses. The sleep measures at W1 and W2 were all observed and included: sleep minutes, sleep efficiency, sleep activity, and number of long wake episodes. Significant interactions were examined (Aiken & West, 1991) and plotted using Preacher’s interaction utility (Preacher, Curran, & Bauer, 2006) at high (+1 SD) and low (−1 SD) levels of the predictor and moderators.

RESULTS

At W1, on average and based on sleep diaries, children went to bed at 8:59 p.m. (SD = 41 minutes) and woke up at 6:27 a.m. (SD = 37 minutes) (Table 1). At W2, children went to bed at 9:10 p.m. (SD = 44 minutes) and woke up at 6:30 a.m. (SD = 50 minutes). Bivariate relations involving the two influential covariates (BMI, illness) indicated that W1 BMI (M = 19.02; SD = 4.63) was significantly associated with Wave 1 RSA-B (r = −.16), W1 Sleep minutes (r = −.15), and W2 Sleep Activity (r = .20). Illness/disorder (M = .03; SD = .16) was associated with some variables in regression models but not in bivariate analyses.

In the main analyses, each W2 sleep parameter was predicted by W1 sleep, potential confounds (i.e., BMI and illness/disorder), the two-way interaction between conflict and RSA-R, the two-way interaction between RSA-R and W1 sleep, and the three-way interaction between conflict, RSA-R, and W1 sleep. Fit indices for all regression models were acceptable; no RMSEA value
<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. RSA-B (msec)</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. RSA-R/C0</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Marital conflict—W1</td>
<td></td>
<td></td>
<td></td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Sleep Minutes—W1</td>
<td>.06</td>
<td>.01</td>
<td>–</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Sleep Efficiency—W1</td>
<td>.13</td>
<td>.07</td>
<td>–.12</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Sleep Activity—W1</td>
<td>.07</td>
<td>.10</td>
<td>–.05</td>
<td>.78**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Long Wake Episodes—W1</td>
<td>.09</td>
<td>.13</td>
<td>.05</td>
<td>–.76**</td>
<td>–.97**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Sleep Minutes—W2</td>
<td>.08</td>
<td>.04</td>
<td>.08</td>
<td>–.64**</td>
<td>–.87**</td>
<td>.81**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Sleep Efficiency—W2</td>
<td>.17**</td>
<td>–.03</td>
<td>.02</td>
<td>.25**</td>
<td>.42**</td>
<td>–.32**</td>
<td>–.33**</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Sleep Activity—W2</td>
<td>.01</td>
<td>.08</td>
<td>.03</td>
<td>.37**</td>
<td>.35**</td>
<td>–.40**</td>
<td>–.43**</td>
<td>.72**</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Long Wake Episodes—W2</td>
<td>–.09</td>
<td>.05</td>
<td>–.04</td>
<td>–.22**</td>
<td>–.34**</td>
<td>.34**</td>
<td>.35**</td>
<td>–.69**</td>
<td>–.96**</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>459.92</td>
<td>88.68</td>
<td>12.95</td>
<td>3.57</td>
<td>451.37</td>
<td>88.49</td>
<td>13.16</td>
<td>3.49</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(SD)</td>
<td>(1.02)</td>
<td>(.66)</td>
<td>(.59)</td>
<td>(55.79)</td>
<td>(6.98)</td>
<td>(6.03)</td>
<td>(2.12)</td>
<td>(44.91)</td>
<td>(6.92)</td>
<td>(5.81)</td>
<td>(2.36)</td>
</tr>
</tbody>
</table>

Note. RSA-B = Respiratory sinus arrhythmia during baseline; RSA-R = Respiratory sinus arrhythmia reactivity to lab challenge. Mean of untransformed marital conflict 2.92 (SD = 2.98).

*p < .05.

**p < .01.
exceeded .08. Findings for the prediction of all W2 sleep outcomes are presented in Table 2. Main effects in the models accounted for between 14% and 23% of the variance in W2 sleep parameters, primarily because of the autoregressive effects of W1 sleep. Evidence showed interactions between W1 conflict, children's sleep, and RSA-R as predictors of W2 sleep. Interactions accounted for significant amounts of variance in W2 sleep parameters, ranging from 11% to 17% of the variance. Below, we interpret the highest order interaction predicting each of the W2 sleep parameters.

**Sleep Minutes**

Figure 1a shows that for children with fewer sleep minutes at W1 (i.e., \(-1 \times SD\)) and lower levels of RSA withdrawal (i.e., \(-1 \times SD\)), marital conflict was negatively related to W2 sleep minutes (although the slope looks significant, it only approached conventional levels of statistical significance). Conversely, for children with fewer sleep minutes at W1 and higher levels of RSA-R (greater RSA withdrawal; \(+1 \times SD\)), marital conflict was positively related to W2 sleep minutes. In the context of more sleep minutes at W1, W2 sleep minutes were consistently high (i.e., marital conflict was not predictive of W2 sleep minutes). Thus, greater RSA-R functioned as a protective factor in the relation between marital conflict and children's future sleep, but only for children who already suffer from fewer sleep minutes. Further, predicted means indicated that the shortest sleep at W2 was observed for children who were exposed to higher levels of marital conflict and had poor sleep at W1 in conjunction with lower levels of RSA-R (\(M = 405.21\) minutes or 6.75 hr). In the context of higher marital conflict, the three other groups exhibited similarly longer sleep (\(M = 463.52\) minutes or 7.73 hr to 476.13 minutes or 7.94 hr). Interactions accounted for 11% of the variance in W2 sleep minutes and the whole model accounted for 29% of the variance in this sleep variable (see Table 2).

**Sleep Efficiency**

Similar results for W2 sleep efficiency were found. Figure 2b shows that W1 marital conflict was not related to W2 sleep efficiency for children who had better efficiency at W1. W2 sleep efficiency for these children was consistently high. By contrast, children with lower sleep efficiency at W1 showed different relations between marital conflict and W2 sleep, which were dependent on RSA-R. Marital conflict was positively related to W2 efficiency for children who had less sleep efficiency and greater RSA-R at W1. A negative association was observed between conflict and W2 sleep efficiency for children with both lower efficiency and lower levels of RSA-R at W1 (\(p = .05\)). Similar to sleep minutes, the lowest level of sleep efficiency at W2 was observed
<table>
<thead>
<tr>
<th>Wave 1 Variables</th>
<th>Sleep Minutes</th>
<th>Sleep Efficiency</th>
<th>Sleep Activity</th>
<th>Long Wake Episodes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$B$ ($SE$)</td>
<td>$\beta$</td>
<td>$R^2$</td>
<td>$B$ ($SE$)</td>
</tr>
<tr>
<td>BMI</td>
<td>$-0.52$ (.67)</td>
<td>$0.05$</td>
<td>$-0.03$ (.10)</td>
<td>$-0.02$</td>
</tr>
<tr>
<td>Illness/Disorder</td>
<td>$58.40$ (18.97)</td>
<td>$0.21^{**}$</td>
<td>$2.96$ (2.90)</td>
<td>$0.07$</td>
</tr>
<tr>
<td>RSA-B</td>
<td>$5.88$ (3.22)</td>
<td>$0.13$</td>
<td>$0.23$ (.49)</td>
<td>$0.03$</td>
</tr>
<tr>
<td>RSA-R</td>
<td>$-4.92$ (5.24)</td>
<td>$-0.07$</td>
<td>$-0.25$ (.79)</td>
<td>$-0.02$</td>
</tr>
<tr>
<td>Sleep parameter$^a$</td>
<td>$0.36$ (.06)</td>
<td>$0.45^{**}$</td>
<td>$0.47$ (.07)</td>
<td>$0.47^{**}$</td>
</tr>
<tr>
<td>Marital conflict</td>
<td>$4.80$ (5.40)</td>
<td>$0.06$</td>
<td>$0.95$ (.81)</td>
<td>$0.08$</td>
</tr>
<tr>
<td>Main effects</td>
<td></td>
<td>$0.18$</td>
<td>$0.17$</td>
<td>$0.14$</td>
</tr>
<tr>
<td>MC $\times$ RSA-R</td>
<td>$-23.46$ (9.45)</td>
<td>$0.19^*$</td>
<td>$-4.08$ (1.45)</td>
<td>$-0.21^{**}$</td>
</tr>
<tr>
<td>MC $\times$ Sleep$^a$</td>
<td>$-0.10$ (.12)</td>
<td>$-0.07$</td>
<td>$-0.03$ (.15)</td>
<td>$-0.02$</td>
</tr>
<tr>
<td>RSA-R $\times$ Sleep$^a$</td>
<td>$0.33$ (.10)</td>
<td>$0.34^{**}$</td>
<td>$0.53$ (.12)</td>
<td>$0.52^{**}$</td>
</tr>
<tr>
<td>MC $\times$ RSA-R $\times$ Sleep$^a$</td>
<td>$0.30$ (.15)</td>
<td>$0.20^*$</td>
<td>$0.46$ (.17)</td>
<td>$0.32^{**}$</td>
</tr>
<tr>
<td>Interactions</td>
<td></td>
<td>$0.11$</td>
<td>$0.12$</td>
<td>$0.17$</td>
</tr>
<tr>
<td>$R^2$ explained by full models</td>
<td></td>
<td>$0.29$</td>
<td>$0.29$</td>
<td>$0.31$</td>
</tr>
</tbody>
</table>

Notes: Unstandardized coefficients are presented in the first column and standard errors are in parentheses. Standardized regression coefficients are presented in the second column ($\beta$). RSA-B = Respiratory sinus arrhythmia during baseline; RSA-R = Respiratory sinus arrhythmia reactivity to lab challenge; MC = Marital Conflict.

$^a$Sleep parameter refers to the corresponding measure at Wave 1 (e.g., Wave 1 sleep minutes when predicting Wave 2 sleep minutes).

*p < .05.

**p < .01.
for children exposed to higher marital conflict who had lower levels of both W1 sleep efficiency and RSA-R ($M = 80.21\%$). Conversely, the three other groups of children exhibited better sleep efficiency in the context of higher marital conflict ($M = 90.14–93.94\%$); sleep efficiency $< 90\%$ indicates poor sleep quality (Sadeh, Raviv, & Gruber, 2000). Interactions accounted for 12\% of the variance in W2 sleep efficiency and the whole model accounted for 29\% of the variance.

### Sleep Activity

Children with higher levels of W1 sleep activity in conjunction with lower levels of RSA-R exhibited the highest levels of W2 sleep activity (Figure 1c). For children with both higher W1 sleep activity and RSA-R, marital conflict was negatively related to W2 activity, which offers further support for the role of RSA-R as a protective factor in the relation between marital conflict and future sleep, but only for children who already suffer from relatively poor sleep. For children with lower levels of W1 sleep activity, marital conflict was unrelated to W2 activity, regardless of RSA-R. Predicted means show children with the highest level of sleep activity at W2 were those exposed to higher marital conflict and had both high sleep activity at W1 and lower levels of RSA-R ($M = 21.02$); other groups exposed to higher marital conflict had substantially lower sleep activity scores ($7.63–12.22$). The interactions accounted for 17\% of variance in W2 sleep activity and the total model accounted for 31\% of the variance.

### Long Wake Episodes

Marital conflict was positively related to W2 long wake episodes for children with lower levels of RSA-R and more frequent long wake episodes at W1 (Figure 1d). Conversely, this relation was negative for children with higher levels of RSA-R and more frequent W1 wake episodes. There were no significant relations between marital conflict and W2 wake episodes for children with fewer wake episodes at W1, regardless of RSA-R. Children with the highest number of long wake episodes were those exposed to higher marital conflict and who had both worse sleep and lower levels of RSA-R (predicted mean $= 6.42$). Their counterparts who were still exposed to higher levels of marital conflict but had other combinations of prior sleep problems and RSA-R had wake episodes that ranged between 1.81 and 3.01. Note that $\geq 3$ such long wake episodes may indicate significant sleep problems (Sadeh, Raviv, & Gruber, 2000). Interactions accounted for 11\% of the variance in W2 wake episodes and the full model accounted for 34\% of the variance in this sleep variable.
DISCUSSION

This study demonstrates the importance of interactions between marital conflict, bioregulation, and initial sleep in the longitudinal prediction of sleep problems. Main effects of marital conflict on sleep were not observed, but three-way interactions among marital conflict, initial sleep, and RSA-R predicted later sleep. Two-way interactions were also evident but were subsumed by three-way interactions that were interpreted. A consistent pattern of interactive effects emerged across actigraphic assessments of sleep minutes and quality parameters. Among children with better sleep (more minutes, higher efficiency, lower activity, and fewer long wake episodes) at W1, sleep was highly stable, remaining high in minutes or quality at W2 regardless of the level of marital conflict or vagal regulation (RSA-R). Sleep was also stable among children with poorer sleep at W1 in conjunction with low exposure to marital conflict. Thus, in families with low levels of marital conflict, children’s sleep was stable, remaining either relatively more or less optimal. This is supportive of research on the moderate stability of sleep in children over time and is consistent with the recognition that trait-like biological factors underlie this stability (Jenni, Molinari, Caflisch, & Largo, 2007). The pattern of interaction effects was consistent and indicates that longer and better quality sleep exerts a broad protective effect (Luthar, Cicchetti, & Becker, 2000) in relations between marital conflict and subsequent sleep. In contrast, in families with high levels of marital conflict, sleep improved over time among children with higher levels of RSA-R (increased withdrawal) and worsened over time among children with lower levels of RSA-R. In particular, higher marital conflict predicted lower sleep efficiency, higher sleep activity, and more frequent long wake episodes at W2 among children with poor initial sleep accompanied by lower levels of RSA-R. Results suggest that sleep may be more volatile among children with initially poor sleep (depending on family stress and PNS regulation) and more stable among children with initially better sleep (even in the context of marital conflict). These complex patterns of interactions indicate that RSA-R, in combination with poor sleep, has a protective-enhancing effect, meaning that greater RSA-R seems to play a unique role in supporting engagement with stressful contexts to predict better sleep outcomes, even at high levels of risk (Luthar et al., 2000).

Associations between marital conflict and children’s disrupted sleep bridge conceptual frameworks, which propose that secure conditions enable sleep (and threatening conditions undermine sleep; Dahl, 1996) and that feelings of emotional security derive from family relationships that are perceived as safe and supportive (Cummings & Davies, 2010). Children’s experiences with marital conflict and possibly increased threat to security may produce arousal and vigilance, and thereby disrupt children’s sleep. With an
independent sample from the one used in the present investigation, marital conflict was related to children’s emotional insecurity about the marital relationship which, in turn, was linked with lower sleep quality and minutes cross-sectionally (El-Sheikh et al., 2007). Further, security in the child-mother, child-father, and marital relationships at W1 predicted actigraphy-based sleep problems 2 years later (Keller & El-Sheikh, 2011).

Consistent with cumulative risk and biopsychosocial models of development emphasizing interactions at multiple levels of functioning, findings suggest, however, that sources of emotional security in the family (e.g., marital conflict) are not lawfully associated with sleep. Rather, the direction and strength of these associations depend on other systems (e.g., PNS) that may provide compensatory regulation and protection, or amplify dysregulation and exacerbate sleep problems in the context of marital conflict. The protective and vulnerability functions of RSA-R are consistent with Polyvagal Theory (Porges, 2007), parallel well replicated evidence with marital conflict and internalizing or externalizing outcomes (El-Sheikh & Erath, 2011), and extend this research to children’s sleep as an outcome. RSA-R, or vagal withdrawal, accelerates heart rate and increases metabolic output rapidly, reflects awareness of environmental challenge and mobilization of physiological resources that support responses to stress, and is a correlate or index of emotion regulation (Porges, 2007). Children with lower levels of RSA-R, as well as poor initial sleep and high exposure to marital conflict, exhibited particularly shorter and poorer sleep quality at W2. Thus, the costs of poor PNS regulation (i.e., lower levels of RSA-R or withdrawal) appear much greater in the context of environmental stress (e.g., marital conflict) and poor bioregulation (e.g., poor initial sleep). Although it was anticipated that a higher level of RSA-R would operate as a protective factor, attenuating the association between marital conflict and sleep problems, marital conflict actually predicted better sleep among children with poor initial sleep and greater RSA-R. However, children with poor initial sleep, higher marital conflict, and higher levels of RSA-R reached—but did not exceed—the number of minutes and quality of sleep at W2 of children with better initial sleep.

Children with better initial sleep continued to exhibit such sleep at W2 even in the context of high marital conflict, thus some children appear to “sleep through” marital conflict. Although we did not examine marital conflict or sleep at earlier ages, results suggest that establishing more optimal sleep by middle childhood may offer some resilience to environmental stress in later childhood. Given the rise in biological and social stress as well as behavioral and emotional problems around the transition to adolescence, this possibility should be further explored with data that spans a wider age range. Further, the finding that for some children sleep does not seem to be affected by family conflict is consistent with stress-sleep models, which
postulate that the link between stress and sleep problems depends on several factors including coping style and the nature of the stressor (e.g., chronic vs. acute) (Sadeh, 1996; Sadeh, Keinan, & Daon, 2004). Thus, assessment of children’s coping style in relation to parental marital conflict and chronicity of exposure to such conflict could explicate relations between family stress and sleep.

Limitations

This study has several limitations. Sleep parameters were significantly associated, which may underlie consistency in the pattern of effects. Nevertheless, examination of various sleep parameters, even if associated, is warranted for explication of children’s sleep in the context of risk. In addition, RSA-R was assessed during a problem-solving challenge. Although RSA-R is somewhat stable in children of the ages examined (El-Sheikh & Erath, 2011), it is possible that physiological responses during this task do not closely reflect physiological responses in the context of marital conflict. At the same time, star tracing is a relatively negative, frustrating task that may generate responses that are at least somewhat comparable to conflict situations.

Future Directions

Future research could extend the present research in several ways. Results were observed in a community sample of children. It will be important to consider whether the pattern of effects may be different for children exposed to more severe marital violence or who suffer from clinically significant sleep disorders. Although two waves of data are a strength of the investigation, three or more waves in future studies would allow a more thorough investigation of developmental effects. Associations among constructs are likely complex and transactional. For example, one mechanism through which children’s sleep may influence family conflict is by way of the effects of sleep problems on children’s emotion dysregulation and behavior problems that, in turn, shape their interactions with parents. In addition, the present study included one index of autonomic nervous system activity (RSA-R). Although difficult to model statistically, simultaneous consideration of multiple autonomic nervous system parameters (e.g., RSA and indices of sympathetic nervous system activity) in future research could yield a better understanding of the development of children’s sleep over time. Finally, future research designed to match adaptive physiological responses with diverse individual characteristics (e.g., gender) and environmental stressors would be informative.
CONCLUSION

The present study provides consistent evidence that marital conflict predicts increased sleep problems among children with initially poor sleep and less vagal withdrawal, whereas better initial sleep or greater vagal withdrawal serve protective functions. These results advance a more nuanced perspective on the effects of marital conflict on children’s sleep over time, implicating bioregulatory systems that can provide protection or increase vulnerability in the context of high marital conflict.

ACKNOWLEDGMENTS

We wish to thank the staff of our research laboratory for data collection and preparation, and the school personnel, children, and parents who participated.

REFERENCES


