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Children's sleep and daytime functioning: Increasing heritability and environmental associations with sibling conflict

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Abstract

Children's sleep has both environmental and genetic influences, with stressful family environmental factors like household chaos and marital conflict associated with sleep duration and quality (El-Sheikh, Buckhalt, Mize, & Acebo, 2006; Fiese, Winter, Sliwinski, & Anbar, 2007). However, it is less clear whether sibling conflict is related to sleep duration and children's sleep problems (e.g., nighttime wakings, parasomnias). In addition, few studies have tested whether associations between sleep and stressful family environmental factors are accounted for by an underlying set of genes or shared and unique environmental factors. Participants were 582 twins with sleep assessed longitudinally at 12, 30 months, and 5 years of age. Sibling conflict was assessed at 5 years. Greater sibling conflict was associated with shorter sleep duration and greater number of total sleep problems, over and above the influence of general household stress and other covariates. The heritability of sleep duration increased with age. Shared environmental factors accounted for the covariance between sibling conflict and sleep duration and total sleep problems. Findings hold promise for interventions, including educating parents about fostering positive sibling relationships and healthy sleep habits.

Keywords

conflict; health; middle childhood; siblings; twins

1 | INTRODUCTION

Ten to 13 h of sleep per night is ideal for young children to attain proper daytime functioning (naps not taken into account; National Sleep Foundation, 2015). Yet, approximately 25% of infants and children experience sleep restriction or normative sleep problems, including poor sleep quality and nighttime wakings (Owens, 2004). Additionally, the sleep-wake cycle is one of the earliest biological processes to stabilize, and early sleep problems may hold implications for later sleep behavior and health (Bruni, 2010; Chaput et al., 2016; Gruber, 2013). Thus, understanding the genetic and environmental influences on various components of sleep in infancy and early childhood is critical, with a particular need to clarify specific environmental mechanisms that contribute to early sleep problems.

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SUPPORTING INFORMATION

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Family-level factors such as household chaos and family conflict have been linked to sleep problems in infancy and childhood (El-Sheikh, Buckhalt, Mize, & Acebo, 2006; Fiese, Winter, Sliwinski, & Anbar, 2007; Sadeh & Anders, 1993), but the sibling relationship is a unique and understudied part of children's early environment that may also be related to early sleep problems. Specifically, sibling conflict is an important predictor of children's later behavioral, social, and emotional adjustment (Bekkhus, Staton, Borge, & Thorpe, 2011; Lockwood, Kitzmann, & Cohen, 2001; Stormshak, Bellanti, & Bierman, 1996), including aspects of temperament (e.g., self-regulation) that are widely associated with sleep problems (Atkinson, Vetere, & Grayson, 1995; Weinraub et al., 2012).

Drawing from a strong history of research supporting the important influences of the home environment more generally on children's sleep (e.g., Fiese et al., 2007), as well as theories highlighting links between family stress, affect regulation, and arousal (Dahl, 1996; Repetti, Taylor, & Seeman, 2002), the current study aimed to extend prior research linking family stress and children's sleep. Specifically, we tested associations between sleep and sibling conflict, an aspect of the children's family environment that has not yet been studied. Furthermore, we also aimed to estimate underlying genetic influences that may account for associations between children's sibling conflict and sleep using the twin design to estimate whether nighttime sleep duration and sleep problems were heritable in infancy and early childhood, and model genetic and environmental contributions to associations between children's sibling conflict and sleep duration and sleep problems.

1.1 | Theoretical framing

The Risky Families Model or Theory suggests that both genetic predispositions and contextual factors (e.g., hostility, high conflict) contribute to family stress and may place children at risk for negative mental and physical health outcomes (Repetti et al., 2002). Child sleep may be one developmental outcome that is particularly sensitive to stress within the family context, as family conflict has been linked to child sleep problems in prior studies (e.g., El-Sheikh et al., 2006; El-Sheikh, Buckhalt, Cummings, & Keller, 2007). Sibling conflict is an understudied family stress factor that may contribute to poor sleep or sleep problems for children. Indeed, one way in which family stressors such as sibling conflict may be associated with restricted sleep or sleep problems for children is through increased cognitive or physiological activity and arousal (Alfano, Pina, Zerr, & Villalta, 2010; Dahl, 1996). Dahl's (1996) model of sleep regulation and arousal (physiological and cognitive) suggests transactional relations between sleep quality, emotion regulation, and arousal regulation, such that family stressors such as sibling conflict potentially increase arousal and impact emotion regulation ability in children, and these factors may lead to restricted sleep or other sleep problems. Concurrently, heightened arousal and decreased emotion regulation due to sleep problems may increase the likelihood of conflict (Dahl, 1996).

Prior research supports links between family stressors, physiological or emotional arousal, and child outcomes like sleep. For example, greater marital conflict has been associated with greater internalizing and externalizing symptoms and poorer cognitive functioning, particularly for girls with greater physiological and emotional arousal (sadness); however, greater marital conflict was related to more internalizing symptoms for boys, with greater

physiological arousal mediating this link (El-Sheikh & Buckhalt, 2005). Related research suggests a pathway from high marital conflict to increased child emotional insecurity and emotional problems, and finally to shorter child sleep duration and poorer sleep quality (El-Sheikh et al., 2007). Finally, increased emotional intensity, lower emotional regulation, and greater cognitive arousal before bedtime has predicted shorter sleep duration and greater sleep disturbances in middle childhood and adolescence (e.g., daytime sleepiness; Alfano et al., 2010; El-Sheikh & Buckhalt, 2005).

1.2 | Psychosocial factors and child sleep

Numerous studies have found associations between stressful family environmental factors and child sleep. General home disorganization and stress have been linked to sleep disruptions and disturbances in childhood and adolescence (e.g., Bartel, Gradisar, & Williamson, 2015; Billows et al., 2009; Gregory, Eley, O'Connor, Rijsdijk, & Plomin, 2005). Additionally, greater family disorganization has been associated with greater sleep onset latency (i.e., time to fall asleep in bed), greater daytime sleepiness, and shorter sleep duration in a sample of adolescents, with sleep hygiene mediating these associations (Billows et al., 2009). Family relationships (e.g., marital, parent–child) are established risk factors for poor sleep and sleep problems in childhood (El-Sheikh et al., 2006, 2007), and family conflict has been also linked with inadequate sleep in childhood (Smaldone, Honig, & Byrne, 2007). Specifically, higher marital or parental conflict has been associated with shorter sleep duration, greater sleepiness, and lower sleep efficiency in a middle childhood sample (El-Sheikh et al., 2006). Parent–child conflict has been linked to child internalizing and externalizing problems through reduced sleep efficiency and longer nighttime waking episodes (Kelly, Marks, & El-Sheikh, 2014), suggesting links between family relationships and child sleep problems may be important for developmental outcomes. Mother–child relationships characterized by higher conflict have also predicted greater child sleep problems in middle and late childhood (Bell & Belsky, 2008).

Although sibling conflict has not been examined in relation to sleep outcomes, it is another stressful family environmental factor that may increase physiological or cognitive arousal and affect child sleep duration and sleep problems. Sibling relationships are a unique and important part of the family and social environment and may be critical for children's outcomes, as they combine the emotional closeness and frequent contact of parent–child relationships with the greater equality and age-similarity of peer relationships. Like peer relationships, sibling relationships are characterized by both cooperation and conflict, and recent theories posit frequent fluctuations between conflict and cooperative events between siblings (Cox, 2010; McHale, Updegraff, & Whiteman, 2012). Thus, findings regarding peer relationships and sleep may extend to the sibling relationship, with at least one study in a large nationally representative sample of adolescents (ages 12 to 15) reporting that fewer positive peer associations and social interactions were related to more adolescent-reported sleep disruptions (Maume, 2013). Unlike friendships with peers, children cannot select out of relationships with siblings, which may make conflict-heavy sibling relationships a more constant sort of stress. Thus, associations between sibling conflict and sleep parameters in childhood should be tested, given links between other types of conflict and social relationships and child and adolescent sleep.

1.3 | Behavioral genetic influences on children's sleep

Although family stressors are often assumed to be related to children's outcomes through environmental pathways, genetic predispositions may contribute to both family stress and child sleep, as well as their association. Previous twin studies examining genetic and environmental influences on normative sleep problems over time have found that these outcomes tend to be moderately to highly heritable, but genetic and environmental contributions to individual sleep parameters vary and may differ by age (Barclay, Gehrman, Gregory, Eaves, & Silberg, 2015; Barclay & Gregory, 2013; Plomin, DeFries, Knopik, & Neiderhiser, 2013). For example, parent-reported sleep duration in young twins was primarily accounted for by additive genetic factors in one study (approximately 71%; Gregory, Rijsdijk, & Eley, 2006), and another study found additive genetics contributed 46% of the variance in parent-reported sleep problems in middle childhood, with parent-reported sleep problems remaining stable across middle childhood (Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009). In addition to genetic influences, sleep duration also has shared environmental influences, with one study showing that the majority of variance in parent-reported sleep duration in infancy and early childhood could be attributed to shared and nonshared environmental factors and the remainder was explained by genetic factors (35%; Brescianini et al., 2011). Furthermore, environmental factors may account for a large part of the covariance between sleep and interpersonal stressors in childhood. For example, parent-reported family chaos explained a significant proportion of the covariance (36%) in the association between sleep problems and anxiety in a genetically informed study of young twins (ages 3 to 4; Gregory et al., 2005).

However, fewer twin studies have examined genetic and environmental contributions to the covariation between psychosocial factors and child sleep, and it is unknown whether sibling conflict and child sleep may be linked for genetic or environmental reasons. Prior behavioral genetic studies have found that sibling conflict shows additive genetic (41%), as well as shared environmental (28%), and nonshared environmental influences (31%; Lemery & Goldsmith, 2003). Such findings have the potential to inform possible points of intervention for improving child sleep and reducing sibling conflict. If environmental factors primarily account for covariation between sibling conflict and sleep indicators, clinicians may be able to inform parents that sibling conflict may be a risk factor for poor sleep behavior (or vice versa), and direct parents regarding possible ways to reduce sibling conflict or introduce changes in children's environment or bedtime routine that promote better sleep.

1.4 | Current study

No studies to date have tested whether sibling conflict is related to child sleep duration or sleep problems. Thus, the first aim was to test whether sibling conflict was associated with sleep duration and total sleep problems, while covarying broader family stress (i.e., household chaos) and demographic factors. We hypothesized that greater sibling conflict would be related to shorter nighttime sleep duration and greater total sleep problems. The second aim was to use the twin design to model genetic and environmental influences on sleep duration at 12, 30 months, and 5 years of age. Specifically, we hypothesized that nighttime sleep duration would be accounted for primarily by additive genetic influences at each age, although we expected shared environmental factors to contribute as well (e.g.,

Gregory et al., 2006, 2009). We also estimated genetic and environmental contributions to sibling conflict and total sleep problems at 5 years, hypothesizing that sibling conflict would be primarily accounted for by additive genetic factors (Lemery & Goldsmith, 2003) and total sleep problems would have primarily additive genetic influence with the remaining variance mostly attributed to shared environmental factors (Gregory et al., 2006). Finally, we examined the association between sibling conflict and child sleep parameters at 5 years using bivariate behavioral genetic models. We had no specific hypotheses regarding the covariance between sibling conflict and child sleep parameters, as these associations have not previously been tested.

2 | METHODS

2.1 | Participants

The Arizona Twin Project (ATP; Lemery-Chalfant, Clifford, McDonald, O'Brien, & Valiente, 2013) includes 291 twin pairs and their primary caregivers (582 individual twins; monozygotic or MZ = 151, same sex dizygotic or DZ = 210, opposite sex dizygotic or DZ = 221) who were studied across three ages: 12, 30 months, and 5 years. The sample was evenly split between males and females at each time point (47–49% male), and is diverse with approximately 55.3% European American, 28.3% Latino, 6.2% Asian American, and 5.4% African American, 1.6% American Indian, and 1% Native Hawaiian families. Most mothers were married (77.8%), with 1.7% separated, 2% divorced, 0.3% widowed, 6% always single, 10.6% in a partnership, and 0.3% reporting "other" family status. At 30 months of age (M = 30.64 months, SD = 0.22), 504 twins and their mothers participated. The 5-year assessment of ATP included 406 twins and their mothers (M = 4.8 years, SD = 0.39). A lower sample size at the 5-year assessment resulted from it being a partial assessment of the full ATP sample due to time and budget constraints.[1] Attrition analyses indicated that there were no differences on family demographics between those who participated at 5 years and those who did not on sex, ethnicity, socioeconomic status (SES; 30 months), and family structure (30 months; ps > .05). In addition, there were no differences in nighttime sleep duration or total sleep problems assessed at 30 months of age between twins who participated at 5 years and those who did not (ps > .05).

2.2 | Procedure

Parents of twins were recruited from state birth records when twins were infants. When the twins were 12 and 30 months old, primary caregivers (>95% mothers) completed telephone or online interviews assessing twin zygosity, demographics, and children's health and development, including sleep duration. When twins were approximately 5 years old, primary caregivers completed a telephone or online interview assessing twin characteristics and home environment, including household chaos, sibling relationships and sleep habits and duration. Participating families were compensated for survey completion at each wave,

[1]For 5-year assessment: 60% European American, 24.1% Latino, 5.9% Asian, 4.4% African American, 1.5% Native Hawaiian, 1% Native American, 2.5% 'Other'. For relationship status of primary caregiver at 5 years: 77.8% married, 1.9% separated, 4.2% divorced, .5% widowed, 4.7% always single, 4.7% in a partnership, .9% 'other' (note: some primary caregivers did not provide family structure at 12, 30 months, and 5 years).

receiving US $40 at 12- and 30-month waves and $15 at the 5-year assessment. At each assessment, all measures and methods were approved by an institutional review board.

2.3 | Measures

2.3.1 | Nighttime sleep duration—Nighttime sleep duration was measured at 12, 30 months, and 5 years. Primary caregivers were asked to report each twins' typical bedtime, wake time, and number of hours and minutes slept at night in a typical week (usually the last week or the most recent "typical" week; Owens, Spirito, & McGuinn, 2000). Nighttime sleep duration was calculated as the total number of hours a child slept during the night on average for a typical week at each age.

2.3.2 | Child sleep habits questionnaire—The Child Sleep Habits Questionnaire (CSHQ) was used at the 5-year assessment and is a 35-item revised parent-report measure of multiple dimensions of sleep including total sleep duration, sleep duration problems, bedtime resistance, sleep latency, nighttime wakings, sleep anxiety, parasomnias, and daytime sleepiness (Owens et al., 2000). Items were rated on a 5-point Likert scale, with higher scores indicating more sleep problems for a specific behavior [e.g., *Twin A* struggles at bedtime (cries, refuses to stay in bed, and so on), *Twin A* wakes up more than once during the night, *Twin A* is restless and moves a lot during their sleep]. A total sleep problem score was computed for each twin by summing scores from all of the items on each of the scales. Items and scales are not typically highly correlated with one another in community samples of children, as individual items and each of the CSHQ scales represent unique sleep disturbances which may not be related so alpha coefficients are not appropriate (Owens et al., 2000).

2.3.3 | Sibling relationship questionnaire—The Sibling Relationship Questionnaire (SRQ) is a parent-report measure that includes a scale measuring sibling conflict (Furman & Buhrmester, 1985), which was completed at 5 years of age using a mean of the five-item conflict scale (Cronbach's $\alpha = 0.87$). Items were rated on a 5-point Likert scale separately for each twin, with higher scores indicating more conflict. Because items assessed conflict individually for each twin (e.g., How much does *Twin A* insult and call *Twin B* names? How much does *Twin B* insult and call *Twin A* names?), we were able to estimate genetic and environmental influences using twin modeling.

2.3.4 | Zygosity—Mothers reported zygosity using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991), a 32-item parent-report measure that differentiates between MZ and DZ twins by using information about pregnancy (e.g., number of amnions) and physical differences between the twins. Parent-reported zygosity is between 93% and 98% accurate in characterizing twin zygosity compared to genotyping, making this questionnaire a valid alternative (Forget-Dubois et al., 2003; Goldsmith, 1991). Any ambiguous twin pairs (could not determine zygosity) were coded as missing, and parent-report of zygosity was supplemented with expert ratings of photos and videos of the twins.

2.3.5 | Covariates—Given prior research showing significant associations with sleep indicators and/or sibling conflict, a number of covariates were examined in all phenotypic

regression models: age (Sadeh, Raviv, & Gruber, 2000), sex (Sadeh et al., 2000), ethnicity (Crosby, LeBourgeois, & Harsh, 2005), family structure (MacKinnon, 1989; Mannering et al., 2011), SES (Dunn, Slomkowski, & Beardsall, 1994; Stein, Mendelsohn, Obermeyer, Amromin, & Benca, 2001), household chaos at 5 years of age (Gregory et al., 2005; Kretschmer & Pike, 2009), and sleep duration at 30 months of age (as earlier sleep is often a strong predictor of later sleep; Lam, Hiscock, & Wake, 2003). We created binary variables for demographic covariates, such that sex was coded 0 = 'Male' and 1 = 'Female', ethnicity was coded 0 = 'European American' and 1 = 'All Other Ethnicities', and family structure was coded 0 = 'Married' and 1 = 'Other Family Structures'. Family SES was a mean composite of standardized mother's education level, father's education level, and total family income before taxes assessed at 5 years. Household chaos at 5 years of age accounted for broader family stress and conflict using the Confusion, Hubbub and Order Scale (CHAOS; Matheny, Wachs, Ludwig, & Phillips, 1995), which assesses the level of confusion and disorder in the children's home environment. A single mean score was derived for both twins (one score per family), with higher scores characterizing a more chaotic, disorganized, and hurried home (Cron-bach's α = 0.76).

For quantitative genetic analyses, the effects of age and sex were regressed out of sibling conflict and sleep indicators and residual scores were saved. All quantitative genetic analyses were tested with the effects of additional significant covariates (ethnicity, family structure, and SES) regressed out (see Supporting Information Tables S1–S3), and variance and covariance estimates were not different from models with only sex and age regressed out. As such, quantitative genetic models with age and sex regressed out are reported, as is standard practice for incorporating covariates into twin models (McGue & Bouchard, 1984).

2.4 | Statistical approach

2.4.1 | Regression analyses—Mixed model regression analyses were conducted in SPSS 22 to account for twin interdependence and examine associations between sibling conflict and child sleep parameters. Predictors in regression analyses (e.g., sibling conflict) were centered at zero and unstandardized beta estimates are reported.

2.4.2 | Behavior genetic analyses—Univariate and bivariate quantitative behavioral genetic models (i.e., ACE models) were conducted using OpenMx (Boker et al., 2011), an R-based program that estimates genetic and environmental variance and covariance using structural equation models with maximum likelihood estimation. The univariate ACE model parses the variance in a single phenotype into latent additive genetic (A), shared environmental (C), and nonshared environmental (E) components on the basis of phenotypic variance and covariance between cotwins. Additive genetic factors (A) are the sum of genetic influences on a trait, shared environmental factors (C) are aspects of environment that make individuals more similar to one another, and nonshared environmental contributions (E) represent aspects of environment that make individuals more distinct or different from one another. Traits are thought to be more genetically influenced if the phenotypic correlation between cotwins is stronger for MZ twins (who share 100% of their segregating DNA) than DZ twins (who share 50% on average). In the univariate ACE model, additive genetic influences on a phenotype are set to correlate 1.0 for MZ twins and 0.5 for

DZ twins. Shared environmental influences are expected to affect MZ and DZ twins to the same degree regardless of genetic relatedness, and are set to correlate 1.0 across MZ and DZ groups. Nonshared environmental variance encompasses all nongenetic factors that reduce phenotypic covariance between cotwins, including measurement error, and is uncorrelated across cotwins.

Univariate ACE models were fit independently for sibling conflict, child nighttime sleep duration, and total sleep problems, and the significance of A and C parameters was tested by systematically dropping them from the model, and comparing the fit of full and reduced models using the −2-log likelihood chi-square test of fit (−2LL). Because the E parameter contains measurement error, it was not dropped from any model. Akaike's Information Criterion (AIC; Akaike, 1974), which penalizes models with a larger number of parameters, was used to assess model fit. Lower AIC values indicate better model fit.

The bivariate Cholesky decomposition was used to decompose the covariance between two traits into A, C, and E components (see Figure 1) by examining cross-twin cross-trait covariances separately for MZ and DZ twins. In the current study, two bivariate Cholesky decompositions were fitted to examine the covariance between (a) sibling conflict and nighttime sleep duration, and (b) sibling conflict and total sleep problems. Genetic and environmental correlations were also conducted to show the extent to which additive genetic, shared environmental or nonshared environmental influences on sibling conflict are correlated with additive genetic, shared environmental or nonshared environmental influences on sleep parameters.

3 | RESULTS

3.1 | Preliminary analyses

Descriptive statistics, zero-order correlations, and twin intra-class correlations are summarized in Table 1. All variables were normally distributed and did not require linear or logarithmic transformation. No variable exceeded the recommended cutoff for positive or negative skew (2.00) or kurtosis (7.00; Muthén & Kaplan, 1985). Nighttime sleep duration was positively correlated across all three time points. Sibling conflict at 5 years was negatively correlated with nighttime sleep duration at 12, 30 months, and 5 years, and positively correlated with concurrent total sleep problems. MZ twins were more similar than DZ twins on nighttime sleep duration at 12 and 30 months, as well as on total sleep problems and sibling conflict at 5 years, suggesting a role for additive genetic influences.

3.2 | Aim 1: Multiple regression analyses

Mixed model regressions for sibling conflict predicting total child sleep problems and change in nighttime sleep duration at 5 years are summarized in Table 2. Nighttime sleep duration was stable across time (from 30 months to 5 years), and greater sibling conflict was associated with greater declines in concurrent nighttime sleep duration, after accounting for other covariates and demographic variables in each model (Table 2). Greater sibling conflict was also associated with more total sleep problems (Table 2). In the same model, longer nighttime sleep duration at 30 months of age was associated with fewer total sleep problems

whereas greater household chaos at 5 years of age was concurrently associated with more total sleep problems (Table 2). Children in families with parents who were not married (e.g., divorced, in a partnership, single, widowed) demonstrated more total sleep problems.

3.3 | Aim 2: Heritability of sleep duration and daytime sleepiness

3.3.1 | Nighttime sleep duration and total sleep problems—Fit statistics for univariate ACE models and standardized variance components are summarized in Table 3. For nighttime sleep duration at 12 months, the full ACE model yielded the best fit, with the greatest proportion of variance accounted for by shared environmental factors (C = 0.81). At 30 months, the CE model yielded the best fit for nighttime sleep duration, with the greatest proportion of the variance again accounted for by the shared environmental factor (C = 0.83). The full ACE model yielded the best fit for nighttime sleep duration at 5 years, with the greatest proportion of the variance was accounted for by the shared environmental factor (C = 0.60), with moderate heritability (A = 0.36). The full ACE model yielded the best fit for total child sleep problems at 5 years of age, which was somewhat heritable (A = 0.28), with significant shared environmental contributions (C = 0.65).

3.3.2 | Sibling conflict at 5 years—The full ACE model fit the data best for sibling conflict (see Table 3 for fit statistics and standardized variance components), with the greatest proportion of the variance accounted for by the shared environment (C = 0.86).

3.4 | Aim 3: Bivariate ACE models

3.4.1 | Sibling conflict and nighttime sleep duration at 5 years—A bivariate Cholesky decomposition of sibling conflict and nighttime sleep duration revealed the full ACE–ACE model to be the best fitting model, after dropping the A and E contributions to the covariance between the two phenotypes (see Table 4 for fit statistics and Table 5 for standardized variance components). Sibling conflict was primarily influenced by the shared environment (C11 = 0.58), with the remaining variance divided between additive genetic (A11 = 0.22) and nonshared environmental contributions (E11 = 0.20). The covariance between sibling conflict and nighttime sleep duration was fully accounted for by shared environmental factors, explaining 7% of the total variance in nighttime sleep duration. After accounting for sibling conflict, the variance in nighttime sleep duration was accounted for by shared environmental (C22 = 0.51) and additive genetic factors (A22 = 0.38), with little contribution of the nonshared environment (E22 = 0.04). Genetic and environmental correlations showed that shared environmental influences on sibling conflict were correlated with shared environmental influences on nighttime sleep duration at 0.33.

3.4.2 | Sibling conflict and child sleep problems at 5 years—For sibling conflict and total child sleep problems, a bivariate ACE–ACE model dropping the A and E contributions to covariance between the two phenotypes provided the best fit (see Table 4 for fit statistics and Table 5 for standardized parameter estimates). Most of the variance in sibling conflict was accounted for by the shared environment (C11 = 0.86), with the remaining variance in sibling conflict accounted for by additive genetic factors (A11 = 0.09) and nonshared environmental contributions (E11 = 0.05). The covariance between sibling conflict and total sleep problems was accounted for entirely by shared environmental factors,

explaining about 7% of the total variance in total sleep problems. After accounting for sibling conflict, total child sleep problems were accounted for by additive genetic influences (A22 = 0.30), shared environmental (C22 = 0.55), and nonshared environmental contributions (E22 = 0.07). Genetic and environmental correlations showed that shared environmental influences on sibling conflict were correlated with shared environmental influences on total sleep problems at 0.34.

4 | DISCUSSION

The current study aimed to test whether sibling conflict was linked with children's nighttime sleep duration and total sleep problems, and to examine genetic and environmental influences on associations between sibling conflict and child sleep. Sibling conflict was related to child sleep duration and sleep problems over and above the impact of family stressors (e.g., household chaos), and household chaos was also a strong predictor of child total sleep problems. Sleep duration was relatively stable across infancy and toddlerhood, and showed moderate heritability at 12 months and 5 years of age. Although additive genetic factors independently contributed to some of the variation in sibling conflict, nighttime sleep duration, and sleep problems, shared environmental factors fully explained the covariance between sibling conflict and sleep parameters.

4.1 | Sibling conflict and sleep problems

Sibling conflict (at 5 years) was associated with greater declines in concurrent nighttime sleep duration from 30 months to 5 years and more sleep problems at 5 years over and above the effect of concurrent household chaos and covariates. These findings add to the current literature by establishing links between sibling relationships and child sleep, and extend past studies that have shown that conflict and negative sibling relations are linked to poor emotional and behavioral outcomes in childhood (e.g., Bekkhus et al., 2011; Lockwood et al., 2001). Greater household chaos was related to greater total sleep problems, although it was not related to concurrent nighttime sleep duration. This suggests that child sleep duration and sleep problems are independently linked with both proximal stressors in the home (i.e., sibling conflict), as well as broader family stressors like household chaos. Given other research demonstrating bidirectional associations between family stressors and child sleep (e.g., Bell & Belsky, 2008), future studies should test bidirectional associations between sibling conflict and child sleep using closely spaced measurement to clarify direction of effects.

4.2 | Heritability of child sleep parameters and sibling conflict

Similar to previous findings (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010; Gregory et al., 2006), we found genetic and shared environmental influences on sleep duration and sleep problems. Nighttime sleep duration showed primarily shared environmental influences at 12 and 30 months, and a moderate additive genetic contribution at 5 years. The greatest proportion of the variance in sleep problems was accounted for by shared environmental factors, with most of the remaining variance accounted for by additive genetic factors. Finally, we found that the greatest proportion of the variance in sibling conflict at 5 years was accounted for by shared environmental factors, with additive genetic factors also

playing a role. These behavioral genetic findings differ slightly from previous empirical work (e.g., Lemery & Goldsmith, 2003), although prior work showing considerable additive genetic influences on sibling conflict was conducted with a wider age range of children (ages 3–8) and a less diverse sample (96% European American; Lemery & Goldsmith, 2003), which may explain why we did not find larger genetic influence on sibling conflict that these previous studies report. Furthermore, the large shared environmental influences we detected may be explained by reporter bias, as estimates of sleep and sibling conflict were based on primary caregiver reports. For example, Nixon et al. (2008) found that there was almost an hour difference between actigraphy reports of sleep duration and parent-reported sleep duration via diaries, such that parent reports significantly overestimated sleep duration. Martinez et al. (2014) showed similar findings, with parent reports of sleep duration overestimating sleep duration by about 20 min compared to actigraphy. As such, shared environmental estimates for sleep and sibling conflict may be inflated in the current study, and future studies should aim to capture objective sleep and sibling conflict (e.g., actigraphy, video recordings) in addition to parent reports of child sleep and conflict.

Overall, additive genetic factors influenced sleep problems and significantly contributed to nighttime sleep duration, with the heritability of sleep duration slightly changing across ages. This may be less surprising given that studies report smaller heritability estimates of sleep duration in infancy and young childhood (see Brescianini et al., 2011; sample age = 1.5 years) whereas studies examining sleep duration in middle and late childhood suggest moderate to strong heritability (age 8; Gregory et al., 2006). Our finding of greater heritability in sleep duration at 5 years is consistent with other research often finding increases in the heritability of other traits with age, such as temperament, intelligence quotient (IQ), and weight (see Plomin et al., 2013).

4.3 | Environmental influences on associations between sibling conflict and sleep

The associations between sibling conflict and sleep indicators were solely explained by shared environmental factors, suggesting that sibling conflict may impact children's sleep duration and the number of sleep disturbances children experience for environmental reasons. As noted, some research shows that other types of family conflict (i.e., marital, parent–child) and less positive peer relationships are associated with inadequate sleep and increased sleep problems in childhood (Bell & Belsky, 2008; Kelly et al., 2014; Maume, 2013).

Furthermore, individual or environmental stressors such as sibling conflict can lead to increased cognitive or physiological activity and arousal, which may disrupt sleep for children. As such, cognitive and physiological arousal may be one mechanism that accounts for the association between sibling conflict and child sleep duration and sleep problems (Alfano et al., 2010). Sibling conflict (or other stressors in the home environment) may increase physiological or cognitive and affective arousal, which in turn could interfere with nighttime sleep duration, leading to more dysregulated sleep and increases in overall sleep problems. Links between sibling conflict and child sleep are also likely bidirectional, and heightened arousal and decreased emotion regulation due to restricted sleep and sleep problems may increase sibling conflict. Some research supports these associations and

pathways, with studies showing that greater physiological and emotional arousal may moderate and mediate associations between family stressors and conflict like marital or parental conflict and child sleep and other developmental outcomes (e.g., internalizing, externalizing, cognitive problems; El-Sheikh & Buckhalt, 2005; El-Sheikh et al., 2006), as well as directly predict shorter sleep duration and more sleep problems in middle childhood and adolescence (Alfano et al., 2010; El-Sheikh & Buckhalt, 2005). These studies highlight how and why family stress factors like sibling conflict may be related to childhood sleep duration and sleep problems, and future studies and analyses should aim to test physiological and affective arousal as a mechanism connecting sibling conflict to child sleep outcomes, including how sibling conflict and sleep may influence one another over time.

4.4 | Study limitations

Despite a number of strengths, including using a population-based sample of twins measured longitudinally, this study also has several limitations. First, we did not assess total sleep problems and sibling conflict at every age. In future studies, the same or similar sleep parameters and sibling conflict should be measured at multiple time points to more clearly test bidirectional associations. Second, only subjective reports of sleep parameters and sibling conflict were used, which may have biased estimates of sleep and conflict and inflated shared environmental variance. Future studies should adopt a multimethod approach to sleep measurement including objective measurement (i.e., actigraphy or polysomnography) as well as parent- or self-report, depending on the age of the child (Gregory et al., 2011; Shochat, Cohen-Zion, & Tzischinsky, 2014). Finally, the twin method assumes that twins are representative of the general population; this assumption is supported for other outcomes, such as internalizing and externalizing symptoms in early and middle childhood (Plomin et al., 2013; Robbers et al., 2009), but has not yet been tested for children's sleep. Additionally, the twin method assumes MZ and DZ twin pairs do not differ on their similarity for trait-relevant environmental factors. Despite these assumptions, the twin design is still the best way to estimate both genetic and environmental contributions to trait variation when the estimates are interpreted in a broad context or at a population level (e.g., Gregory et al., 2009).

5 | CONCLUSION

The current study aimed to understand whether there were phenotypic and quantitative behavioral genetic associations between sibling conflict and child sleep duration and sleep problems, as well as examine additive genetic, shared environmental, and nonshared environmental contributions to sibling conflict, nighttime sleep duration and sleep problems in a longitudinal, population-based sample of twins. We found associations between greater sibling conflict and shorter child nighttime sleep duration and more sleep problems, and greater household chaos was also associated with greater child sleep problems. Although we found both childhood sleep duration and sleep problems to have moderate genetic influence, it was shared environmental influences that solely accounted for the covariance between sibling conflict and both nighttime sleep duration and sleep problems, with shared environmental influences on sibling conflict showing moderate positive correlations with shared environmental influences on sleep indicators. These findings suggest that sibling

conflict may be an important factor in children's environment that impacts nighttime sleep duration and total child sleep problems, and sibling relationships should be considered when designing family-based interventions for children's sleep. Future studies should aim to test person- and family-level factors that may serve as moderators or mediators of the association between sibling conflict and child sleep behavior in hopes that identifying such mechanisms will better inform parents, health practitioners and researchers regarding the most effective ways to improve children's sleep and other developmental outcomes.

Findings may help inform public policy and health practitioners by providing parents information about individual specific, modifiable factors that may negatively impact children's sleep patterns. Given our findings, it may be useful to educate parents that sibling conflict may be a risk factor for poor sleep behavior, and inform parents about possible techniques or ways to reduce sibling conflict. Alternately, depending on the direction of effects, changes in children's environment that promote better sleep may also be effective in reducing sibling conflict. Overall, findings hold promise for sleep and sibling interaction interventions, as well as for future studies that aim to understand the nuances of relations between family relationships and children's sleep.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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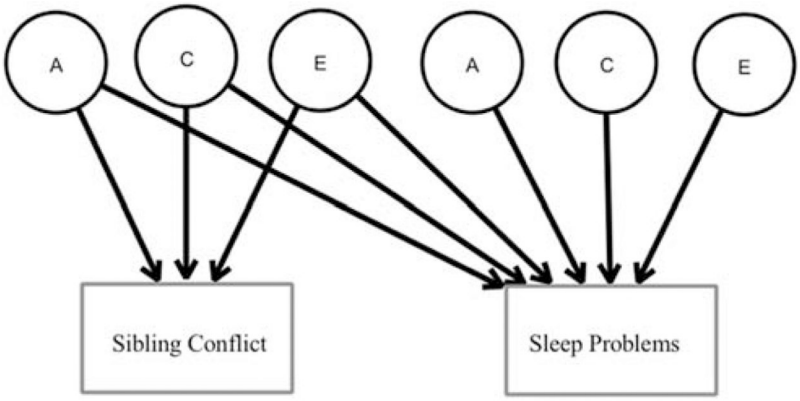

FIGURE 1.

Example bivariate ACE model for associations between sibling conflict and total sleep problems for a single twin. Full bivariate models will estimate associations between sibling conflict and sleep indicators for each twin, as well as their covariances

TABLE 1

Zero-order correlations, descriptive statistics, and twin intra-class correlations

	1	2	3	4	5	MZ	DZ_{ss}	DZ_{os}
1. Nighttime sleep duration (12 months)	-					0.96	0.92	0.87
2. Nighttime sleep duration (30 months)	0.41**	-				0.86	0.87	0.85
3. Nighttime sleep duration (5 years)	0.16*	0.38**	-			0.95	0.79	0.80
4. Total sleep problems (5 years)	−0.11	−0.23**	−0.29***	-		0.94	0.82	0.75
5. Sibling conflict (5 years)	−0.18*	−0.16*	−0.20**	0.22**	-	0.84	0.61	0.78
Mean	10.15	10.41	10.25	54.79	2.59			
Standard deviation	1.50	1.14	1.03	14.60	0.82			
Minimum	5.00	6.00	6.75	7.00	1.00			
Maximum	12.00	12.00	12.50	99.00	5.00			
Skewness	−1.04	−0.94	−0.50	−0.43	0.33			
Kurtosis	1.13	0.74	0.25	1.68	−0.09			

Note. Nighttime sleep duration is measured in hours at each assessment. Total sleep problems is a sum of scores from all items in the Child Sleep Habits Questionnaire (Owen et al., 2001). MZ = monozygotic (identical twins) and includes all male and female monozygotic twin sets; DZ_{SS} = same sex dizygotic (fraternal) twins and includes all male and female same sex dizygotic twin sets; DZ_{OS} = opposite sex dizygotic twins and includes all sets of dizygotic male-female twins.

* $p < .05$;

** $p < .01$;

*** $p < .001$.

TABLE 2

Sibling conflict predicting nighttime sleep duration and total child sleep problems in mixed model regressions

Model predictors	Nighttime sleep duration (h) Est (SE)	Total sleep problems Est (SE)
Constant	12.03 (1.08)**	51.98 (15.71)**
Sex	−.10 (0.08)	−0.40 (1.18)
Age (5 years)	0.31 (0.22)	0.53 (3.2)
SES (5years)	0.15 (0.11)	0.49 (1.56)
European American	0.25 (0.17)	−1.43 (2.4)
Family structure (5 years)	0.30 (0.22)	**6.48 (3.14)***
Nighttime sleep duration (30 months)	**0.16 (0.06)****	**−2.60 (0.80)*****
Household chaos (5 years)	−0.06 (0.34)	**14.20 (5.04)****
Sibling conflict (5 years)	**−0.17 (0.08)***	**2.29 (1.17)***

Note. Models with nighttime sleep duration and total sleep problems were run independently. Nighttime sleep duration and total child sleep problems were both assessed at 5-year assessment. European American (race/ethnicity) was coded such that 0 = 'European American' and 1 = 'All Other Ethnicities'. Family structure was coded such that 0 = 'Married' and 1 = 'Other Family Structures'. Est. = partial regression coefficient estimate. *SE* = robust standard error.

* $p < .05$;

** $p < .01$;

*** $p < .001$. Bolded estimates denote significant values.

TABLE 3

Univariate ACE model fit statistics and estimates for sibling conflict and childhood sleep duration and sleep problems

Scale	Model	−2LL	*df*	AIC	Δ *df*	Δ −2LL	*p*	A	C	E
Nighttime sleep	**ACE**	**1430.49**	**534**	**362.49**	-	-	-	**0.15 (0.100.19)**	**0.81 (0.67–0.99)**	**0.04 (0.02–0.04)**
duration (12 months)	AE	1579.96	535	509.96	1	149.46	<.001			
	CE	1457.25	535	387.25	1	26.79	<.001			
	E	1939.94	536	867.94	2	509.45	<.001			
Nighttime sleep	ACE	1002.8	413	176.8	-	-	-	0.05 **(0.00–0.27)**	0.80 (0.61–1.00)	0.15 **(0.10–0.22)**
duration (30 months)	AE	1075.78	414	247.78	1	72.98	<.001			
	CE	**1003.29**	**414**	**175.29**	**1**	**0.49**	**0.48**	-	**0.83 (0.67–1.01)**	**0.17 (0.14–0.20)**
	E	1243.61	415	413.61	2	240.81	<.001			
Nighttime sleep	**ACE**	**836.24**	**372**	**92.24**	-	-	-	**0.36 (0.26–0.47)**	**0.60 (0.43–0.82)**	**0.04 (0.02–0.05)**
duration (5 years)	AE	877.35	373	131.35	1	41.11	<.001			
	CE	872.91	373	126.91	1	36.67	<.001			
	E	1085.98	374	337.98	2	249.74	<.001			
Sleep problems	**ACE**	**2900.8**	**377**	**2146.8**	-	-	-	**0.28 (0.18–0.41)**	**0.65 (0.46–0.86)**	**0.07 (0.04–0.10)**
(5 years)	AE	2946.04	378	2190.04	1	45.24	<.001			
	CE	2919.36	378	2163.36	1	18.56	<.001			
	E	3136.72	379	2378.72	2	235.92	<.001			
Sibling conflict	**ACE**	**690.35**	**377**	**−63.35**	-	-	-	**0.09 (0.04–0.45)**	**0.86 (0.42–0.85)**	**0.05 (0.04–0.45)**
[5 years)	AE	718.79	378	−37.21	1	28.44	<.001			
	CE	693.17	378	−62.83	1	2.82	0.09			
	E	841.43	379	82.43	2	151.07	<.001			

Note. A = additive genetic components, C = shared environmental component, and E = nonshared environmental component. Bolded models denote the best fitting model. The −2LL is the chi-squared measure of model fit, and the AIC is the Akaike's Information Criterion. Δ *df* and Δ x^2 shows the change in the degrees of freedom and in chi-squared values (respectively) when dropping model parameters. *p* denotes the *p*-value level of significance for the chi-squared test. A, C, and E are standardized variance components or estimates according to the total variance for that phenotype. Variance-based confidence intervals are presented in parentheses, and are based on standardized path estimates.

TABLE 4

Bivariate Cholesky Model fit statistics for two bivariate models, including sibling conflict at 5 years with concurrent nighttime sleep duration and total sleep problems

Scales	Model	−2LL	*df*	AIC	Δ *df*	Δ −2LL	*p*
Sibling conflict and nighttime sleep duration	ACE-ACE	1342.85	673	−3.15	-	-	-
	ACE-ACE*	**1344.63**	**675**	**−5.37**	**2**	**1.78**	**0.41**
Sibling conflict and total sleep problems	ACE-ACE	3511.79	775	1961.79	-	-	-
	ACE-ACE*	3513.97	**777**	**1959.97**	**2**	**2.19**	**0.34**

Note. A = additive genetic components, C = shared environmental component, and E = nonshared environmental component. Bolded models denote the best fitting models. The asterisk (*) denotes that A and E paths were also dropped on the covariance between the two phenotypes. The −2LL is the chi-squared measure of model fit, and the AIC is the Akaike's Information Criterion, which is an additional measure of model fit. Δ*df* shows the change in the degrees of freedom, which occurs when model parameters are dropped. Δx^2 is the change in chi-squared values when dropping model parameters. *p* denotes the *p*-value level of significance for the chi-squared test.

TABLE 5

Bivariate Cholesky decompositions for two bivariate models, including sibling conflict at 5 years with concurrent nighttime sleep duration and total sleep problems

Scales	Model	A11	C11	E11			
Sibling conflict andnighttime sleep duration	ACE-ACE	0.22 (0.07–0.50)	0.58 (0.36–0.83)	0.20 (0.12–0.28)			
		A21 0.00 (0.00–0.00)	C21** 0.06 (0.01–0.20)	E21 0.001 (0.00–0.01)	A22 0.38 (0.27–0.52)	C22 0.51 (0.32–0.74)	E22 0.04 (0.03–0.06)
		A11	**C11**	**E11**			
	ACE-ACE*	**0.22 (0.05–0.50)**	**0.58 (0.37–0.85)**	**0.20 (0.13–0.29)**			
		A21 0.00 (0.00–0.00)	**C21** ** **0.07 (0.01–0.19)**	**E21 0.00 (0.00–0.00)**	**A22 0.38 (0.27–0.51)**	**C22 0.51 (0.33–0.74)**	**E22 0.04 (0.03–0.06)**
		A11	C11	E11			
Sibling conflict andtotal sleep problems	ACE-ACE	0.09 (0.04–0.16)	0.86 (0.68–1.06)	0.05 (0.03–0.07)			
		A21 0.00 (0.00–0.00)	C21** 0.07 (0.01–0.15)	E21 0.001 (0.00–0.02)	A22 0.30 (0.20–0.44)	C22 0.55 (0.38–0.76)	E22 0.07 (0.05–0.11)
	ACE-ACE*	**A11 0.09 (0.04–0.16)**	**C11 0.86 (0.68–1.06)**	**E11 0.05 (0.03–0.07)**			
		A21 0.00 (0.00–0.00)	**C21** ** **0.07 (0.01–0.15)**	**E21 0.00 (0.00–0.02)**	**A22 0.30 (0.20–0.44)**	**C22 0.55 (0.38–0.76)**	**E22 0.07 (0.05–0.11)**

Note. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates.

*A21 and E21 paths were dropped in models.

**CIs for C21 paths in the full and reduced models correspond with the percent of the variance in the second phenotype (7% for sleep duration and 7% for total sleep problems) accounted for by the first phenotype (sibling conflict).