Genetic and Environmental Influences on Parenting, Sibling Conflict, and Childhood Sleep in Five-Year-Old Twins

by

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ABSTRACT

Understanding how interpersonal relationships, such as parenting and sibling relationships, may contribute to early sleep development is important, as early sleep dysregulation has been shown to impact later sleep behavior (Sadeh & Anders, 1993), as well as cognitive and behavioral functioning (Gregory et al., 2006; Soffer-Dudek et al., 2011). In addition, twin studies provide an optimal opportunity to estimate genetic and environmental contributions to parenting, sibling relationships and child sleep, as they are influenced by both genetic and contextual factors. As such, the current thesis examined whether parental punitive discipline and sibling conflict were associated with child sleep duration, dysregulation and daytime sleepiness at 12 months, 30 months, and five years in a longitudinal sample of young twins recruited through birth records (Lemery-Chalfant et al., 2013). Mixed model regression analyses and quantitative behavioral genetic models (univariate and bivariate) were conducted to explore bidirectional relations and estimate genetic and environmental contributions to parental punitive punishment, sibling conflict and child sleep parameters. Sleep duration and dysregulation showed stability over time. Parental punitive discipline did not predict concurrent or future sleep parameters, nor were there bidirectional relations between punitive discipline and child sleep behaviors. Greater sibling conflict at five years was associated with shorter concurrent child sleep duration and greater daytime sleepiness, suggesting that sibling conflict may be a critical interpersonal stressor that negatively impacts child sleep. Shared environmental factors also accounted for the greatest proportion of the covariance between sibling conflict and sleep duration and daytime sleepiness at five years. These findings hold promise for sleep and sibling interaction interventions, including educating parents about fostering positive

sibling relations and teaching caregivers to utilize specific parenting behaviors that may encourage better child sleep behaviors (e.g., establishing bedtime routines). Future studies should aim to understand the nuances of associations between family relationships (like punitive discipline and sibling conflict) and child sleep, as well as other explore person- and family-level factors, such as child negative emotions and parenting, that may influence associations between family relationships and child sleep.

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Introduction

As children approach preschool age, it is estimated that 10 to 13 hours of sleep per night is ideal for children to attain proper daytime functioning (naps not taken into account; National Sleep Foundation, 2015). Despite these recommendations, approximately 25% of infants and children universally experience sleep problems at some point in their lives (Owens, 2004), suggesting that a number of children may experience significant sleep problems beginning in infancy and early childhood. Thus, understanding psychosocial processes that may contribute to early sleep development is critical, as the sleep-wake cycle is one of the earliest biological processes to stabilize, and early sleep dysregulation may hold implications for later sleep behavior (Bruni, 2010; Gruber, 2013; Sadeh & Anders, 1993) and competence across a broad array of domains, such as cognitive and behavioral functioning (Bates, Viken, Alexander, Beyers, & Stockton, 2002; Gregory, Rijsdijk, & Eley, 2006; Soffer-Dudek, Sadeh, Dahl, & Rosenblat-Stein, 2011). Furthermore, understanding how genetic and environmental factors may contribute to sleep development, as well as how sleep behavior impacts mental and physical health outcomes in childhood, may better inform pediatric doctors and clinical interventions regarding child sleep. For example, state and national programs providing information regarding child sleep may be able to target and teach specific parenting practices that foster optimal nighttime sleep duration or sleep regulation for children.

Prior research suggests that increased household chaos, parental negative mood, and marital conflict all significantly contribute to sleep problems in infancy and childhood (El-Sheikh, Buckhalt, Mize, & Acebo, 2006; Fiese, Winter, Sliwinski, &

Anbar, 2007; Sadeh & Anders, 1993). However, little research has examined whether other important familial psychosocial factors, such as parenting practices and sibling relationships, also contribute to sleep outcomes in childhood. For example, parent-child conflict (one aspect of parenting) predicted increased nighttime waking and poorer sleep efficiency in middle childhood (Kelly, Marks, & El-Sheikh, 2014). In addition, parental over-involvement (e.g., anticipating child night wakings, waking child for feedings) early in infancy and childhood was associated with more sleep dysregulation and shorter sleep duration (Iacovou & Sevilla, 2012; Sheridan et al., 2013). However, no studies to date have explored the specific influences of punitive parenting and sibling conflict on child sleep behavior. Punitive parenting and sibling conflict may act as underlying mechanisms leading to shorter child nighttime sleep duration, sleep dysregulation, and greater daytime sleepiness, in that punitive parenting practices and sibling conflict may serve as proximal (and potentially chronic) stressors for a child that disrupt nighttime sleep and lead to poor sleep behavior. Thus, I explored whether punitive parenting and sibling conflict impacted child nighttime sleep duration, sleep dysregulation, and daytime sleepiness in the current study.

Beyond basic relations between parenting, sibling relationships and child sleep, researchers can utilize quantitative behavioral genetics (e.g. using twin designs) to understand genetic and environmental contributions to punitive parenting, sibling conflict, and child sleep, as they are influenced by both genetic and contextual factors throughout the lifespan (Vitaro, Brendgen, & Arseneault, 2009). As such, the twin design helps elucidate genetic and environmental factors that influence each variable *independently* (punitive parenting, sibling conflict, and child sleep), as well as genetic

and environmental contributions to the *associations among variables* (e.g., covariance between punitive parenting and child sleep; Osbourne & George, 1959; Purcell & Koenen, 2005; Vitaro et al., 2009). Furthermore, twin studies can demonstrate changes in genetic and environmental contributions concurrently and over time using sophisticated statistical techniques (e.g. genetically informed cross-lag models). Current theorists have called for longitudinal twin study designs that may clarify fluctuations in genetic and contextual contributions across sensitive developmental periods like infancy and toddlerhood (Barclay & Gregory, 2013; Vitaro et al., 2009). The current study examined child sleep as a biological process in five-year-old twins as they transitioned to kindergarten. The school transition may be a sensitive period that impacts interpersonal relationships (parent-child and sibling), as well as sleep patterns, as bedtime, wake time, and nap schedules may shift with the onset of school (Eccles et al., 1993). Indeed, one study demonstrated that the transition to kindergarten was associated with shorter objective sleep duration and earlier bedtimes, but only for children who did not attend preschool before entering kindergarten (no effects found for children who attended preschool prior to kindergarten; Cairns & Harsh, 2014). Another recent study of sleep across the transition to kindergarten found both earlier wake times and shorter sleep duration after beginning kindergarten compared to pre-kindergarten wake times and sleep duration (Berger, 2015). Furthermore, studies have shown that transitioning to early school start times and school-mandated hours interferes with *adolescent* sleep schedules and biological sleep rhythms, leading to poor sleep behavior (e.g., Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998).

Given limited research regarding associations between parenting practices, sibling relationships, and early childhood sleep outcomes, the current study examined whether punitive parenting practices were associated with sleep behavior (nighttime duration, dysregulation, and daytime sleepiness) in preschool-age twins both concurrently and bidirectionally. I also explored whether sibling conflict was associated with sleep behavior across early childhood, acting as a risk factor for short nighttime sleep duration, greater sleep dysregulation, and greater daytime sleepiness (concurrent and bidirectional relations). Finally, I examined phenotypic as well as quantitative behavioral genetic associations between interpersonal family relationships (punitive parenting and sibling conflict) and child sleep parameters. As such, I used univariate quantitative behavioral genetic models to examine additive genetic, shared environmental, and non-shared environmental contributions to punitive parenting, sibling conflict, and child nighttime sleep duration, sleep dysregulation and daytime sleepiness at various points in early childhood. I also conducted bivariate quantitative behavioral genetic models to explore the additive genetic, shared environmental, and non-shared environmental contributions to the associations between punitive parenting and sibling conflict, and child nighttime sleep duration, sleep dysregulation and daytime sleepiness at 12 months, 30 months, and five years of age.

Overview of Current Thesis. First, theoretical models relevant to children's sleep, parenting, sibling relationships, and current behavioral genetic methods and models are reviewed. Following theoretical contributions in each section, I provide a review of literature regarding child sleep, parenting, sibling relations, and behavioral genetic analyses of child sleep, parenting, and sibling relationships. Next, I provide in-

depth information regarding the current study's hypotheses, sample and methodology, and analyses. Finally, I discuss the meaning of the findings and the implications they have for public policy and health practitioners.

Child Sleep

One specific biological regulatory process that may be related to various mental and physical health outcomes is the sleep-wake cycle. The sleep-wake cycle is one of the earliest biological rhythms to develop and regulate, making sleep in childhood an important consideration for child outcomes (Sadeh, Raviv, & Gruber, 2000). Research has consistently shown that sleep disruption in childhood is a risk factor for future outcomes; specifically, sleep dysregulation in children has been associated with a range of emotional and behavioral regulation problems (e.g., Bates et al., 2002).

Early sleep development and regulation. Development of the sleep-wake cycle begins in utero and continues to develop immediately after birth (Rosen, 2008). In addition, evolution of the sleep-wake cycle coincides with other biological growth processes, including rapid development of the brain and central nervous system (Peirano, Algarin, & Uauy, 2003). Infants also begin exhibiting spontaneous activity that corresponds with growth of these biological processes, contributing to periods of waking (movement) and sleeping for infants (Peirano et al., 2003; Pouthas, Provasi, & Droit, 1996). Thus, simultaneous ontogeny of multiple biological and physical processes occurs, with autonomic brain processes, CNS reactivity, physical activity, and the circadian (sleep) processes all interacting to form the sleep-wake cycle (Peirano et al., 2003).

As the sleep-wake cycle forms, infants begin showing greater nighttime sleep duration and longer periods of wakefulness throughout the day (Peirano et al., 2003). In

particular, sleep-wake cycle formation is aided by exposure to specific periods of light and darkness throughout the day, which reinforces sleep and wake schedules (Borbely & Achemann, 1999; Peirano et al., 2003). Formation of the sleep-wake cycle also encourages synchronization of biological and physical processes, allowing sleep consolidation and regulation to stabilize. Sleep consolidation is the infants' ability to sleep through the night, whereas sleep regulation is the ability to successfully shift from periods of waking to sleeping (Sadeh & Anders, 1993). Sleep consolidation and regulation are critical for early sleep duration, as research has suggested that lack of sleep consolidation may perpetuate sleep problems through early childhood and adolescence (Sadeh & Anders, 1993). Additionally, the inability to regulate sleep likely leads to frequent sleep disruption and shorter sleep duration, which has been associated with reduced cognitive functioning, trouble with social interaction, and emotional-regulation problems later in childhood (Bates et al., 2002; Soffer-Dudek et al., 2011).

As infants transition to toddlerhood, it is estimated that 11 to 14 hours of sleep a day is ideal (National Sleep Foundation, 2015). Preschool-age children also require similar amounts of sleep; studies have estimated 10 to 13 hours are necessary for proper daytime functioning (National Sleep Foundation, 2015). The sleep-wake cycle is typically firmly established by five years of age (Waterhouse, Fukuda, & Morita, 2012). This stabilization co-occurs with school entry for children in most countries; thus, the transition to school may serve as a sensitive period for sleep development, with children moving from more napping throughout the day to fewer naps and increased sleep consolidation at night. Overall, sleep development continues from infancy through childhood, and various environmental factors (e.g., transitioning to school, family

relationships) may change the course of sleep development, impacting child sleep duration, sleep regulation, and daytime sleepiness levels.

Heritability of children's sleep. Beyond phenotypic changes in children's sleep, it is critical to understand whether there are specific additive genetic, shared environmental, and non-shared environmental factors contributing to various child sleep parameters. In regards to heritability estimates of child sleep behavior, electroencephalogram (EEG) studies comparing monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twins have indicated that a significant proportion of various aspects of sleep architecture (non-REM sleep, slow wave sleep, etc.) is accounted for by genetic factors in infancy (Gould, Austin & Cook, 1978) and young adulthood (Ambrosius, Lietzenmaier, Wehrle, Wichniak, & Kalus, 2008; Linkowski, 1999). Likewise, other studies have demonstrated that approximately 46% of the variance in *subjective* (e.g., parent or child reported) sleep difficulties in middle childhood twins is mostly accounted for by genetics, and subjective sleep problems appear to be stable across middle childhood in twins (e.g., Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009). However, other studies suggest only about 35% of subjective sleep duration is accounted for by genetic factors, with the remaining proportion of variance in sleep duration in infancy and early childhood attributed to shared and non-shared environmental factors in univariate ACE models (Brescianini et al., 2011). Furthermore, environmental factors may account for associations (covariance) between sleep and mental health or interpersonal stressors in childhood. For example, family chaos accounted for a significant proportion of the covariance in the association between sleep problems and anxiety in childhood (36%; Gregory et al., 2005). Overall, specific objective measures of

sleep may suggest strong genetic influences on child sleep parameters, whereas subjective measures of sleep indicate strong shared and non-shared environmental influences on sleep.

Other research indicates that genetic, shared environmental, and non-shared environmental contributions may vary depending on who provides the report of nighttime sleep (parent or child), as well as the sleep parameter being measured, such as sleep duration, sleep onset latency and night wakings (Gregory et al., 2006). Given this heterogeneity, it is necessary to further examine additive genetic, shared environmental, and non-shared environmental contributions to different types of sleep parameters for children (e.g., sleep duration, daytime sleepiness). As such, the current study examined genetic and environmental contributions to parent-reported child nighttime sleep duration, sleep dysregulation, and daytime sleepiness across childhood (12 mos, 30 mos, and five years).

Parenting and Child Sleep

Although parent-child relationships and marital conflict have been linked to child sleep (Bordeleau, Bernier, & Carrier, 2012; El-Sheikh et al., 2006; Kelly et al., 2014; Sheridan et al., 2013), few studies have investigated relations between specific parenting styles and child sleep behavior. Given prior studies that indicate various aspects of parenting may influence sleep patterns and normative sleep trajectories in children, the current study tested whether the frequency of punitive parenting practices was associated with shortened nighttime sleep duration, increased sleep dysregulation and greater daytime sleepiness in young twins.

Parenting styles. Parenting practices play a critical role in early child developmental trajectories, with warm, responsive parenting linked to better child outcomes, including greater changes or increases in cognitive and social learning in early childhood (e.g., Landry, Smith, & Swank, 2003). In contrast, punitive parenting (i.e., harsh parenting, high punishment) has been associated with child anxiety (Laskey & Cartwright-Hatton, 2009) and greater child aggression (Stormshak, Bierman, McMahon, & Lengua, 2000; Trickett & Kuczynski, 1986). This evidence suggests that parenting styles are important for child development, and parental behavior may be particularly important in differentially shaping child mental and physical health outcomes. As parents play a large role in child developmental outcomes, further research examining the role of punitive parenting practices on child health outcomes, such as sleep, is necessary.

The most well known theory used to understand the role of parenting on children's developmental outcomes is Baumrind's (1966) Parenting Styles. Baumrind's (1966) parenting styles framework structures views on parental discipline and informs bidirectional relations between parenting and child behavior. Within Baumrind's (1966) parenting styles theory, the categorization of parenting styles includes permissive, authoritarian and authoritative parenting. Permissive parenting is characterized by non-punitive punishment, indulgent, and accepting behavior. Parents who utilize permissive parenting are supportive and accessible to their children, despite lack of control or authority over their children (Baumrind, 1966; Baumrind, 1978). Authoritarian parenting acts in opposition to permissive parenting, and is characterized by punitive punishment, high behavioral and moral standards, and absolute control over the child (Baumrind, 1966; Baumrind, 1978). Authoritarian punishment is often irrational and may not be fully

understood by children; thus, children exposed to authoritarian parenting practices may develop avoidant behavior in situations in which punishment may occur. Children exposed to authoritarian parenting practices may also learn more aggressive behaviors from receiving punishment and may display increases in externalizing behavior (Baumrind, 1966). Finally, authoritative is typically regarded as the optimal parenting style and consists of a balance between firm discipline and allowing the child freedom to make decisions (Baumrind, 1966; Baumrind, 1978). A key aspect of authoritative parenting is that punishment is reasonable and parents explain decisions related to punishment to the child. In rationalizing punishment for certain behaviors, children learn reasoning for punishment and may be better equipped to regulate behavior later.

Defining parental punitive discipline. Although knowledge of parenting styles is important, examining parental disciple practices may reveal how or why children display particular behavioral or emotional outcomes. Given that each of the three parenting types outlined by Baumrind (1966) contain varying degrees of punitive discipline, there is a need to identify outcomes related to punitive discipline, quality of parenting may be a vulnerability (risk) factor within child development.

In terms of defining parental punitive discipline, Baumrind (1966) notes that punitive discipline may include verbal threats or hostile remarks toward the child (e.g., ridicule or strong disapproval), as well as corporal punishment including spanking or slapping the child on the hand, buttock or face. Other researchers have similarly defined parental punitive discipline as physical punishment or inconsistent parenting (Stormshak, Bierman, McMahon, & Lengua, 2000). Using these definitions of punitive discipline, Stormshak et al. (2000) demonstrated that greater punitive discipline was associated with

greater oppositional and aggressive behaviors in a large sample of kindergarteners. Similarly, maternal and paternal punitive discipline directly influenced child emotion regulation and aggression (respectively) in a sample of Chinese preschool-age children (Chang, Schwartz, Dodge, & McBride-Chang, 2003). Harsh physical punishment was also linked to conduct problems in a sample of preschool-age children, whereas harsh verbal punishment was associated with self-concept problems in the same study (Berzenski & Yates, 2013). In addition, lower parental warmth and higher levels of punitive discipline have been associated with reduced child academic performance (Parker, Boak, Griffin, Ripple, & Peay, 1999). Beyond preschool age, parental punitive discipline has been linked to more depressive symptoms in children compared to non-depressed children (Puig-Antich et al., 1985) increased child externalizing problems (Johnston & Jassy, 2007), and more conflictual and non-supportive peer relations (Domitrovich & Bierman, 2001) in middle and late childhood. Overall, we can surmise that multiple studies and researchers hold similar definitions for punitive discipline, and parental punitive disicpline is associated with a number of behavioral, emotional and social outcomes in early and middle childhood.

Just as parental punitive discipline can be defined in various ways, there are also multiple ways researchers can measure or evaluate punitive discipline. Parental punitive discipline has been measured in terms of level or amount of punitive discipline, but it may also be evaluated in terms of the frequency, intensity, and thresholds at which punitive discipline has lasting, harmful effects on affected children. Considering frequency, intensity and threshold levels for punitive discipline may provide valuable

additional information about punitive parenting, as various aspects of parental punitive discipline may predict different developmental outcomes.

Associations between parenting and child sleep. As noted, few studies have examined whether specific parenting styles influence sleep patterns and normative sleep trajectories in children. However, positive parent-child interactions (both maternal and paternal) have predicted greater parent-reported nighttime sleep percentage (e.g., Bordeleau et al., 2012). In addition, parent- and child-reported marital conflict, as well as actual parent-child conflict, have predicted increased daytime sleepiness and sleep activity, shorter sleep duration, and reduced sleep efficiency in eight-year-olds (El-Sheikh et al., 2006), as well as reduced sleep efficiency and more nighttime waking episodes across late childhood (ages 9 to 11; Kelly et al., 2014). Finally, maternal over-involvement (excessive warmth or being overbearing) with the child at bedtime at 12 and 18 months was associated with later sleep problems and shorter sleep duration when the child reached five years (Sheridan et al., 2013). Together, these findings suggest that various parenting styles or behaviors may influence early child sleep behaviors.

Parental punitive discipline specifically may also be linked to child sleep problems. For example, maternal hostility and hostile parenting in the first year of life predicted poorer sleep behavior almost five years later (Rhoades et al., 2012). In contrast, Bates et al. (2002) studied a high-stress sample of families and were unable to show that low maternal warmth led to greater sleep dysregulation in preschool children. Benoit, Seanah, Boucher, & Minde (1992) showed that children with diagnosed sleep disorders were more likely to have insecurely attached mothers (low emotional support). Extreme cases, such as child abuse and neglect, have demonstrated that children who were

sexually abused (not by parents in all cases) had more parasomnias before and after psychiatric admission than physically abused or non-abused children (Sadeh et al., 1995). In addition, Martinez-Roig, Domingo-Salvany, and Llorens-Terol (1983) found that some children who experienced parental physical or emotional punishment displayed subsequent sleep disturbances. Given inconsistent findings, I examined the association between frequency of punitive discipline and child sleep behavior.

Although this literature provides some support for the link between various aspects of parenting and child sleep problems, there is no literature to date examining the association between parental punitive discipline in particular and childhood sleep. It is possible that parental punitive discipline acts as a significant interpersonal stressor for the child, thereby creating a sense of anxiety or uncertainty surrounding punishment for the child's behavior. Heightened anxiety or uncertainty regarding parental punitive punishment may lead to nighttime anxiety or increased sleep onset latency, both of which indicate disrupted sleep in childhood.

Bidirectional effects of parenting and sleep. Child sleep problems may also play an evocative role, such that child sleep problems in infancy and early childhood lead to tension or stress in the family, particularly within marital relations and parent-child interaction (Sadeh & Anders, 1993). It is also possible that child sleep problems lead to greater child externalizing behavior (e.g., problem behavior, acting out) or displays of negative affectivity. If this is the case, increases in child behavioral problems and negative emotionality may in turn evoke more negative or hostile parenting. Lack of child sleep likely leads to greater parental fatigue as well, potentially resulting in higher family conflict and perpetual child sleep problems (Sadeh & Anders, 1993). Given that no

studies to date have reported whether child nighttime sleep duration and daytime sleepiness impact parenting practices, the current study tested whether there are bidirectional relations between punitive parenting practices and child sleep parameters.

Heritability of parenting practices. In addition to understanding parenting at a phenotypic level, parenting practices can be examined in the context of heritability, showing additive genetic, shared environmental, and non-shared environmental influences. Positive support from parents is approximately 86% heritable, whereas negative control and negative affect aspects of parenting are about 24% and 48% heritable, respectively (Losoya, Callor, Rowe, & Goldsmith, 1997). Another study demonstrated that shared environmental factors account for almost 62% of the variance in harsh parenting, 47% of the variance in negative parenting control, and 49% of the variance in positive parenting control in univariate models, with only 0-12% accounted for by heritability (Deater-Deckard, 2000). Finally, one study showed that parenting characterized by negative control contains mostly shared environmental influence (65% of variance accounted for), with additive genetic factors estimated at 35% (Oliver, Trzaskowski, & Plomin, 2014). Thus, heritability estimates related to parenting in these studies appear to be more heavily influenced by parental factors (shared or common environmental factors), which evoke particular child responses. However, it seems that negative parenting (i.e., control or affect) was influenced moreso by the shared environment, whereas positive parenting (e.g., support) showed greater additive genetic influence (see Deater-Deckard, 2000, Losoya et al., 1997, and Oliver et al., 2014).

Although these studies provide evidence for genetic and environmental contributions to parenting, no studies to my knowledge have examined the link between

punitive parenting practices and child sleep behavior using bivariate behavioral genetic models. Additionally, research has shown how additive genetic, shared environmental, and non-shared environmental variances shift over time for parenting, and research notes the bidirectional influence between parenting and child outcomes, with child emotions and behavior evoking particular parenting styles (McGue et al., 2005). Understanding whether genetic and environmental factors may influence the relationship between parent-child interaction and child sleep may inform parenting practices and potential interventions for child sleep problems. The current study examined heritability estimates of parental punitive discipline and child nighttime sleep duration, sleep dysregulation, and daytime sleepiness in univariate behavior genetic models to identify fluctuations in genetic and environmental variance over time. Furthermore, I examined genetic and environmental associations between parental punitive discipline and child sleep parameters in bivariate behavior genetic models to determine whether genetic or the environment account for this association.

Sibling Relationships and Child Sleep

Despite lack of research examining the role of sibling interaction in various child sleep parameters, it is possible that sibling relationships (specifically sibling conflict) play a role in child sleep behavior and disruption, particularly if the one or both siblings show difficult temperament, high negative affectivity or if siblings consistently remain in close proximity to one another (e.g., share a room, sleep in the same bed). Given that other familial relationship (e.g., parent-child relationship) have been documented and/or hypothesized to impact child sleep behavior, the current study explored whether higher

levels of sibling conflict were related to child shortened nighttime sleep duration and greater daytime sleepiness in young twins, including examining bidirectional relations.

Sibling relationships. Despite being understudied, siblings have special, life-long relationships, much like the parent-child relationship (Cox, 2010; McHale, Updegraff, & Whiteman, 2012; Whiteman, McHale, & Soli, 2011). Furthermore, more than 85% of children in the United States grow up with a sibling (Conger & Kramer, 2010; McHale et al., 2012). Like the parent-child relationship, siblings typically share an environment, allowing for siblings to influence one another through shared emotional experiences and social interaction.

Several common theories related to familial relationships may be appropriate when discussing sibling relationship development. The Family Systems Theory (Bowen, 1976) examines transactional, interpersonal relations within the family and may be applicable to sibling relationship development. Attachment Theory is another possible model that may explain the way in which sibling relationships develop (Ainsworth, 1978). Ainsworth (1978) and other theorists (e.g., Whiteman et al., 2011) suggest that although children may form attachments with caregivers first, older siblings or same age siblings may also serve as secure bases for emotional attachment early in life.

Social learning theory (Bandura, 1977) may also help inform sibling relationship development specifically, in that sibling relationships are critical for children to learn how to interact socially, emotionally and behaviorally with others (Lockwood, Kitzmann, & Cohen, 2001; Stormshak, Bellanti, & Bierman, 1996). For example, the ability to understand others' motivations for behavior may lead to more sibling cooperation, whereas inability to engage in perspective-taking may lead to increased sibling conflict

(e.g., Whiteman et al., 2011). Social learning theory may also be related to the proposed association between sibling relationships and childhood sleep, as it is possible for one sibling to model either "good" or "bad" sleep behavior, sleep hygiene or bedtime routines for another sibling. For example, a child who shows inconsistent bedtime routines or regularly cries before bedtime may model this set of behaviors for his or her twin, leading to poorer sleep behaviors and sleep disruption for both children. Thus, social learning theory may explain how and why children display particular behavior around bedtime, which may lead to sleep problems.

Each of these theoretical models shares a common thread: they all propose transactional relationships in the sibling dyad and hypothesize that siblings influence one another behaviorally, emotionally and socially. Thus, these theoretical frameworks directly relate to the current study's efforts of understand the association between the quality of sibling relationships and child health behaviors. These theories bolster the hypothesis that transactional relations in sibling dyads may significantly influence health behaviors, particularly sleep.

Sibling relationship theory. There are a number of key elements essential to sibling relationship development in early childhood (Kramer, 2010; McHale et al., 2012). Theories suggest that sibling gender, age or birth order, and the number of siblings within a family unit are all factors that may influence sibling relationship development (McHale et al., 2012). Kramer (2010) also proposed six critical elements that characterize sibling relationships: Positive engagement, cohesion and shared experiences, perspective taking, regulation, conflict resolution, and parenting. According to Kramer's (2010) theory, the levels of each of these factors within a given sibling dyad may lead to more conflictual or

cooperative sibling relations. Indeed, theory suggests that sibling relationships tend to fluctuate between positive (cooperative) and negative (conflict) socioemotional events on a broad level (Cox, 2010). Furthermore, recent theories posit that the peer-like relationships between siblings that demonstrate a balance between conflict and cooperative events are necessary for developing healthy sibling relationships (Cox, 2010; McHale et al., 2012). At least one study has shown that twins seek more interaction with one another and show distress when separated, suggesting cohesion and shared experiences among siblings (Tancredy, 2004). Bekkhus, Stanton, Borge, and Thorpe, (2011) also found that DZ twins displayed less warmth compared to MZ twins, indicating that different types of siblings may show varying amounts of conflict and cooperation.

In addition to conceptualizing sibling relationships based on valence (positive cooperation or negative conflict), researchers have proposed that sibling relationships can be evaluated in terms of frequency of conflictual or cooperative events and behavior, as well as the intensity of these encounters (Cox, 2010; McHale et al., 2012). Determining the valence, frequency and intensity of sibling conflict or cooperative events may help show how sibling relationships develop over time by demonstrating how positive or negative a sibling relationship may be overall. As such, the current study examined negatively valence sibling relationships (sibling conflict), and explored factors related to sibling conflict to better understand how conflictual sibling relationships influenced individual child health outcomes like sleep.

Previous research on sibling relationships. Studies have shown that conflict and cooperation in the sibling relationship may be linked to later behavioral outcomes in childhood (e.g., Bekkhus et al., 2011). For example, low closeness and high conflict

within sibling relationships (twins particularly) has been linked to externalizing behavior in childhood over time (Bekkhus et al., 2011). Specifically, higher sibling conflict was linked to increased hyperactivity and conduct problems before children entered preschool and immediately following the start of preschool. Sibling conflict may also be a family stressor that impacts future child outcomes, such as peer interaction (Lockwood et al., 2001; Stormshak et al., 1996). Finally, sibling relationships may be linked to both social and emotional outcomes (Stormshak et al., 1996). Individuals who had sibling relationships characterized by approximately equal levels of conflict and cooperation demonstrated more positive social and emotional competence compared to sibling dyads with higher levels of conflict. Overall, recent research indicates that the sibling relationship may be an important relationship within the family system that influences social, emotional and behavioral outcomes in childhood.

Given the association between sibling conflict and numerous psychosocial and emotional outcomes, it is also likely that sibling relationships are related to individual child sleep behaviors. Although there is no research to my knowledge linking sibling relationships to child sleep outcomes, the complex interpersonal interactions that occur in sibling dyads likely influence child health behaviors like sleep. For example, it is possible that high sibling conflict reduces sleep quality, sleep duration or increases night waking, particularly if the one or both children in the sibling dyad show difficult temperament, more negative emotionality or siblings are consistently in close proximity with one another (e.g., share a room, sleep in same bed or room). In addition, sibling conflict may influence child sleep in similar ways as negative parent-child interactions.

Bidirectional relationships between sibling conflict and childhood sleep parameters may also exist, such that sibling conflict may lead to sleep disruption in childhood, such that if the siblings are in close proximity consistently or are emotionally reactive, it is possible that sleep may become disrupted or dysregulated. However, poor sleep behaviors early in childhood may lead to increases in behavioral problems (conflict between twins) or problems regulating emotion. If this is the case, it is likely that poor sleep behavior leads to more conflictual sibling relationships, potential through child externalizing and internalizing problems. Overall, studies are needed to examine whether there is an association between sibling conflict and childhood sleep, as well as the directionality of this association. As such, the current study tested bidirectional relations between sibling conflict and child nighttime sleep duration, sleep dysregulation, and daytime sleepiness (over time).

Heritability of sibling conflict. Some aspects of the sibling relationship such as cooperation and conflict behavior have been tested in quantitative behavioral genetic studies, showing that sibling relationships may demonstrate genetic and environmental influences. One study suggests that sibling cooperation contains additive genetic influence that is also shared with child temperament, while sibling conflict appears to be linked to difficult temperament through shared genetic influences (Lemery & Goldsmith, 2002). Sibling conflict also shows unique additive genetic, shared environmental and non-shared-environmental influence (a^2 = .32, c^2 = .34, e^2 = .33; Lemery & Goldsmith, 2002). These findings suggest that sibling-specific characteristics (i.e., temperament) may influence heritability of sibling relationships across time. Although I did not test whether person-level factors (like temperament) moderate the heritability of sibling relationships

over time, I examined genetic and environmental contributions to sibling conflict in a univariate analysis to determine whether genetics or the environment more heavily influenced sibling conflict. The current study also addressed gaps in the literature by testing genetic and environmental contributions to the association between sibling conflict and child sleep using bivariate behavior genetic models.

Twins as Siblings

Twins are unique siblings, such that twins share the prenatal environment, and are at approximately the same level of maturity across development. Furthermore, shared genetic composition between co-twins allows researchers to examine genetic and contextual factors that contribute to behavior and development (Plomin, DeFries, Knopik, & Neiderhiser, 2013). The twin method is one way of exploring the contribution of genetic and environmental factors on developmental outcomes in the population (Plomin et al., 2013). Initially, individual differences in genetic and environmental contributions to behavior were examined by comparing MZ and DZ twins (Plomin et al., 2013). Comparing MZ and DZ twins helps show heritability of traits (Plomin et al., 2013), such that any differences observed between MZ twins can be attributed to environmental factors alone, given that MZ twins share 100% of their genetic composition (Plomin et al., 2013). On the other hand, any behavioral differences between DZ twins may be attributed to both genetic and environmental factors, as DZ twins only share approximately 50% of their genetic composition (Plomin et al., 2013). However, improved statistics allow for more sophisticated methods of examining genetic and environmental contributions to behavior (i.e., quantitative behavioral genetics; Plomin et al., 2013).

ACE Model of Behavioral Genetics. The behavioral genetic analysis most often used with the twin method to show genetic and environmental contributions to a particular phenotype (behavior or trait) is called the ACE model (Neale & Cardon, 1992). The ACE model is used primarily with MZ and DZ twins to demonstrate variance in additive genetic, shared environmental and non-shared environmental factors that contributes to a phenotypic trait or outcome (Kohler, Behrman, & Schnittker, 2011; Neale & Cardon, 1992). The additive genetic (A) portion of the model accounts for the likelihood that multiple genes influence a phenotype (Kohler et al., 2011; Neale & Cardon, 1992). Furthermore, the proportion of additive genetic contribution to a behavior will differ between MZ and DZ twin pairs, due to the differences in percentage of shared genetic composition between twin types. Shared environmental factors (C) represent any aspect of the environment that is shared or common for a set of twins and may influence a phenotype. Shared environmental factors are assumed to equal one (100%) among MZ and DZ twins pairs raised together; in studies of twins raised apart, the shared environmental component is expected to be extremely low (Kohler et al., 2011; Neale & Cardon, 1992). Thus, the shared environmental component of the ACE model represents the phenotypic variation accounted for by shared factors in the twins' environment. As such, the C component in behavior genetic analyses is expected to be relatively low because we expect there to be little phenotypic variation in aspects of twins' environments that are shared. Finally, the non-shared environmental component (E) in the ACE model represents variation in the phenotype accounted for by contextual experiences the twins *do not* share (Kohler et al., 2011; Neale & Cardon, 1992). The E component is thought to be important for both MZ and DZ twins in measuring contextual

factors that the twins may experience independently, such as schooling or peer groups. Overall, the ACE model is critical as it informs and describes contributions to behavior for twins, showing proportions of variation in genetic and environmental factors in specific phenotypic traits. The ACE model is important to the current study, as a major goal was to determine genetic and environmental contributions to individual differences and relations among punitive disicipline, sibling conflict and child nighttime sleep duration, sleep dysregulation and daytime sleepiness.

Twins and sleep. Previous studies involving twin pairs and sleep have primarily aimed to show genetic and environmental influences on normative sleep problems across time or on disorders related to sleep (e.g., depression or anxiety; Gregory et al., 2005). However, studies involving twins and sleep behavior have not yet considered the role of the sibling relationship and how sibling conflict contributes to ongoing sleep normative problems (i.e., shortened sleep duration and greater daytime sleepiness). Thus, a major goal of the current study was to understand how sibling conflict among twins was associated with nighttime sleep duration, sleep dysregulation and daytime sleepiness in childhood.

Current Study

The purpose of the current master's thesis was to determine whether there was a phenotypic association between parental punitive discipline at 30 months of age (one indicator of parenting) and sleep behavior when twins were both 30 months and five years old (nighttime sleep duration, sleep dysregulation, and daytime sleepiness). I explored whether high sibling conflict among five-year-old twins was associated with concurrent sleep behavior, acting as risk factors for poor nighttime sleep duration and

increased daytime sleepiness. Bidirectional associations between parental punitive discipline, sibling conflict, and child sleep parameters (at 12 and 30 mos) were also tested. Finally, I examined the association between punitive discipline, sibling conflict, and child sleep using a behavioral genetic design. As such, I examined univariate additive genetic, shared environmental, and non-shared environmental contributions to punitive discipline (30 mos), sibling conflict (five years), child nighttime sleep duration (12 mo., 30 mos, and five years), child sleep dysregulation (12 mos, 30 mos, and five years), and child daytime sleepiness (five years). I also conducted bivariate behavioral genetic models to estimate the additive genetic, shared environmental, and non-shared environmental contributions to the associations between parental punitive discipline at 30 months and child sleep at 30 months and five years, as well as the association between sibling conflict at five years and child sleep parameters at five years (Figure 1). Overall, I used three different types of models in the current study: phenotypic, univariate ACE, and bivariate ACE behavior genetic models.

Phenotypic analyses. I conducted phenotypic analyses to determine bidirectional associations between the frequency of punitive discipline and child nighttime sleep duration, sleep dysregulation and daytime sleepiness, as well as between levels of sibling conflict and concurrent child nighttime sleep duration and daytime sleepiness. Previous research has demonstrated that stress and conflict within the parent-child relationship is a risk factor for multiple physical and mental health outcomes (Laskey & Cartwright-Hatton, 2009; Stormshak et al., 2000; Trickett & Kuczynski, 1986). Thus, I hypothesized that greater frequency of parental punitive discipline at 30 months would be associated with shorter nighttime sleep duration and greater child sleep dysregulation at 30 months,

as well as shorter nighttime sleep duration and increased daytime sleepiness at five years. Similarly, I expected both shorter nighttime sleep duration and greater sleep dysregulation at 12 to be associated with more frequent punitive discipline at 30 months. Child daytime sleepiness was not used to predict parental punitive punishment at 30 months, as daytime sleepiness was only measured at the five-year assessment.

Furthermore, studies have shown that conflict and negative sibling relations are linked to poor emotional and behavioral outcomes in childhood (Bekkhus et al., 2011; Lockwood et al., 2001; Stormshak et al., 1996). Thus, I hypothesized that greater conflict in sibling relationship at five years would be associated with shorter concurrent nighttime sleep duration and increased daytime sleepiness in twins. Similarly, I expected shorter nighttime sleep duration and greater sleep dysregulation at 12 and 30 months would be associated with greater sibling conflict at five years.

Univariate analyses. I conducted heritability estimate analyses to determine additive genetic, shared environmental, and non-shared environmental contributions independently for measures of parental punitive discipline, sibling relationships, and child sleep parameters.

Sleep. Behavioral genetic studies revealed that sleep architecture and sleep behavior is highly heritable and has significant additive genetic contributions in childhood and adolescence (Ambrosius et al., 2008; Gregory et al., 2009). Furthermore, studies have shown that shared environmental factors contribute to various sleep parameters, such as night wakings and sleep duration (e.g., Brescianini et al., 2001). From this, I hypothesized that the univariate ACE model would show that the greatest proportion of the variance in the sleep parameters (sleep duration, daytime sleepiness and

sleep dysregulation) would be accounted for by additive genetic factors and shared environmental factors. However, I expected that sleep duration, daytime sleepiness and sleep dysregulation would show differing amounts of genetic contribution, as research has suggested that the amount of genetic and/or environmental contributions to distinct sleep parameters may vary (see Barclay & Gregory, 2013). For example, Gregory, Rijsdijk, & Eley (2006) found that parent-reported sleep duration in young twins was primarily accounted for by additive genetic factors (approximately 71%), whereas roughly equal amount of genetic and environmental influences contributed to parent-reported daytime sleepiness (a^2 = .32, c^2 = .34, e^2 = .33) in young twins.

In addition, given a large body of research that has suggested the heritability of many behaviors (including sleep) increases over time (Plomin et al., 2013), I hypothesized that the genetic contributions to sleep duration and dysregulation would increase over time in the current study. Heritability fluctuations in daytime sleepiness were not tested, as this sleep parameter was only been measured at the five-year assessment.

Parental punitive discipline. Given previously noted research indicating that the greatest proportion of the variance in parenting is accounted for by shared environmental factors (Deater-Deckard, 2000; Losoya et al., 1997; Oliver et al., 2014), I hypothesized that the largest proportion of the variance in punitive discipline would be accounted for by shared environmental factors, followed by additive genetic factors.

Sibling relations. A small amount of literature has suggested that sibling conflict contains unique and shared genetic contributions and some shared and non-shared environmental influences (Lemery & Goldsmith, 2002). Given these findings, I

hypothesized that the largest proportion of the variance in the sibling conflict would be accounted for by additive genetic factors.

Bivariate Analyses. I also conducted bivariate ACE models to determine covariance of additive genetic, shared environmental, and non-shared environmental contributions in the association between parental punitive punishment and child sleep, as well as the association between sibling conflict and child sleep parameters.

***Parenting and sleep*.** As previously mentioned, I expected that most of the variance in punitive discipline would be accounted for by shared environmental factors, and a significant proportion of variance in sleep would also accounted for by additive genetic and shared environmental factors (Deater-Deckard, 2000; Gregory et al., 2005). Thus, I hypothesized that the greatest proportion of covariance in the association between punitive discipline and child sleep would be primarily accounted for by shared environmental factors.

***Sibling conflict and sleep*.** Despite lack of literature regarding heritability of sibling relationships, some research has suggested moderate unique and shared genetic contributions to the sibling relationship (e.g., Lemery & Goldsmith, 2002). In addition, previous research has noted moderate genetic contributions to child sleep (e.g., Brescianini et al., 2011). As such, I hypothesized that the greatest proportion of the covariance in the association between sibling conflict and child sleep parameters would be accounted for by additive genetic factors.

Methods

Participants

The Arizona Twin Project (ATP; Lemery-Chalfant, Clifford, McDonald, O'Brien, & Valiente, 2013) includes 582 twins (MZ = 172, same sex DZ = 234, opposite sex DZ = 252) who have been studied across three time points: 12 months of age, 30 months of age, and five years of age. The complete ATP sample (12-month assessment) is diverse, with approximately 55% European-American, 28.3% Latino, 6% Asian American and 5% African American families. In addition, the sample is evenly split between males and females at all three data collections, with an average of 52% male twins and 47.5% female twins participating at each time point. Socioeconomic status (SES) was calculated at all three time points and was strongly correlated between 30 months to five years (r = 0.86, $p < .01$), showing stability in SES. At 30 months of age, 504 twins participated and the primary caregiver completed a telephone or online interview measuring the frequency of punitive and inductive parenting techniques, the home environment, and other twin developmental characteristics. The most recent wave of ATP included 406 twins and their parents measured at approximately five years of age (M = 4.8 years, SD = 0.39; 51.2% male) as the twins were transitioning to kindergarten. Approximately 60.6% of the ATP sample at five years was European-American and 24.1% were Latino (5.9% Asian, 4.4% African-American, 1.5% Native Hawaiian, 1% Native American, and 2.5% Other).

Attrition analyses were conducted to examine mean differences on the study and demographic covariates (e.g., ethnicity, family structure, SES) between families or individuals who *did not* complete interviews at both 30 months and five-years (in addition to the 12-month assessment) and families who *did* participate at 30 months and

five years. Individuals who did not participate at both 30 months and five years were more likely to be of diverse ethnic background (e.g., Latino, African American, Asian) than individuals who completed interviews at both 30 months and five years, $t(756) = 2.48$, $p < .01$, $M_{diff} = .22$, $SE_{diff} = .09$. Individuals who participated at both 30 months and five years were more likely to be married (rather than separated, divorced, etc.) than individuals who did not complete interviews at both 30 months and five years, $t(65) = 3.05$, $p < .01$, $M_{diff} = .21$, $SE_{diff} = .08$ (approximation tests used and equal variances not assumed). Finally, individuals who did not participate at both 30 months and five years were more likely to have shorter nighttime sleep duration than individuals who did complete interviews at both 30 months and five years, $t(393) = -2.70$, $p < .01$, $M = -.40$, $SE = .15$. There were no other differences in the samples based on SES, punitive discipline, sibling conflict, sleep dysregulation, and daytime sleepiness.

Measures

Child Sleep Habits Questionnaire (CSHQ). The Child Sleep Habits Questionnaire is a 48-item revised parent-report measure of multiple dimensions of sleep, such as total sleep duration, sleep duration problems, bedtime resistance, sleep latency, nighttime wakings, sleep anxiety, parasomnias and daytime sleepiness (Owens, Spirito, & McGuinn, 2000). The current study utilized two scales in the CSHQ: Nighttime sleep duration and daytime sleepiness. Raw nighttime sleep duration and daytime sleepiness scores were used as outcome variables.

Nighttime sleep duration was the raw total number of hours a child slept during the night on average, as reported by the primary caregiver. Nighttime sleep duration, rather than total sleep duration (sum of hours of sleep during the day and at night), was

selected for analyses due to concerns that primary caregivers would not provide an accurate estimate of daytime sleep duration (e.g., naps), given that approximately 70% of the children (N = 294) attended a preschool and/or childcare (in-home or out-of-home) during the day at the five-year assessment.

Daytime sleepiness was characterized by difficulty waking up in morning and frequently falling asleep during daytime activities. Daytime sleepiness was a seven-item scale (Cronbach's α = .73). The seven items were scaled on a five-point Likert scale, where 1 was "*Never,*" 3 was "*Sometimes"* and 5 was "*Always*." No items were reverse scored. Individual sum scores were constructed for each twin using the full daytime sleepiness scale (according to scale scoring in Owens et al., 2000), where higher total sum scores for each twin reflected greater daytime sleepiness.

Infant Toddler Social and Emotional Assessment (ITSEA). At 12 and 30 months, primary caregivers completed the ITSEA, a 139-item survey that captures four broad domains of infant and toddler development: child internalizing and externalizing behavior, regulation and competencies (Carter, Little, Briggs-Gowan, & Kogan, 1999). The current study utilized a measure of average nighttime sleep duration at 12 and 30 months, as well as the ITSEA sleep scale, which measured sleep dysregulation using five items (e.g., *Twin A* has trouble falling asleep or staying asleep). The five items were scaled on a three-point Likert scale, where 0 was "*Not true/Rarely,*" 1 was *"Somewhat true/Sometimes,"* and 2 is "*Very true/Often.*" One item was reverse scored to show that higher scores reflected greater sleep dysregulation. We computed a mean sleep dysregulation score for each twin at 12 months (Cronbach's α = .68) and 30 months (Cronbach's α = .73), where higher scores indicated more sleep dysregulation for a given

twin. It is of note that one item in the 12-month assessment was left out; thus, the mean composite consisted of only four items at 12 months, and the 30-month composite consisted of five items. The item left out at the 12-month assessment was "*Twin A/B* must be held to go to sleep." This item was likely purposely removed, as most infants at 12 months are still struggling to attain consistent sleep-wake patterns and many parents may endorse this sleep behavior for their twins given the development period of the twins at 12 months.

Parental Responses to Child Misbehavior (PRCM). At the 30-month assessment, parental punitive discipline was reported as a measure of parenting and the parent-child relationship. The Parental Responses to Child Misbehavior survey is a 12-item revised parent-report measure that assessed the frequency of parental responses to a variety of child misbehaviors (Holden, Coleman, & Schmidt, 1995). PRCM items were scaled on a five-point Likert scale, where 1 was "*Always or Almost Always*" and 5 was "*Never or Almost Never*" (Holden et al., 1995). All items were reversed scored, such that higher scores (closer to 5) reflected higher frequency of a particular parental discipline behavior.

An exploratory factor analysis (EFA) was conducted to distinguish between items that characterized punitive parental punishment and items reflecting inductive or authoritative parental punishment (Caughy, Miller, Genevro, Huang, & Nautiyal, 2003). The EFA allowed us to form meaningful composite measures of individual differences in punitive discipline and identify the underlying factor structure. Results yielded a two-factor structure, which is consistent with prior research (Caughy et al., 2003). The first factor (punitive discipline factor) included threatening, spanking with hand, yelling in

anger, and slapping the hand (Cronbach's α = .60). The second factor (authoritative discipline) included reasoning, diversion, negotiation and withdrawing privileges (Cronbach's α = .55). Only the punitive discipline factor was utilized in the analyses, and mean PRCM punitive discipline scores were computed using the four items that loaded on this factor. Mean punitive punishment scores were computed separately for each twin, with higher PRCM scores indicating more punitive discipline for a given twin. Individual PRCM scores for each twin allowed us to conduct heritability estimates individually.

Sibling Relationship Questionnaire (SRQ). The Sibling Relationship Questionnaire is a 15-item revised parent-report measure that assessed the interpersonal relationship of the twins in three areas: closeness or warmth, conflict, and power (measured at five years; Furman & Buhrmester, 1985). The current study utilized the five items within the conflict scale (i.e., How much does *Twin A* insult and call *Twin B* names? How much does *Twin B* insult and call *Twin A* names?). SRQ items were scaled on a five-point Likert scale, where 1 is "*Hardly at All*" and 5 is "*Extremely Much.*" We computed a mean SRQ conflict score for each twin, where higher scores indicated more conflict behavior for a given twin (Cronbach's α = .87). SRQ conflict scores were computed for each twin individually, allowing us to estimate genetic and environmental influences using the ACE model.

Zygosity. Zygosity was assessed through primary-caregiver reports using the Zygosity Questionnaire for Young Twins (ZQYT; Goldsmith, 1991). The ZQYT is a 32-item measure that differentiates between MZ and DZ twins using parent report of pregnancy (e.g., prematurity, use of fertility treatment, twin birthweight), and physical appearance differences between the twins (e.g., hair color, height, eye color). Goldsmith

(1991) noted that multiple studies have shown parent-report zygosity measures to be between 93% and 98% accurate in characterizing twin zygosity, making questionnaires a reliable alternative to genotyping (see Jackson, Snieder, Davis, & Treiber, 2001 and Forget-Dubois et al., 2003).

Covariates. Age, sex, twin ethnicity, family structure, and current socioeconomic status (SES) were included in all phenotypic models as covariates. Ethnicity was measured by asking the primary caregiver to indicate which ethnicity he or she considered the twins to be, selecting from "European American," "Hispanic or Latino (specifying specific origin, i.e., Mexican descent)," "African American," 'Asian," "Native American," "Native Hawaiian or Pacific Islander," "Mixed," or "Other-specify." In analyses, a dummy code for ethnicity was created, such that 0 = "European American" ethnicity, and 1 = "All Other Ethnicities" to distinguish between White participants and more ethnically diverse participants. Family structure was reported by the primary caregiver and measured relationships status of the twins' parents using the question, "What is your current relationship status?" Answer choices included statuses such as "Married," "Separated," "Divorced," "Widowed," "Always single," "In a partnership" and "Other-specify." In analyses, a dummy code for family structure was created, such that 0 = "Married" and 1 = "Other Family Structures" to distinguish between families in which parents were married and all other family structures. Family SES was measured by taking the mean of the mother's education level, father's education level and total family income before taxes.

Procedure

When the twins were 12 months of age, researchers interviewed the full sample of 582 twins and their mothers concerning twin zygosity, infant physical health, maternal perceived stress and parenting characteristics. Researchers also gathered information about birth risks and pre- and perinatal birth complications through hospital and state birth records. At 30 months, parents completed a telephone or online interview, including questions related to the twins' development, parenting style, the home environment, and child characteristics. At the most recent data collection (five years), families completed a telephone or online survey containing questions related to twin preparedness for kindergarten, home environment, sibling relationships, and individual sleep habits (among others). At 12 and 30 months, bilingual staff conducted interviews when necessary, and the survey included measures previously used in diverse populations when possible. Participating families were also compensated for survey completion at each wave, receiving US $50 at 12- and 30-month data waves and $15 gift card at five-year assessment.

Statistical Approach

Phenotypic analyses. Twin age, sex, ethnicity, family structure, and current SES were included in all phenotypic models as covariates. Bivariate correlations between covariate, predictor and outcome variables were also conducted, including sleep variables at 12 months, 30 months and five years to examine sleep stability across time. Mixed model regression analyses were conducted in SPSS 22 to determine whether basic associations existed between parental punitive discipline and child sleep parameters, as well as between sibling conflict and child sleep parameters. Bidirectional relations were

tested between parental punitive discipline at 30 months and child sleep parameters at 12 months. Bidirectional relations between sibling conflict at five years and child sleep parameters at 12 months and 30 months were also tested. Mixed model regression analyses were utilized to account for nested data at the family level, specifically accounting for twin co-interdependence in parental reports of twin relationships (parent-child and sibling relationships) and twin sleep behavior. Parental punitive discipline, sibling conflict, nighttime sleep duration, sleep dysregulation, and daytime sleepiness were centered at zero for phenotypic analysis when used as a predictor in analyses. Thus, unstandardized beta estimates were reported from the mixed model regression analyses.

Univarite analyses. Quantitative behavioral genetic models (univariate and bivariate) were conducted and fit in OpenMx (Boker et al., 2011), an R-based program that utilizes maximum likelihood estimation, including estimating pathways (covariances) and model fit using structural equation modeling (SEM). Univariate variables were created by regressing twins' age and sex out of each predictor (punitive discipline and sibling conflict) and residual scores were saved. Univariate variables were also created for nighttime sleep duration, daytime sleepiness and sleep dysregulation and residual scores were saved.

To calculate the genetic and environmental contributions to each predictor and outcome variable in univariate decompositions, the additive genetic (A), shared environmental (C), and non-shared environmental (E) variance were each divided by the total variance (V), such that Total Decomposition = A/V + C/V + E/V (Loehlin, 1996). The full ACE models were fit independently for each of the five variables and variances were decomposed. The A and C parameters were systematically dropped from the full

model to test whether a reduced model provided a better fit for a given variable. The E parameter contains random measurement error and was therefore not dropped from the models. Model fit for univariate models was examined using the chi-square goodness of fit index (-2LL or χ^2), as well as chi-square different tests (or log likelihood tests; indicated by $\Delta \chi^2$) which compares model fit of nested models. Non-significant probability values for the χ^2 difference test indicated that a reduced model did not fit the data significant worse compared to the full ACE model (better model fit). In contrast, significant probability values for the χ^2 difference test indicated that reduced model fit the data significantly worse compared to the full model. Akaike's Information Criterion (AIC; Akaike, 1974) was also used to assess model fit by taking into account the number of parameters in a model and penalizing models with larger number of parameters estimated. AIC values that were lower (or even negative) indicated better model fit.

Bivariate analyses. Bivariate behavioral genetic analyses (ACE models) were conducted using Cholesky ACE decompositions in OpenMx. Bivariate Cholesky decompositions provided genetic and environmental contributions to the variance for each individual phenotype, as well as decomposed any covariance shared between the two phenotypes. Thus, Cholesky bivariate ACE decompositions were conducted to decompose individual variance and covariance between the two phenotypes in four sets of models: 1) punitive discipline and child sleep parameters at 30 months, 2) punitive discipline and child sleep parameters at five years, 3) sibling conflict and nighttime sleep duration at five years, and 4) sibling conflict daytime sleepiness at five years.

Results

Preliminary Analyses

Means, standard deviations, ranges, skew, and kurtosis for the predictors (raw scores for punitive discipline and sibling conflict) and sleep outcome variables are presented in Table 1. None of the variables exceeded the recommended cutoff for positive or negative skew (2.00), nor did any variables exceed the cutoff for kurtosis (7.00; Muthén & Kaplan, 1985).

Phenotypic bivariate correlations between punitive discipline and sibling conflict and the sleep outcome measures are provided in Table 2. Punitive discipline at 30 months was positively associated with concurrent sleep dysregulation ($r(478) = .10$, $p = .03$), and sibling conflict at five years ($r(328) = .12$, $p = .04$). Sibling conflict at five years was negatively associated with nighttime sleep duration at both 30 months ($r(313) = -.13$, $p = .02$) and five years ($r(393) = -.18$, $p < .001$). Sibling conflict at five years was also positively correlated with concurrent daytime sleepiness ($r(398) = .26$, $p < .001$).

Bivariate correlations between the covariates and sleep outcome measures were also conducted (see Table 2). Older age at the 30-month assessment was associated with shorter concurrent nighttime sleep duration ($r(466) = -.11$, $p = .02$). Ethnicity was negatively correlated with nighttime sleep duration at 30 months, such that individuals who were of diverse ethnic background (e.g., Latino, African American, Asian) had shorter nighttime sleep duration ($r(468) = -.19$, $p < .001$). Higher SES at the 30-month assessment was associated with longer concurrent nighttime sleep duration 30 months ($r(428) = .29$, $p < .001$), and higher SES at five years was positively correlated with longer nighttime sleep duration at 30 months ($r(312) = .27$, $p < .001$) and five years

($r(393) = .13$, $p = .01$). Family structure (specifically, parents who were not married) at 30 months was associated with shorter nighttime sleep duration at 30 months ($r(430) = -.19$, $p < .001$) and five years ($r(361) = -.13$, $p = .02$).

Sleep duration and dysregulation at 12 and 30 months were also included in the bivariate correlation table to demonstrate the stability of the sleep parameters over time (see Table 2). Nighttime sleep duration was relatively stable across time, as nighttime sleep duration at 12 months was positively associated with nighttime sleep duration at 30 months ($r(411) = .42$, $p < .001$) and five years ($r(448) = .20$, $p < .001$). Sleep dysregulation was also stable in infancy and toddlerhood, as there was a positive correlation between sleep dysregulation at 12 and 30 months ($r(446) = .35$, $p < .001$).

Phenotypic Multiple Regression Analyses

Punitive discipline models. Using mixed model regression (30 mos age, sex, ethnicity, SES, family structure, and 12 mos sleep parameters as covariates), punitive discipline at 30 months was not associated with nighttime sleep duration at 30 months (see Model 1 in Table 3). In the same model, however, longer nighttime sleep duration at 12 months predicted longer nighttime sleep duration at 30 months. In addition, non-white individuals (e.g., Latino , African American, or Asian etc.) showed shorter nighttime sleep duration and higher SES at the 30-month assessment was associated with longer nighttime sleep duration. Exploratory interaction models between punitive discipline and ethnicity, as well as between punitive discipline and SES, were conducted, but were nonsignificant. In addition, punitive discipline at 30 months was not associated with sleep dysregulation at 30 months (see Model 2 in Table 3). However, in the same model,

greater sleep dysregulation at 12 months was associated with greater sleep dysregulation at 30 months.

Punitive discipline at 30 months was not associated longitudinally with nighttime sleep duration at five years or daytime sleepiness at five years (see Models 3 and 4 in Table 3). Within the nighttime sleep duration model, older age at the five-year assessment significantly predicted shorter nighttime sleep duration at age five. Longer nighttime sleep duration at 30 months was also associated with longer nighttime sleep duration at five years. In both models, covariates included 30-month age, sex, ethnicity, SES at 30 months, and family structure at 30 months.

In testing bidirectional effects (see Models 1 and 2 in Table 4), nighttime sleep duration at 12 months did not predict punitive discipline at 30 months (12 mos age, sex, ethnicity, SES and family structure as covariates). In the same model, however, higher SES at 12 months was associated with lower frequency of parental punitive punishment at 30 months. Similarly, sleep dysregulation at 12 months did not predict punitive discipline at 30 months, but higher SES at 12 months was associated with lower frequency of parental punitive discipline at 30 months.

Sibling conflict models. Higher sibling conflict was associated with more concurrent daytime sleepiness and shorter nighttime sleep duration. Covariates in the model included age, sex, ethnicity, SES and family structure at five years, as well as nighttime sleep duration at 30 months. In the nighttime sleep duration model, longer nighttime sleep duration at 30 months also predicted longer nighttime sleep duration at five years. See Models 1 and 2 in Table 5 for all results.

In testing bidirectional effects (see all models in Table 6), neither 12-month nor 30-month nighttime sleep duration predicted sibling conflict at five years (12 mos age, sex, ethnicity, SES and family structure as covariates). In both models, females showed more sibling conflict at five years. Similarly, neither 12-month nor 30-month sleep dysregulation were associated with sibling conflict at five years, but being female was associated with greater sibling conflict at five years in both the 12- and 30-month model.

Twin Intra-class Correlations

Twin intra-class correlations were conducted to examine whether identical twins were more similar to each other than fraternal twins (see Table 7 for complete ICCs). Findings indicated that MZ twins were more similar on sleep dysregulation scores at both 12 (ICC = .77) and 30 (ICC = .67) months than DZ twins (12 mos ICC = .50; 30 mos ICC= .44, respectively). MZ twins were also more similar on nighttime sleep duration at five years (ICC = .95) than DZ twins (ICC = .80), as well as on daytime sleepiness scores at five years (MZ = .86; DZ = .64). Finally, MZ twins were more similar on sibling conflict scores at five years (ICC = .84) than DZ twins (ICC = .70).

Univariate ACE Models

Punitive discipline. The full univariate ACE model for parental punitive discipline at 30 months was the best fitting model for the data, $\chi^2(427) = 278.63$, AIC = -575.37. The complete punitive parental discipline model comparison table for fit statistics is provided in Table 8. Using the ACE model fit, the standardized variance components were estimated, such that the greatest proportion of the variance in punitive discipline was accounted for by the shared environmental factor ($c^2 = .86$), with the little remaining

variance accounted for by additive genetic (a^2 = .13) and non-shared environmental contributions (e^2 = .01; see Figure 2 for full model).

Sibling conflict. The full univariate ACE model for sibling conflict at five years was a good fit for the data, $\chi^2(377) = 690.35$, AIC = -63.35. However, the reduced CE model did not fit significantly worse than the full ACE model, suggesting that the CE model fit the data best, $\chi^2(378) = 693.17$, AIC = -62.83, $\Delta \chi^2 = 2.82$, p = .09. The complete sibling conflict comparison table for fit statistics is provided in Table 8. The standardized variance components for sibling conflict were estimated based on the reduced CE model. The greatest proportion of the variance in sibling conflict was accounted for by the shared environmental factor (c^2 = .74), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .26; see Figure 3 for best fitting model).

Nighttime sleep duration (12 months). The full ACE model yielded the best fit for nighttime sleep duration at 12 months, $\chi^2(534) = 1430.49$, AIC = 362.49. I tested reduced models, including AE, CE and E; however, all three reduced models fit the data significantly worse than the full ACE model for nighttime sleep duration at 12 months (see Table 9 for comparison of model fit statistics). Standardized variance components for nighttime sleep duration were estimated from the full ACE model (Figure 4), such that the greatest proportion of the variance in nighttime sleep duration at 12 months was accounted for by shared environmental factors (c^2 = .82). Nighttime sleep duration demonstrated very little heritability (additive genetic, a^2 = .14) and non-shared environmental contributions at 30 months (e^2 = .04).

Nighttime sleep duration (30 months). The full ACE model fit the data well, $\chi^2(413) = 1002.80$, AIC = 176.80. However, the CE model yielded the best fit for nighttime sleep duration at 30 months, $\chi^2(414) = 1003.29$, AIC = 175.29 $p = .48$, indicating that the CE model did not fit the data significantly worse than the full model (see Table 9 for comparison of model fit statistics). Standardized variance components for nighttime sleep duration were estimated from the CE model (Figure 5), such that the greatest proportion of the variance in nighttime sleep duration at 30 months was accounted for by the shared environmental factor ($c^2 = .83$), with very little non-shared environmental contributions ($e^2 = .17$).

Nighttime sleep duration (five years). The full ACE model yielded the best fit for nighttime sleep duration at five years, $\chi^2(372) = 836.24$, AIC = 92.24. I tested reduced models, including AE, CE and E; however, all three reduced models fit the data significantly worse than the full ACE model for nighttime sleep duration at five years (see Table 9 for comparison of model fit statistics). Standardized variance components for nighttime sleep duration were estimated from the full ACE model (Figure 6), such that the greatest proportion of the variance in nighttime sleep duration at five years was accounted for by the shared environmental factor ($c^2 = .61$). Nighttime sleep duration also demonstrated moderate heritability (additive genetic, $a^2 = .35$), but very little non-shared environmental contributions ($e^2 = .04$).

Sleep dysregulation (12 months). The full ACE model yielded the best fit for sleep dysregulation at 12 months, $\chi^2(535) = 525.96$, AIC = -544.04. I tested reduced models, including AE, CE and E; however, all three reduced models fit the data significantly worse than the full ACE model for sleep dysregulation at 12 months (see

Table 9 for comparison of model fit statistics). Standardized variance components for sleep dysregulation were estimated from the full ACE model (Figure 7), such that sleep dysregulation at 12 months was moderately heritable (additive genetic, a^2 = .46), but also showed shared environmental (c^2 = .29) and non-shared environmental contributions (e^2 = .26).

Sleep dysregulation (30 months). The full ACE model was a good fit for sleep dysregulation at 30 months, $\chi^2(448) = 117.53$, AIC = -776.47. However, the reduced AE model yielded the best fit for sleep dysregulation at 30 months, $\chi^2(448) = 118.15$, p = .43, such that the AE model did not fit the data significantly worse than the full ACE model for sleep dysregulation at 30 months (see Table 9 for comparison of model fit statistics). Standardized variance components for sleep dysregulation were estimated from the full AE model (Figure 8), such that sleep dysregulation at 30 months was highly heritable (additive genetic, a^2 = .71), as well as showing non-shared environmental contributions (e^2 = .29).

Daytime sleepiness (five years). The full ACE model yielded the best fit for the daytime sleepiness scale at five years, $\chi^2(359) = 1890.58$, AIC = 1172.58. I tested reduced models, including AE, CE and E; however, all three reduced models demonstrated significantly worse fit than the full ACE model for daytime sleepiness at five years (see Table 9 for comparison of model fit statistics). Standardized variance components for nighttime sleep duration were estimated from the full ACE model (Figure 9), such that daytime sleepiness was moderately heritable (additive genetic, a^2 = .55). Daytime sleepiness also demonstrated shared environmental contributions (c^2 = .33), with little of the variance explained by non-shared environmental contributions (e^2 = .12).

Bivariate ACE Models

Given that there was no phenotypic association between punitive parental discipline at 30 months and either of the sleep outcome variables at 30 months or five years, bivariate models were not conducted.

Sibling conflict and nighttime sleep duration. A bivariate Cholesky decomposition of sibling conflict and nighttime sleep duration at five years revealed the full ACE-ACE to be the best fitting model, after dropping the A and E contributions to the covariance between the two phenotypes ($\chi^2(675) = 1344.63$, AIC = -5.37, $p = .41$; full fit statistics in Table 10). The standardized variance components on the first phenotype (sibling conflict) showed that the greatest proportion of the variance in sibling conflict was accounted for by shared environment ($c_{11} = .58$), and the remaining variance in sibling conflict was divided between additive genetic ($a_{11} = .22$) and non-shared environmental contributions ($e_{11} = .20$). For the second phenotype (nighttime sleep duration), the standardized variance components revealed that the greatest proportion of the variance in nighttime sleep duration was also accounted for by shared environment ($c_{22} = .58$). Most of the remaining variance in nighttime sleep duration was accounted for by additive genetic factors ($a_{22} = .37$), with almost no variance in nighttime sleep duration accounted for by non-shared environmental contributions ($e_{22} = .04$). All covariance between sibling conflict and nighttime sleep duration was accounted for by shared environmental factors (shared c = 1.0), which explained 7% of the total variance in nighttime sleep duration (see Figure 10).

Sibling Conflict and Daytime Sleepiness. A bivariate Cholesky decomposition of sibling conflict and daytime sleepiness at five years revealed the CE-ACE to be the

best fitting model, dropping the A and E contributions to covariance between the two phenotypes ($\chi^2(676) = 2485.53$, AIC = 1133.31, $p = .99$; full fit statistics in Table 10). The standardized variance components on sibling conflict showed that the greatest proportion of the variance in sibling conflict was accounted for by shared environment ($c_{11} = .72$), and the remaining variance in sibling conflict was accounted for by non-shared environmental contributions ($e_{11} = .28$), as the additive genetic component (a_{11}) was dropped. For daytime sleepiness, the standardized variance path estimates revealed that greatest proportion of the variance in daytime sleepiness was accounted for by additive genetics ($a_{22} = .46$). Most of the remaining variance in nighttime sleep duration was accounted for by shared environmental factors ($c_{22} = .39$), with little variance in daytime sleepiness accounted for by non-shared environmental contributions ($e_{22} = .15$). All covariance between sibling conflict and daytime sleepiness was accounted for by shared environmental factors (shared c = 1.0), which explained 12% of the total variance in daytime sleepiness (see Figure 11 for details).

Discussion

The goal of the current study was to examine concurrent and longitudinal phenotypic and quantitative behavior genetic associations between specific familial relationships (parental punitive discipline and sibling conflict) and child sleep parameters, including nighttime sleep duration, sleep dysregulation, and daytime sleepiness. These goals were addressed with two broad aims. First, I tested concurrent and bidirectional relations between punitive discipline and child sleep parameters, as well as between sibling conflict and child sleep parameters. Second, I examined associations between punitive discipline, sibling conflict, and child sleep by testing univariate and

bivariate behavior genetic models for punitive discipline, sibling conflict, and child sleep parameters at three time points.

Regarding the first aim, no basic or bidirectional effects were detected for punitive discipline and child sleep, suggesting that punitive discipline was not associated with child sleep parameters. However, there were concurrent effects between sibling conflict and child sleep parameters. Analyses for the second main aim revealed punitive discipline and sibling conflict were influenced primarily by shared environmental factors. Sleep parameters showed varying amount of genetic and environmental influence, with these contributions fluctuating over time as predicted. In addition, associations between sibling conflict and concurrent sleep parameters were solely explained by shared environmental factors (contrary to my hypotheses), indicating that factors in the twins' common environment or home may account for associations between sibling conflict and child sleep. For example, other aspects of parenting such as parental cognitions about child sleep or level emotional availability may account for associations between sibling conflict and child sleep behavior. Additionally, differential treatment of the twins by parents may contribute to the relationship between sibling conflict and twins' sleep problems.

Phenotypic Analyses and Bidirectional Effects

Punitive discipline. Findings indicated that frequency of punitive discipline when twins were 30 months old was not associated with concurrent or future (five year) nighttime sleep duration, sleep dysregulation or daytime sleepiness. In addition, child nighttime sleep duration and sleep dysregulation at 12 months did not predict punitive discipline, suggesting that there were no bidirectional relations. These finding do not

support my hypothesis that punitive discipline would be associated with concurrent and future nighttime sleep duration and sleep dysregulation. Furthermore, these results are surprising given that previous research has demonstrated stress and conflict within the parent-child relationship (e.g., parental punitive discipline) is a risk factor for multiple physical and mental health outcomes (Laskey & Cartwright-Hatton, 2009; Trickett & Kuczynski, 1986; Stormshak et al., 2000).

While there appears to be no relationships between parental punitive discipline and child sleep parameters in the current study, there are a number of possible explanations as to why this association was not detected. First, parental punitive discipline was conceptualized as the frequency with which a number of parenting behaviors or tactics were utilized for each twin. For example, parents reported how frequently they employed verbal threatening or spanking the child as a means of disciplining each twin. From these parent reports, a composite score was created from four items in the scale that were "punitive" discipline measures. However, it is possible that creating a composite score was not an appropriate way to conceptualize parental punitive discipline. As with the examples of "verbal threatening" or "spanking," it is clear that parents who endorse threatening as a means of discipline may not necessarily also employ spanking as a form of punishment. Thus, items deemed "punitive" may not be mutually exclusive, making a composite score a poor measure of the frequency with which parents use punitive discipline.

However, utilizing a single item in the Parent Response to Child Misbehavior Scale (Holden et al., 1995) may be an equally poor way to measure how punitive discipline is related to child sleep, as using a single item to predict outcomes in regression

analyses makes it difficult to assess the reliability of a measure and is not recommended (Cohen, Cohen, West, & Aiken, 2002). Indeed, preliminary analyses testing whether the four individual punitive discipline items in the PRCM (Holden et al., 1995) showed that no individual punitive discipline items predicted concurrent or future child nighttime sleep duration, sleep dysregulation or daytime sleepiness. Thus, it is possible that no effects of punitive discipline on child sleep parameters were detected because it was difficult to conceptualize and use the PRCM as an accurate predictor of parental punitive discipline.

It is also possible that there were no relations between punitive discipline and child sleep parameters because there is a particular threshold at which punitive discipline has a negative effect on child health outcomes like sleep. In terms of what this threshold may be, it is unclear; however, a meta-analysis and theoretical review by Gershoff (2002) suggested that there may be a continuum of parental discipline with parenting behaviors like ignoring or threatening falling on the "mild" end of the punitive discipline spectrum and behaviors like corporal (physical) punishment representing more intense and harmful parental punitive punishment. If a threshold for negative child outcomes exists on this hypothetical punitive discipline scale, we would expect that consistently high levels of parental punitive discipline would reach this proposed threshold and have a negative influence on child outcomes like sleep. Furthermore, identifying a threshold for parental punitive discipline may inform researchers and parents at which level and frequency certain parental discipline behaviors are harmful for child developmental outcomes.

Finally, it is possible that no significant relations between punitive discipline and child sleep were detected because a single facet of punitive discipline was measured in

the current study: frequency of punishment. As such, no significant main effects of punitive discipline may have been detected because parents in the current study did not use discipline behaviors frequently enough for these punitive discipline behaviors to have a negative influence on child nighttime sleep duration, sleep dysregulation or daytime sleepiness. In addition, the current study recorded a single, retrospective parent-report of punitive discipline, which has been noted as a limitation when defining and measuring parental punitive discipline in past studies (Gershoff, 2002). In fact, the type of punitive punishment used has differential effects on subsequent child behavior (LaVoie, 1974), suggesting that the current study may have found effects of parental punitive discipline if the type of punitive discipline used by each caregiver been assessed. Additionally, recent studies have effectively employed daily diary methods to measure parent- and child-reported emotional and behavioral outcomes (Almeida, 2005; Bates et al., 2002; Sandstrom & Cillessen, 2003). If researchers are able to successfully utilize daily diaries to assess parent and child behaviors, this methodology may help reduce reporter bias and better capture measures of interest like parental punitive discipline. Thus, future studies should consider using daily paper or electronic diaries to more accurately measure parenting behaviors as well as other aspects of home environment, such as child sleep behavior or sibling conflict.

Secondary punitive parenting findings. As noted previously, punitive discipline was not associated with concurrent nighttime sleep duration and dysregulation or future sleep duration and daytime sleepiness. Despite this, numerous covariates in these models were significant. Nighttime sleep duration and sleep dysregulation at 12 months were positively associated with nighttime sleep duration and sleep dysregulation at 30 months,

respectively. In addition, there was a positive association between nighttime sleep duration at 30 months and five years in the punitive discipline model. Finally, age was predictive of shorter sleep duration at 30 months, and individuals who were Latino demonstrated shorter sleep duration at 30 months. Higher SES at 30 months was also associated with longer nighttime sleep duration at 30 months, although no interaction effects (between punitive discipline and demographic variables) were detected in these analyses. These findings indicate that early sleep duration and regulation, in addition to demographic factors, likely influenced later child sleep duration and dysregulation.

In addition, nighttime sleep duration and sleep dysregulation at 12 months were not associated with parental punitive discipline at 30 months, which failed to support my hypothesis (regarding bidirectional effects) that shorter nighttime sleep duration and greater sleep dysregulation at 12 months would be related to higher frequency of parental punitive discipline. However, higher SES at 12 months was also associated with more frequent punitive discipline at 30 months, although no interaction effects (between punitive discipline and demographic variables) were detected. These results suggest that more distal demographic or family-level variables (e.g., SES) may impact parenting practices in addition to child sleep behaviors. Although significant main effects of demographic variables and sleep parameters were not initially predicted, the associations highlighted follow the expected direction of effects.

Sibling conflict. Given that studies have shown conflict and negative sibling relations are linked to poor emotional and behavioral outcomes in childhood (Bekkhus et al., 2011; Lockwood et al., 2001; Stormshak et al., 1996), I tested whether sibling conflict was associated with three child sleep parameters. In contrast to the punitive discipline

findings, sibling conflict at five years *was* associated with concurrent nighttime sleep duration and daytime sleepiness. Specifically, greater sibling conflict was positively associated with child daytime sleepiness at five years and negatively related to nighttime sleep duration at five years. These results support my hypothesis that greater conflict in sibling relationships would be associated with shorter nighttime sleep duration and increased daytime sleepiness.

There were also numerous (nonsignificant) findings that did not support my hypotheses. In particular, neither nighttime sleep duration nor sleep dysregulation at 12 and 30 months predicted later sibling conflict, which was initially hypothesized. However, within these models, nighttime sleep duration at 30 months was predictive of nighttime sleep duration at five years, demonstrating that sleep duration may be stable across time. One potential reason these relationships were not significant is sleep problems or disruptions at 12 and 30 months of age may be too distal from sibling conflict measured when twins are five years old, such that I was not able to detect a relationship between sleep problems at 12 and 30 months and sibling conflict years later. In fact, sleep duration at five years was only associated with concurrent sibling conflict at $p = .05$, suggesting the relationship between sibling conflict and child sleep duration may not be very strong in this particular sample of twins.

Although no significant relations between sibling conflict and child sleep over time were detected, future studies should test bidirectional associations between sibling conflict and child sleep using a longitudinal study design. It is possible that a positive feedback loop exists between level of sibling conflict and child daytime sleepiness, such that greater sibling conflict leads to greater daytime sleepiness which in turn increases the

likelihood of sibling conflict occurring between twins. Likewise, a negative feedback loop may exist between level of sibling conflict and child nighttime sleep duration at five years, such that greater sibling conflict may lead to shortened nighttime sleep duration, which may then increase the level or likelihood of sibling conflict occurring between twins. Thus, future studies should examine bidirectional relations between sibling conflict and child sleep behavior to clarify whether sibling conflict leads to child sleep problems or child sleep problems precede sibling conflict.

Secondary sibling conflict findings. Additionally, sex was a significant covariate in models using nighttime sleep duration and sleep dysregulation to predict later sibling conflict, with results indicating that females demonstrated greater sibling conflict at five years than males. As with the punitive discipline models, the results showing that female twins displayed greater sibling conflict were surprising and unexpected, as numerous studies have reported that males show greater aggression and initiate more conflict than girls in early childhood (see Martin & Ross, 2005; Loeber & Hay, 1997). However, at least once study by Martin and Ross (2005) found that parents reported more aggression towards and from preschool female children, suggesting that parents may be more sensitive to female aggression or conflict in childhood, possibly because aggression is typically less socially acceptable for females compared to male in childhood. Loeber and Hay (1997) also noted that studies have shown all-female groups of children show more conflict than same-age, all-male groups when children are 12 and 30 months of age, indicating that females may in fact show more sibling conflict than males, but perhaps only in same-sex twin dyads or under specific circumstances (e.g., in larger groups). There does not appear to be differences between same-sex female twins and same-sex

male twins or opposite-sex DZ twins on sibling conflict levels in the current sample, but these differences may only occur under specific circumstances as previously noted.

Univariate Behavior Genetic Analyses

Parental punitive discipline. Given research indicating that the greatest proportion of the variance in parenting is accounted for by shared environmental factors (Deater-Deckard, 2000; Losoya et al., 1997; Oliver et al., 2014), I hypothesized that the largest proportion of the variance in punitive discipline at 30 months would be accounted for by shared environmental factors, followed by additive genetic factors. Results indicated that most of the variance in punitive discipline was accounted for by the shared environmental factor, with the little remaining variance accounted for by additive genetic factors, supporting my hypothesis (Deater-Deckard, 2000; Losoya et al., 1997).

One explanation as to why parental punitive discipline may be have been primarily accounted for by shared environmental factors is that parents may use similar frequency of discipline with twins (regardless of zygosity) given the developmental period and age at which the assessment was taken. Indeed, Loeber and Hay (1997) argue that particularly in toddlerhood and during the preschool and school years, punitive discipline is more common, possibly as a way to give children structure and shape socially appropriate behavior. Thus, parents may report similar frequency of punitive discipline as a result of the age or developmental period in which the twins were measured.

Sibling relationships. A small amount of literature has suggested that sibling conflict contains both genetic and environmental influences across early and middle childhood (Lemery & Goldsmith, 2002). However, I found that the greatest proportion of

the variance in sibling conflict at five years was accounted for shared environmental factors, with the remaining variance accounted for by non-shared environmental influences. This finding did not support my hypothesis that the largest proportion of the variance in the sibling conflict would be accounted for by additive genetic factors.

Although my hypothesis regarding sibling conflict was not supported, there are alternative explanations as to why shared environmental factors primarily accounted for the variance in sibling conflict. The previously mentioned study examined genetic and environmental contributions to sibling conflict with twins ranging from three to eight years old. However, the current study measured twins in a much smaller age range (four to five years of age). Given that heritability of specific traits and behaviors fluctuate over time (Plomin et al., 2013), variability in the age at which twins were assessed in the current study compared to Lemery and Goldsmith (2002) may explain differences in genetic and environmental contributions that were detected.

There are also numerous factors in the twins' shared environment that may have influenced sibling conflict. As previously noted, twins being in close proximity to one another, sharing a room or bed, or overall household chaos may act as factors in the twins' shared environment that create or increase sibling conflict. In addition, parents may rate twins' more similarly on sibling conflict, because it may be difficult for parents to determine which twin instigates conflict or may create more conflict. Overall, further research is necessary to understand genetic and environmental contributions to sibling conflict as almost no studies have examined this construct.

Nighttime sleep duration. I estimated genetic and environmental contributions to child nighttime sleep duration and found that nighttime sleep duration fluctuated over

time, with nighttime sleep duration at 12 months showing mostly a shared environmental contribution and small additive genetic influence. At 30 months, nighttime sleep duration showed *no* additive genetic influence and a high shared environmental contribution. At five years, nighttime sleep duration showed a greater additive genetic contribution (than at 12 months) with most of the variance in sleep duration still accounted for by shared environmental factors. My initial hypothesis that the greatest proportion of the variance in nighttime sleep duration would be accounted for by additive genetic and shared environmental factors was supported, although additive genetic factors played a lesser role compared to shared environmental factors. Compared to previous studies showing that almost 70% of the variance in parent-reported nighttime sleep duration was accounted for by additive genetic factors (e.g., Gregory et al., 2006), nighttime sleep duration in the current study showed far less genetic influence. However, Gregory et al.'s study (2006) included 8-year-old twins, at which age we would expect greater heritability of sleep duration, given that the heritability to various health behaviors like sleep increase with age (see Plomin et al., 2013). Thus, my expectation that additive genetic contributions to nighttime sleep duration would increase over time was supported overall, as genetic influence of nighttime sleep duration greatly increased at the five-year assessment from the 12-month and 30-month assessments of nighttime sleep duration.

Although my hypotheses were not fully supported, this is not entirely surprising as some past studies have shown that parent-reported nighttime sleep duration shows considerable shared environmental contributions (e.g., Barclay & Gregory, 2013; Brescianini et al., 2001). It is also possible that shared environmental factors played a prominent role in nighttime sleep duration at all three assessments due to the fact that

nighttime sleep duration was subjectively reported by parents. If parents perceived twins' sleep patterns to be similar (regardless of zygosity), parents may have also reported similar or identical nighttime sleep durations for each twin. This similarity in reporting would explain the high shared environmental contribution to nighttime sleep duration at 12 months, 30 months, and five years. It is also possible that given the developmental period in which nighttime sleep was measured (early childhood and toddlerhood), children generally show similar levels of nighttime sleep duration, regardless of twin or singleton status. This, too, would explain why additive genetic influence on nighttime sleep duration at 12 and 30 months was relatively low in the current study (only increasing at the five-year assessment).

Sleep dysregulation. I found that most of the variance in sleep dysregulation at 12 months was accounted for by additive genetic factors, with moderate shared and nonshared environmental contributions. At 30 months, almost all of the variance in sleep dysregulation was accounted for by additive genetic factors, with the remaining variance due to non-shared environmental influences. These findings support my hypotheses that most variance in sleep dyregulation would be accounted for by additive genetic factors and shared environmental factors. Furthermore, these findings support my hypothesis that additive genetic contributions to sleep dyregulation would increase over time, and that nighttime sleep duration and sleep dysregulation would show differing amounts of genetic influence. In addition to supporting my hypotheses, these findings bolster past research that has shown sleep behavior is highly heritable and has significant additive genetic contributions in childhood and adolescence (Ambrosius et al., 2008; Gregory et al., 2009). In addition, the findings regarding sleep dysregulation confirm research that

suggests genetic and/or environmental contributions to distinct sleep parameters may vary (see Barclay & Gregory, 2013), and the heritability of many behaviors such as sleep increase over time (Plomin et al., 2013).

Daytime sleepiness. In terms of daytime sleepiness, only one univariate model was conducted, given that child daytime sleepiness was only measured at the five-year assessment. The univariate model for daytime sleepiness showed that the greatest proportion of the variance in daytime sleepiness was accounted for by additive genetic factors, with almost all of the remaining variance accounted for by shared environmental factors. These results support my hypothesis that most variance in daytime sleepiness would be accounted for by additive genetic factors and shared environmental factors. Furthermore, these findings support my hypothesis that daytime sleepiness would show differing amounts of genetic influence compared to nighttime sleep duration and sleep dysregulation, given that some aspects of sleep may be more heritable than others (see Barclay & Gregory, 2013). These results also roughly support prior findings on child daytime sleepiness that suggest almost equal genetic and environmental influences on daytime sleepiness, with little impact of the non-shared environment (Gregory et al., 2006). As the non-shared environment represents any experiences not shared by the twins, it is possible that at five years, there are few daytime and nighttime experiences that are unique to the twins. Evidence supporting fewer non-shared experience for the twins at five years is demonstrated in the fact that approximately 75% of the twins had not entered kindergarten when daytime sleepiness was assessed at five years, and just over 50% of the sample utilized in-home child care at five years. Furthermore, of parents who reported that both twins participated in recreations activities, approximately 80% of

twins were placed in the same activity or on the same team. These statistics suggest that the twins in the current sample perhaps spent almost all day and night together in the same home at five years, leaving little opportunity for unique experiences like ones that twins may have in school or extra curricular activities.

Bivariate Analyses

Punitive discipline and child sleep. I did not conduct a bivariate behavior genetic model using punitive discipline and child nighttime sleep duration, sleep dysregulation or daytime sleepiness as they were not significantly associated in phenotypic analysese. Thus, my hypothesis was not supported and my models could not test whether the greatest proportion of covariance in the association between punitive parenting and child sleep parameters was accounted for by shared environmental factors.

One possible explanation for the lack of association between punitive discipline and child sleep parameters was discussed earlier (related to measurement of punitive discipline), and this explanation applies to the bivariate analyses as well. In addition, lack of association between punitive discipline and childhood sleep concurrently or prospectively may be explained by a third variable not considered in this study, such as child effortful control. Effortful control consists of willingly or unwillingly controlling behavior, attention and cognition, and can be considered an aspect of self-regulation (Eisenberg, Hofer, Sulik, & Spinrad, 2014; Eisenberg, Smith, & Spinrad, 2011). Effortful control has been associated with a number of cognitive and physical health outcomes (e.g., eating) and has been shown to increase with age, showing sharp increases in toddlerhood and at preschool age (Eisenberg, Smith & Spinrad, 2011; Graziano, Kelleher, Calkins, Keane, & Brian, 2013). If twins in the current study demonstrated

greater effortful control at 30 month and five years, it is possible caregivers may have reported lower frequency of punitive punishment and fewer sleep problems, given twins' ability to better regulate their behaviors. Indeed, bivariate correlations at the 30-month assessment show greater effortful control was independently associated with lower reported punitive punishment and greater nighttime sleep duration. Greater effortful control at five years was also related to greater nighttime sleep duration at five years but not daytime sleepiness. These correlations suggest that effortful control may play a role in the relationship (or lack thereof) between punitive discipline and child sleep, with greater child effortful control leading to less punitive discipline and perhaps more regulated sleep or bedtime behaviors. Future studies should test whether effortful control and self-regulation relate to critical child sleep outcomes or moderate and mediate associations between parental discipline and child sleep.

Sibling conflict and child sleep parameters. I tested whether the greatest proportion of the covariance in the association between sibling conflict and child sleep parameters would be accounted for by additive genetic factors, and the results did not support my hypothesis, as they showed that the covariance in the association between sibling conflict at five years and concurrent nighttime sleep duration was entirely attributed to shared environmental factors. This finding suggests that some aspect(s) in the twins' shared environment accounts for the association between sibling conflict and nighttime sleep duration. Similarly, I tested whether most of the covariance in the association between sibling conflict and daytime sleepiness at five years would be accounted for by additive genetic factors. The findings regarding this bivariate model also failed to support my hypothesis, as they showed that the covariance in the

association between sibling conflict at five years and concurrent daytime sleepiness was entirely accounted for by shared environmental influences.

There are possible explanations as to why the shared environment fully accounted for the association between sibling conflict and child sleep. First, it is possible that negative emotions associated with sibling conflict (e.g., anger, anxiety) better account for the significant association between sibling conflict and child sleep parameters. If twins experienced highly negative emotions shortly before bedtime or throughout the day as a result of sibling conflict, then it is possible that these negative emotions may have prevented children from falling sleep or staying asleep, which impacts nighttime sleep duration and daytime sleepiness. Indeed, theory suggests that negative emotions do play a role in sleep behaviors, as Dahl (1996) has suggested bidirectional relations between sleep quality and emotion regulation. Some research supports Dalh's (1996) theory, with at least one study showing increased emotional intensity and lower emotional regulation before bedtime predicted shorter sleep duration and greater sleep disturbances in middle childhood (including sleep duration and daytime sleepiness; El-Sheikh & Buckhalt, 2005). In addition, a recent review by Vandekerckhove & Cluydts (2010) highlighted the bidirectional associations between daily emotional events and sleep quality, noting a number of studies linking greater daily stress, anxiety and depression to changes (increases and decreases) in REM sleep, increased sleep latency and reduced sleep duration. Thus, negative emotions associated with sibling conflict may be a shared environmental factor that accounts for some of the covariance between sibling conflict and child sleep problems, with high negative emotional states or emotion regulation

problems acting as a mechanism that connects sibling conflict and normative child sleep problems at age five.

In addition, parenting may serve as shared environmental component that accounts for associations between sibling conflict and child sleep parameters. Punitive discipline was tested as a predictor of sleep outcomes in the current study, but it is possible that specific qualities of parenting such as warmth, control or permissiveness (not measured in the current study) may moderate or mediate associations between sibling conflict and twins' sleep. Punitive discipline at 30 months was positively correlated with sibling conflict at five years in the sample (see Table 2), suggesting that parenting may impact sibling conflict and vice versa. As noted earlier, various aspects of parenting such as maternal hostility or over-involvement have been associated with child sleep problems longitudinally (Rhoades et al., 2012; Sheridan et al., 2013), indicating that specific components of parenting are linked with child sleep behaviors. Although no current studies examine parenting, sibling conflict and child sleep together, findings from the current study and prior research suggest that parenting may be a key shared environmental factor that accounts for some of the covariance between sibling conflict and child sleep parameters. Future studies should test whether multiple facets of parenting moderate or mediate links between sibling conflict and child sleep.

Finally, a third variable in the twins' environment may have influenced the association between sibling conflict and child sleep parameters. For example, general stress in the home environment or household chaos may have contributed to the relationship between sibling conflict and childhood sleep. If twins experience greater household chaos and disorganization, then overall household stress may also increase the

likelihood of sibling conflict (or increase existing sibling conflict levels), as well as increase sleep problems. Indeed, prior research indicates that household chaos is linked to familial relationships and child behavioral outcomes, with greater household chaos independently predicting child problem behaviors (Coldwell, Pike & Dunn, 2006). In the same study, household chaos also exacerbated the association between poor parenting quality and child behavioral problems (Coldwell et al., 2006). In a related study, lower household chaos was linked with better sibling relationships quality in middle childhood, with both positive and harsh parenting moderating this association (Kretschmer & Pike, 2009). Household chaos or disruptions surrounding bedtime have also been linked to greater likelihood of sleep disruptions during the night in middle childhood (Fiese et al., 2007). As such, household chaos or stress may be an additional factor in the twins' shared environment that contributes to both greater sibling conflict and shorter nighttime sleep duration and greater daytime sleepiness (and potentially parenting), thereby accounting for the covariance in the association between sibling conflict and child sleep parameters at five years.

Future Directions

Although longitudinal and bidirectional associations were tested, it is also critical to understand and test whether other person- or family-level variables may influence associations between parental punitive discipline, sibling conflict and child sleep parameters over time. Given that person-level variables, such as emotionality, emotional regulation or effortful control, may serve as mechanisms or factors accounting for the association between parenting and child sleep, as well as between sibling conflict and child sleep problems, future studies should test whether difficult temperament, emotional

regulation, emotionality and effortful control serve as person-level factors that moderate the link between punitive discipline, sibling conflict, and child sleep. A few past studies suggest that child sleep duration and efficiency are linked to temperament types and emotional processing in middle and late childhood (Atkinson et al., 1995; Soffer-Dudek et al., 2011; Weissbluth, 1984), suggesting that examining emotionality or temperament in a sibling conflict and sleep framework may be useful. Likewise, Lemery and Goldsmith (2002) have shown associations between sibling conflict and temperament, such that sibling relationships and temperament have shared genetic influences in twins (middle to late childhood). If difficult temperament or difficulty with regulating emotion (or behavior) moderates the association between punitive discipline and sleep, as well as between sibling conflict and child sleep, I would expect even greater sibling conflict and child sleep problems and perhaps more punitive discipline. In addition, if difficult temperament or emotion regulation problems play a role in the association between sibling conflict and child sleep, emotion regulation or difficult temperament may potentially share some genetic and environmental covariance with punitive discipline, sibling conflict and child sleep parameters in a multivariate behavior genetic model.

Furthermore, given that a stressful family environment or household chaos may also account for the association between parental punitive discipline, sibling conflict and child sleep parameters over time, family-level variables should be tested as moderators or mediators of these relationships. As hypothesized above, household chaos and disorganization may account for the association between parenting, sibling conflict, and child sleep problems (Coldwell et al., 2006; Fiese et al., 2007; Kretschmer & Pike, 2009). If so, household chaos or other significant stressors in the family environment that are

shared by twins may moderate the link between punitive discipline and child sleep or between sibling conflict and child sleep (e.g., Kretschmer & Pike, 2009). Thus, I would expect greater frequency of punitive discipline, greater sibling conflict, and greater child sleep problems if household chaos or disorganization does moderate these associations, with household chaos or disorganization accounting for a proportion of the shared environmental influence in the association between sibling conflict and child sleep.

Overall, future studies should examine other person- and family-level factors that may account for associations between sibling conflict and child sleep parameters across time, as well as moderate these associations. In addition, future studies should test whether person- and family-level factors may moderate relations between punitive discipline and child sleep parameters over time. By examining potential moderators between family relationships and child sleep, researchers should be able to identify possible points of intervention for early sleep behaviors and maximize and improve child sleep.

Conclusion

The purpose of the current master's thesis was to determine whether there was a phenotypic and quantitative behavior genetic association between punitive parental discipline and sleep behavior, as well as between sibling conflict and child sleep behavior in a longitudinal, population-based sample of twins. The results indicated that there were no associations between punitive discipline and child sleep parameters, whereas sibling conflict at five years was associated with concurrent child nighttime sleep duration and daytime sleepiness. Furthermore, shared environmental influences, rather than additive genetic factors, solely accounted for the covariance in the associations between sibling

conflict and both nighttime sleep duration and daytime sleepiness. These findings suggest that a component twins' common environment such as household chaos, family stress or parenting likely accounts for the shared environmental contribution to the association between sibling conflict and both child sleep parameters at five years. Future studies should aim to test person- and family-level factors that may serve as moderators 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 intervene and reduce sleep problems, as well as how to best improve child sleep and other child developmental outcomes.

In terms of how to best intervene and improve child sleep, these findings are critical and may help inform public policy and health practitioners by helping providing parents with information about specific, changeable factors (environmental or behavioral) that may negatively impact their twins' sleep patterns. For example, the current study demonstrates that sibling conflict plays an important role in child sleep duration and daytime sleepiness. As such, 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 in which to reduce sibling conflict. In addition, given that the findings indicate sleep duration and daytime sleepiness may also increase sibling conflict, it is important that health practitioners and researchers also educate parents about parenting practices related to child bedtime routines. If parents are able to foster optimal sleep duration and reduce daytime sleepiness in early childhood by changing everyday bedtime or waking routines, it is possible that child sleep behavior would improve and sibling conflict would decrease. 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 (like punitive discipline and sibling conflict) and child sleep.

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APPENDIX A

TABLES

Table 1

Raw means, standard deviations, ranges, skewness, and kurtosis

Scale	N	*M*	*SD*	Min	Max	Skew	Kurtosis
Punitive parenting (parent report, 30 mos)	486	1.84	0.64	1	4.25	0.99	1.14
Sibling conflict (parent report, 5 yrs)	400	2.77	0.73	1	5	0.23	0.08
Nighttime Sleep Duration (parent report, 12 mos)	565	10.15	1.5	5	12	-1.04	1.13
Nighttime Sleep Duration (parent report, 30 mos)	476	10.41	1.14	6	12	-0.94	0.74
Nighttime Sleep Duration (parent report, 5 yrs)	395	10.25	1.03	6.75	12.5	-0.5	0.25
Sleep Dysregulation (parent report, 12 mos)	570	0.36	0.44	0	2	1.29	1.21
Sleep Dysregulation (parent report, 30 mos)	516	0.63	0.3	0	1.8	1.25	1.35
Daytime Sleepiness	400	13.82	4.42	6	30	0.76	0.31

Note. Punitive discipline and sibling conflict scores are mean composite scores, and daytime sleepiness scores are sum scores of 7 items in the CSHQ daytime sleepiness scale. Nighttime sleep duration is the parent-reported raw number of hours of sleep per night, estimated individually for each twin. Sleep dysregulation is the mean of a five-item dysregulation scale in the ITSEA questionnaire, as reported by primary caregiver for each twin.

Table 2

Bivariate Correlations between sleep parameters (sleep dysregulation, nighttime sleep duration, and daytime sleepiness), sibling conflict, and punitive parenting

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Punitive Discipline (30 mos)	--																	
2. Sibling Conflict (5 yrs)	.12*	--																
3. Sleep Dysregulation (12 mos)	0.06	0.06	--															
4. Nighttime Sleep Duration (12 mos)	-0.03	0.01	-.41**	--														
5. Sleep Dysregulation (30 mos)	.10*	0.06	.35**	-.24**	--													
6. Nighttime Sleep Duration (30 mos)	-0.01	-.13*	-.19**	.42**	-.32**	--												
7. Nighttime Sleep Duration (5 yrs)	-0.09	-.18**	-0.03	.20**	-.14**	.36**	--											
8. Daytime Sleepiness (5 yrs)	0.06	.26**	0.09	-0.11	0.01	-.13*	-.18**	--										
9. Age (12 mos)	0.05	0.07	0.04	-0.05	0.04	-.10*	0.07	0.09	--									
10. Age (30 mos)	-0.09	0.06	-0.01	0.03	0.06	-.11*	-0.09	0.07	0.02	--								
11. Age (5 yrs)	0.01	-0.05	-0.07	0.04	0	0.01	0.03	-0.04	-0.03	.15*	--							
12. Ethnicity	0.02	0.01	.17**	-.28**	.11*	-.19**	-.15*	.12*	-0.05	.09*	-.13**	--						
13. SES (12 mos)	-0.12	-0.13	-0.08	.19**	0.12	.30**	0.05	-0.08	-0.06	-0.03	0.04	-0.3	--					
14. SES (30 mos)	-.13**	.10*	-0.07	.14**	-.12**	.29**	0.03	-0.08	-0.06	-0.03	0.02	-.29**	.97**	--				
15. SES (5 yrs)	.24**	-.18**	-0.08	.16**	-.17**	.27**	.13**	-0.98	0.08	0.01	-0.02	-.25**	.89**	.86**	--			
16. Family Structure (12 mos)	0.04	0.04	0.06	-.22**	0.03	-.19**	-.13*	0.1	0	0.04	-0.01	.26**	-.49**	-.40**	-.31**	--		
17. Family Structure (30 mos)	0.02	0.04	0.08	-.20**	0.02	-.19**	-.13*	.12*	-0.03	0.03	0.02	.26**	-.44**	-.38**	-.32**	.94**	--	
18. Family Structure (5 yrs)	-.11*	0.03	0.08	-.25**	-0.01	-0.09	-.15**	0.07	-0.06	0.04	-0.02	.21**	-.312**	-.21**	-.28**	.75**	.78**	--

Note. One asterisk (*) indicates that the bivariate correlation was significant at the $p < .05$ level. Two asterisks (**) indicate that the bivariate correlation was significant at the $p < .01$ or $p < .001$ level.

Table 3

Mixed model regression analyses for parental punitive discipline at 30 months predicting nighttime sleep duration (30 mos and 5 years), sleep dysregulation (30 mos), and daytime sleepiness (5 years)

	Nighttime Sleep Duration (30 mos)				Sleep Dysregulation (30 mos)		
Predictors	B	SE	Sig.		B	SE	Sig.
Model 1				**Model 2**			
Constant	8.81	0.88	.001***	Constant	0.88	0.18	.001***
Sex	0.02	0.08	0.77	Sex	-0.01	0.03	0.89
Age (30 mos)	-0.07	0.06	0.27	Age (30 mos)	-0.03	0.01	0.05*
SES (30 mos)	0.3	0.1	.003**	SES (30 mos)	-0.03	0.02	0.26
Ethnicity	-0.33	0.15	.04*	Ethnicity	0.07	0.04	0.07
Family Structure (30 mos)	-0.16	0.21	0.46	Family Structure (30 mos)	-0.03	0.05	0.57
Nighttime Sleep Duration (12 mos)	0.25	0.04	.001***	Sleep Dysregulation (12 mos)	0.23	0.03	.001***
Parental Punitive Discipline (30 mos)	0.02	0.1	0.85	Parental Punitive Discipline (30 mos)	0.03	0.02	0.28

Note. B values are unstandardized estimates. One asterisk (*) indicates that the bivariate correlation was significant at $p < .05$. Two asterisks (**) indicate the estimate was significant at $p < .01$, and three asterisks (***) indicate $p < .001$ level.

Table 3 (continued)

Mixed model regression analyses for parental punitive discipline at 30 months predicting nighttime sleep duration (30 mos and 5 years), sleep dysregulation (30 mos), and daytime sleepiness (5 years)

	Nighttime Sleep Duration (5 yrs)				Daytime Sleepiness (5 yrs)		
Predictors	B	SE	Sig.		B	SE	Sig.
Model 3				**Model 4**			
Constant	11.54	1.29	0.001***	Constant	6.85	4.12	0.1
Sex	-0.12	0.08	0.16	Sex	-0.1	0.33	0.75
Age (30 mo)	-1.34	0.45	0.008***	Age (30 mo)	1.7	1.58	0.28
SES (30 mo)	0.01	0.1	0.1	SES (30 mo)	0.07	0.39	0.86
Ethnicity	0.13	0.16	0.42	Ethnicity	0.19	0.61	0.75
Family Structure (30 mo)	-0.11	0.23	0.63	Family Structure (30 mo)	0.65	0.84	0.44
Nighttime Sleep Duration (30 mo)	0.21	0.05	.001***	Parental Punitive Discipline (30 mo)	0.34	0.4	0.4
Parental Punitive Discipline (30 mo)	-0.09	0.1	0.35				

Note. B values are unstandardized estimates. One asterisk (*) indicates that the bivariate correlation was significant at $p < .05$. Two asterisks (**) indicate the estimate was significant at $p < .01$, and three asterisks (***) indicate $p < .001$ level.

Table 4

Mixed model regression analyses for child sleep parameters at 12 months predicting parental punitive discipline at 30 months (testing bidirectional relations)

Predictors	Parental Punitive Discipline (30 mos)			Predictors	Parental Punitive Punishment (30 mos)		
	B	SE	Sig.		B	SE	Sig.
Model 1				**Model 2**			
Constant	1.89	.49	.001***	Constant	1.95	.49	.001***
Sex	-.03	.02	.25	Sex	-.04	.03	.15
Age (12 mos)	-.01	.04	.99	Age (12 mos)	-.01	.04	.91
SES (12 mos)	-.13	.06	.05*	SES (12 mos)	-.13	.06	.05*
Ethnicity	-.01	.10	.99	Ethnicity	-.01	.10	.90
Family Structure (12 mos)	.03	.13	.80	Family Structure (12 mos)	.04	.13	.77
Nighttime Sleep Duration (12 mos)	.01	.02	.98	Sleep Dysregulation (12 mos)	.04	.04	.34

Note. B values are unstandardized estimates. One asterisk (*) indicates that the bivariate correlation was significant at $p <= .05$. Three asterisks (***) indicate significance at the $p < .001$ level.

Table 5

Mixed model regression analyses for sibling conflict at five years predicting concurrent nighttime sleep duration and daytime sleepiness

	Nighttime Sleep Duration (5 yrs)				Daytime Sleepiness (5 yrs)		
Predictors	B	SE	Sig.	Predictors	B	SE	Sig.
Model 1				**Model 2**			
Constant	8.80	1.14	.001***	Constant	14.12	2.93	.001***
Sex	-.05	.08	.49	Sex	-9.00	.31	.78
Age (5 yrs)	-.18	.20	.37	Age (5 yrs)	-.55	.60	.36
SES (5 yrs)	-.01	.15	.95	SES (5 yrs)	.65	.50	.19
Ethnicity	.06	.09	.54	Ethnicity	-.23	.32	.47
Family Structure (5 yrs)	-.15	.20	.43	Family Structure (5 yrs)	.20	.62	.75
Nighttime Sleep Duration (30 mos)	.23	.05	.001***	Sibling Conflict (5 yrs)	1.02	.27	.001***
Sibling Conflict (5 yrs)	-.14	.07	.05*				

Note. B values are unstandardized estimates. One asterisk (*) indicates that the bivariate correlation was significant at $p < .05$. Three asterisks (***) indicate significance at the $p < .001$ level.

Table 6

Mixed model regression analyses for child sleep parameters at 12 and 30 months predicting sibling conflict at five years (testing bidirectional relations)

Predictors	Sibling Conflict (5 yr) B	SE	Sig.	Predictors	Sibling Conflict (5 yr) B	SE	Sig.
Model 1				**Model 2**			
Constant	1.75	.70	.01**	Constant	1.83	.70	.01***
Sex	.13	.06	.03*	Sex	.13	.06	.03*
Age (12 mos)	.08	.06	.18	Age (12 mos)	.07	.06	.21
SES (12 mos)	-.10	.08	.2	SES (12 mos)	-.09	.08	.22
Ethnicity	-.02	.12	.86	Ethnicity	-.05	.12	.66
Family Structure (12 mos)	.05	.16	.74	Family Structure (12 mos)	.04	.16	.79
Nighttime Sleep Duration (12 mos)	.01	.03	.79	Sleep Dysregulation (12 mos)	.11	.08	.16

Predictors	B	SE	Sig.	Predictors	B	SE	Sig.
Model 3				**Model 4**			
Constant	1.76	.83	.04*	Constant	1.73	.81	.04*
Sex	.15	.06	.02*	Sex	.14	.06	.02*
Age (30 mos)	.36	.32	.26	Age (30 mos)	.37	.31	.25
SES (30 mos)	-.12	.08	.15	SES (30 mos)	-.10	.08	.18
Ethnicity	-.06	.13	.62	Ethnicity	-.06	.12	.60
Family Structure (30 mos)	-.18	.19	.34	Family Structure (30 mos)	-.09	.17	.59
Nighttime Sleep Duration (30 mos)	-.05	.05	.27	Sleep Dysregulation (30 mos)	-.16	.12	.21

Note. B values are unstandardized estimates. One asterisk (*) indicates that the bivariate correlation was significant at $p < .05$. Two asterisks (**) indicate the estimate was significant at $p < .01$.

Table 7

Twin intraclass correlations to show MZ and DZ twin similarity on predictor and outcome variables

Sleep and Family Variables	MZ	Same-sex DZ	Opposite-sex DZ
Sleep Dysregulation (12 mos)	.77	.46	.54
Nighttime Sleep Duration (12 mos)	.96	.92	.87
Sleep Dysregulation (30 mos)	.67	.49	.39
Nighttime Sleep Duration (30 mos)	.86	.87	.85
Nighttime Sleep Duration (5 yrs)	.95	.79	.80
Daytime Sleepiness (5 years)	.86	.57	.70
Parental Punitive Discipline (30 mos)	.99	.93	.92
Sibling Conflict (5 years)	.84	.61	.78

Note. Heritability estimates were calculated assuming full ACE models, although reduced models are reported in Results section.

Table 8

Univariate ACE model fit statistics for family predictor and outcome variables, including reduced models

Family Variables	Model	-2LL	*df*	AIC	Δ *df*	Δ χ^2	*p*
Punitive Discipline	**ACE**	**278.63**	**427**	**-575.37**	--	--	--
(30 mos)	AE	439.42	428	-416.58	1	160.79	0
	CE	350.64	428	-505.36	1	5.81	0
	E	820.2	429	-37.8	2	541.57	0
Sibling Conflict	ACE	690.35	377	-63.35	--	--	--
(5 years)	AE	718.79	378	-37.21	1	28.44	0
	CE	**693.17**	**378**	**-62.83**	**1**	**2.82**	**0.09**
	E	841.43	379	83.43	2	151.07	0

Note. Bolded models denote the best fitting models for each predictor and outcome variable. 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. Δ χ^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 9

Univariate ACE model fit statistics for sleep predictor and outcome variables, including reduced models

Sleep Variables	Model	-2LL	*df*	AIC	Δ *df*	Δ χ^2	*p*
Nighttime Sleep Duration (12 mos)	**ACE**	**1430.49**	**534**	**362.49**	--	--	--
	AE	1579.96	535	509.96	1	149.46	0
	CE	1457.25	535	387.25	1	26.79	0
	E	1939.94	536	867.94	2	509.45	0
Nighttime Sleep Duration (30 mos)	ACE	1002.8	413	176.8	--	--	--
	AE	1075.78	414	247.78	1	72.98	0
	CE	**1003.29**	**414**	**175.29**	**1**	**0.49**	**0.48**
	E	1243.61	415	413.61	2	240.81	0
Nighttime Sleep Duration (5 years)	**ACE**	**836.24**	**372**	**92.24**	**--**	**--**	**--**
	AE	877.35	373	131.35	1	41.11	0
	CE	872.91	373	126.91	1	36.67	0
	E	1085.98	374	337.98	2	249.74	0
Sleep dysregulation (12 mos)	**ACE**	**525.96**	**535**	**-544.04**	**--**	**--**	**--**
	AE	531.66	536	-540.34	1	5.7	0.02
	CE	536.83	336	-535.17	1	10.87	0
	E	649.69	537	-424.31	2	123.73	0
Sleep dysregulation (30 mos)	ACE	117.53	447	-776.47	--	--	--
	AE	**118.15**	**448**	**-777.85**	**1**	**0.61**	**0.43**
	CE	126.55	448	-769.45	1	9.01	0
	E	182.4	449	-715.6	2	64.87	0
Daytime Sleepiness (5 years)	**ACE**	**1890.58**	**359**	**1172.58**	**--**	**--**	**--**
	AE	1898.53	360	1178.53	1	7.96	0
	CE	1908.09	360	1188.9	1	17.51	0
	E	2011.64	361	1289.64	2	121.06	0

Note. Bolded models denote the best fitting models for each predictor and outcome variable. 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. Δ χ^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 10

Bivariate Cholesky decompositions and model fit statistics for two bivariate models, including sibling conflict at five years with concurrent nighttime sleep duration and daytime sleepiness

Family and Sleep Variables	Model	-2LL	*df*	AIC	Δ *df*	Δ χ^2	*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 Daytime Sleepiness	ACE	2483.31	673	1137.31	--	--	--
	CE-ACE*	**2483.31**	**676**	**1133.31**	**2**	**0**	**.99**

Note. 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. Δ χ^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.

APPENDIX B

FIGURES

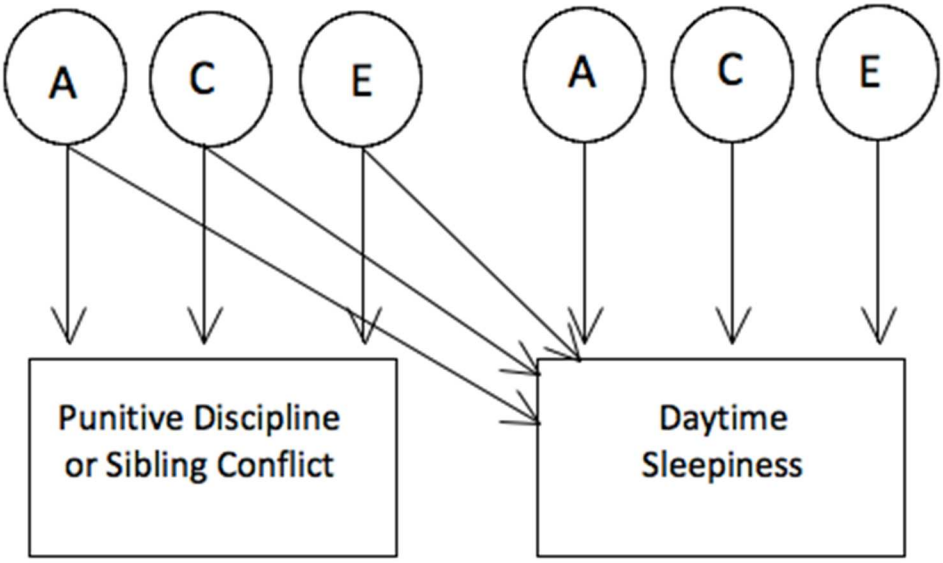

Figure 1. Example univariate and bivariate ACE models. Univariate and bivariate models will be conducted to examine the association between both parent-child relations and twin relations and childhood sleep parameters. Univariate models will include only one phenotype in the model, whereas bivariate models will include both phonotypes and their covariance in the model.

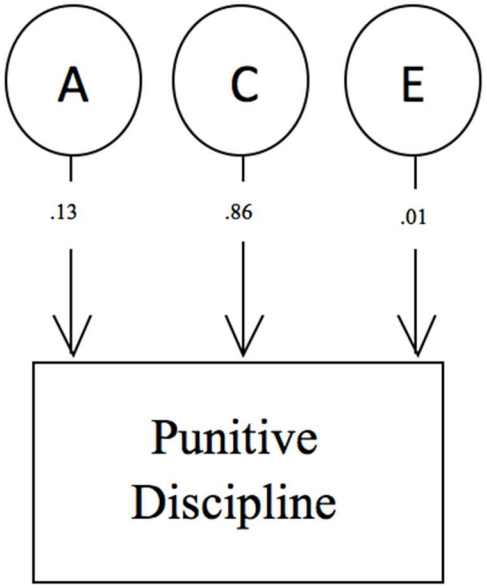

Figure 2. Univariate decomposition of parental punitive discipline at 30 months. Decomposition of variance is taken from the saturated ACE model (best model fit).

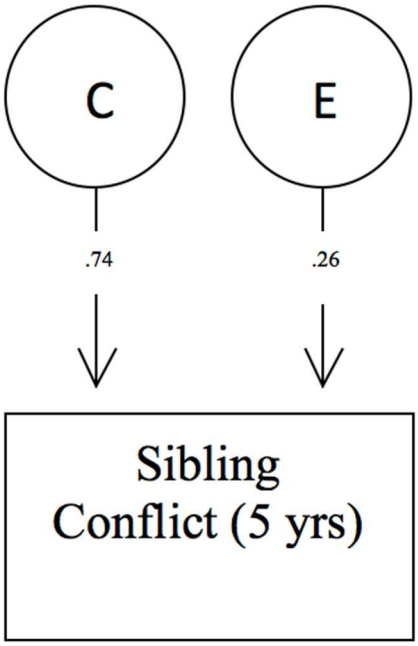

Figure 3. Univariate decomposition of sibling conflict at five years. Decomposition of variance is taken from the reduced CE model (best model fit).

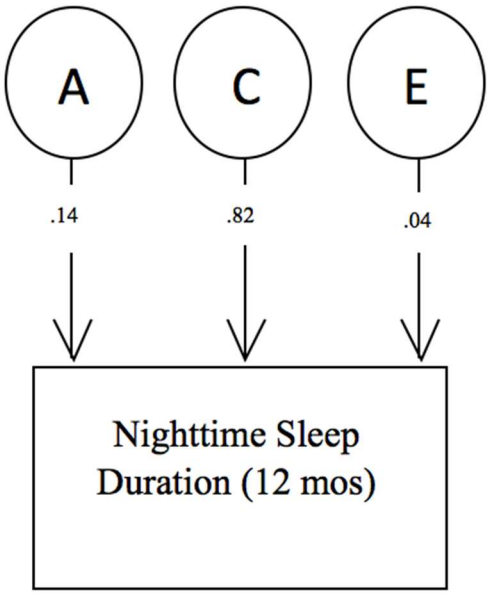

Figure 4. Univariate decomposition of nighttime sleep duration at 12 months. Decomposition of variance is taken from the full ACE model (best model fit).

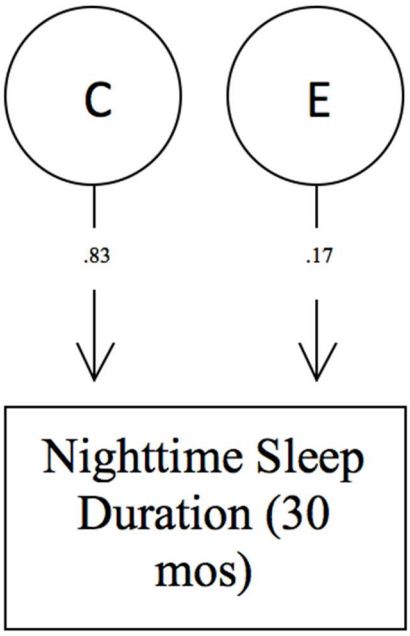

Figure 5. Univariate decomposition of nighttime sleep duration at 30 months. Decomposition of variance is taken from the reduced CE model (best model fit).

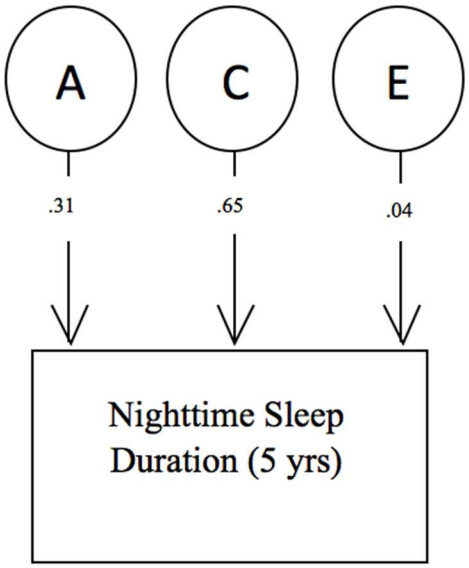

Figure 6. Univariate decomposition of nighttime sleep duration at five years. Decomposition of variance is taken from the full ACE model (best model fit).

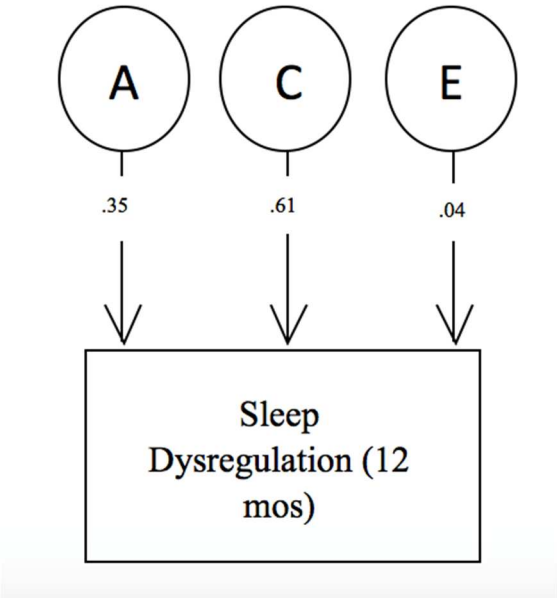

Figure 7. Univariate decomposition of sleep dysregulation at 12 months. Decomposition of variance is taken from the full ACE model (best model fit).

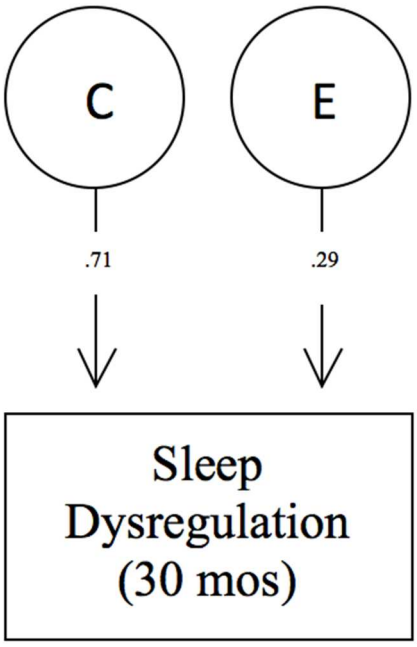

Figure 8. Univariate decomposition of sleep dysregulation at 30 months. Decomposition of variance is taken from the full ACE model (best model fit).

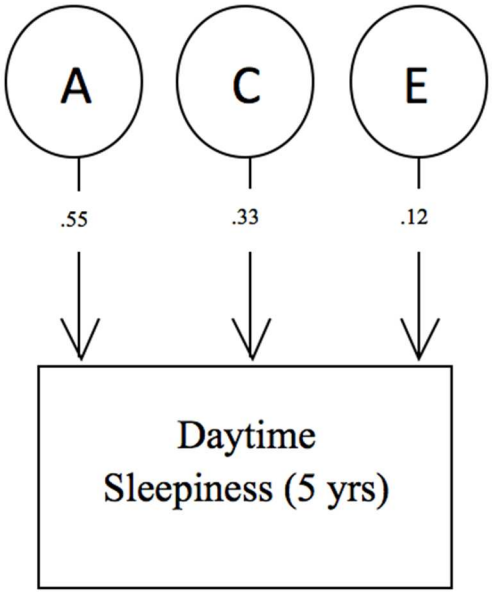

Figure 9. Univariate decomposition of daytime sleepiness at five years. Decomposition of variance is taken from the full ACE model (best model fit).

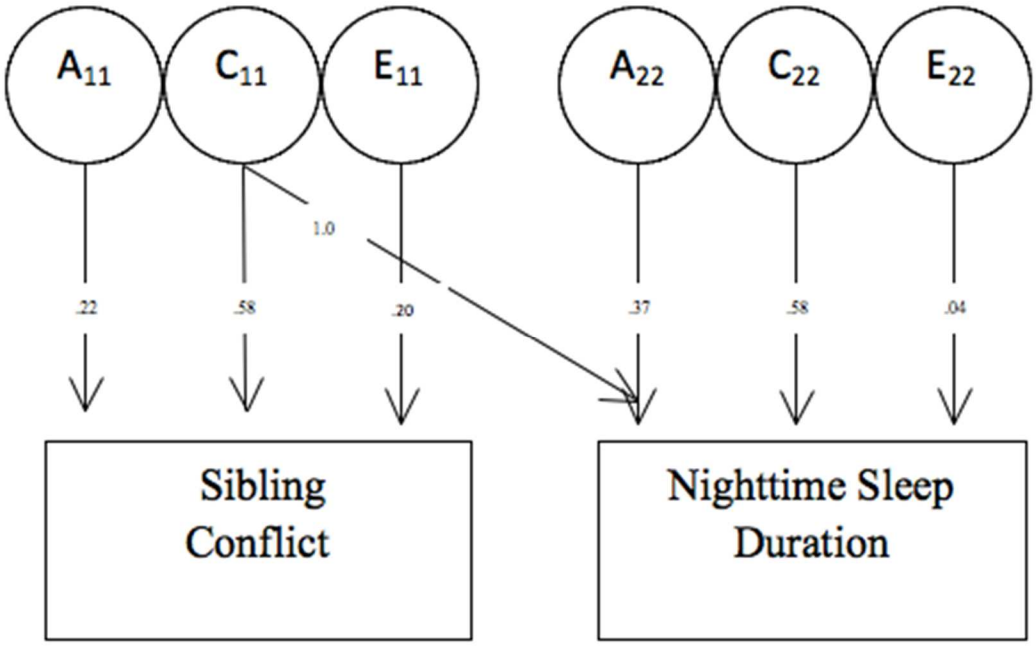

Figure 10. Bivariate Cholesky decomposition of the association between sibling conflict and nighttime sleep duration at five years. Decomposition of variance is taken from the full ACE-ACE model (best model fit), with the AE paths dropped from the shared component (covariance). All path estimates are standardized variance estimates.

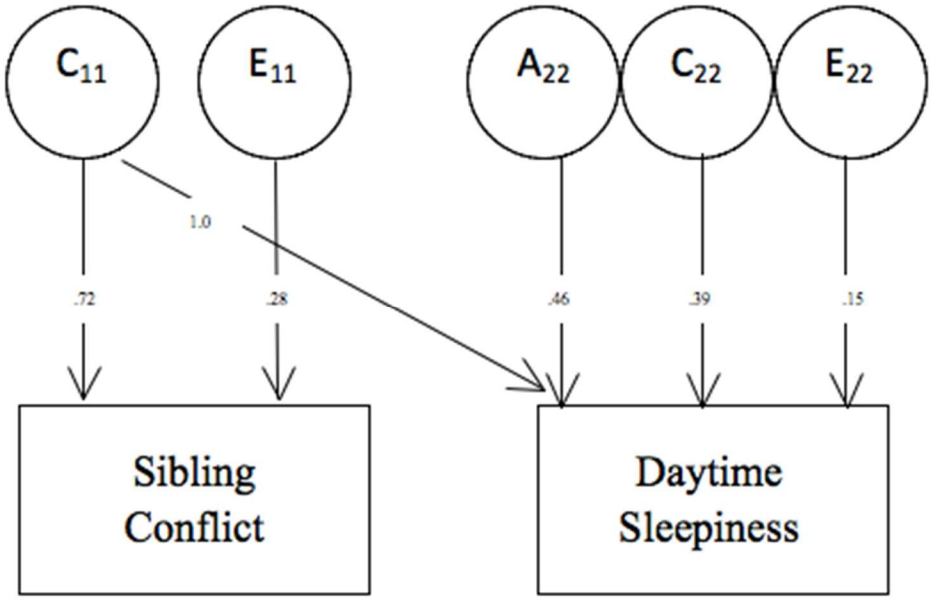

Figure 11. Bivariate Cholesky decomposition of the association between sibling conflict and daytime sleepiness at five years. Decomposition of variance is taken from the full CE-ACE model (best model fit), with the AE paths dropped from the shared component (covariance). All path estimates are standardized variance estimates.