Genetic and Environmental Influences on Associations Among Multiple Sleep Parameters, Weight Indicators and Weight Status, and Effortful Control in Young Twins

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ABSTRACT

Prior research has established associations between sleep duration and body mass index (BMI) scores and risk for obesity in middle childhood, but it is less clear whether other objectively- and subjectively-measured sleep indicators may be associated with BMI scores, weight status (e.g., obesity), and other estimates of weight and body fat such as waist circumference (WC) and percent body fat. Empirical studies have also demonstrated independent associations between broad self-regulation and sleep indicators and BMI scores, but no study to date has tested these factors in a model together and the extent to which associations between normative sleep problems, weight indicators, and effortful control (EC) may be explained by shared genetic or environmental influences. Data from a large longitudinal study of twins was used to test phenotypic associations between sleep problems at eight years and weight indicators at nine years, including whether EC at eight years moderates these associations. Additionally, multiple quantitative behavior genetic models were used to estimate unique and shared genetic and environmental covariances among normative sleep problems, weight indicators, and EC at eight years of age and whether additive genetic influence on weight in middle childhood differs by child weight status group. Phenotypic findings showed that greater sleep duration at eight years predicted greater decreases BMI at nine years of age for children with low levels of EC at eight years. Greater sleep midpoint variability at eight years predicted greater increases in percent body fat from eight to nine years of age for children with low EC at eight years. Behavior genetic findings showed greater environmental influences on parent-reported sleep duration and quality, as well as objective sleep midpoint variability. Similarly, associations between parent-reported sleep duration and sleep midpoint variability and other sleep indicators and EC were primarily accounted for by shared environmental factors. In contrast, there was high additive genetic influence on objective sleep quantity and quality, all weight indicators, and EC. Many of the associations between sleep indicators, sleep and weight indicators, and among weight indicators were entirely accounted for by shared additive genetic factors, suggesting that common, underlying sets of genes explain these relations.

DEDICATION

To Eric, Lily, and Otto.

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CHAPTER 1

INTRODUCTION AND BACKGROUND LITERATURE

About 30% of children sleep eight or fewer hours per night during middle childhood (National Sleep Foundation, 2014), suggesting that children may regularly experience normative sleep problems (i.e., non-clinical sleep problems like short duration, poor sleep quality; Sadeh, Raviv, & Gruber, 2000; Smaldone, Honig, & Byrne, 2007). Furthermore, normative sleep problems in childhood have been associated with numerous negative outcomes including internalizing and externalizing problems and decreased academic performance (Smaldone et al., 2007). Another aspect of health that may be linked with children's normative sleep problems is adiposity, a defining characteristic of obesity (excess body fatness; Cole & Rolland-Cachera, 2002; Tyler & Fullerton, 2008; Wells, 2014). Indeed, cross-sectional and longitudinal studies have consistently linked shorter nighttime sleep duration with higher body mass index (BMI) scores and increased odds of being classified as overweight or obese in childhood (see review by Magee & Hale, 2012).

Although studies have established links between short sleep duration and BMI or risk for obesity in childhood, the mechanisms underlying these associations are less clear. Theoretically, there are biological, psychological, social, and contextual processes that may explain relations between sleep problems and weight indicators; however, these mechanisms are rarely tested. Understanding whether important person-level psychosocial factors (e.g., effortful control [EC]) may explain links between sleep and weight indicators in children or whether relations between sleep and weight indicators may be attributed to genetic or environmental factors is critical, as discerning these processes may help inform interventions aimed at weight loss and reducing childhood obesity, as well as improving child sleep quantity, quality, and timing.

Given these gaps in the literature regarding associations between child sleep problems and weight indicators, the aims of the current dissertation were to: 1) test whether objective and subjective sleep at eight years of age were associated with objective weight indictors (BMI, waist circumference [WC], percent body fat) both concurrently and one year later (controlling for earlier weight), as well as whether EC at eight years moderated links between sleep and weight

indicators, and 2) estimate unique and shared genetic and environmental variances and covariances between sleep, weight indicators, and EC at eight years, and examine genetic and environmental contributions on weight status categories at eight years of age (underweight, healthy, overweight, obese).

Theoretical Frameworks for Childhood Sleep and Weight

Despite broad literature showing links between sleep and weight across the lifespan, there is not a single, overarching conceptual model to explain links between normative sleep problems and weight in children, including various biological, psychological, social, and environmental factors that may account for these relations. However, two main theories are particularly relevant when examining complex, multilevel relationships between sleep and weight in childhood: 1) developmental systems theory (e.g., biopsychosocial and contextual model for sleep; Becker, Langberg, & Byers, 2015; Damon & Lerner, 2008) and 2) the integrative model of health behaviors (e.g., individual differences of stress-induced eating; Fishbein, 2000; Greeno & Wing, 1994).

Developmental Systems Theory. Developmental systems theories propose bidirectional relationships between multiple variables, with these variables and relationships often crossing multiple levels of organization (Damon & Lerner, 2008). An example of this type of theory is Bronfenbrenner's Ecological Systems Model (Bronfenbrenner, 1992; Bronfenbrenner & Morris, 2006). All developmental systems theories share six to seven main principles, providing a firm foundation for theoretical and conceptual models hypothesizing associations among specific variables. Three principles are particularly relevant to the current dissertation. First, developmental systems theories adopt a holistic approach to understanding associations between variables across development, explicitly moving away from reductionism and determinism (Damon & Lerner, 2008). The current dissertation considered associations among multiple sleep and weight indicators, while examining the role of critical covariates (e.g., sex, puberty), EC, and genetics, rather that oversimplifying links among variables and assuming that a particular pathway has long-term, immutable influences on a trait (e.g., genetic determinism; Damon & Lerner, 2008).

Second, developmental systems theories hold that there are multiple levels of organization within human development, beginning with genetic, biological, and physiological variables (Damon & Lerner, 2008), and continuing with individual-level, family-level, community-level (e.g., school, work, peers, etc.), societal-level, and cultural- and chronological-level variables. The current dissertation tested individual-level differences between various sleep and weight indicators, allowing us to better understand associations between sleep and weight indicators at the person-level, genetic contributions to these links, and individual differences in links between sleep, weight indicators, and EC.

Third, developmental systems models acknowledge that specific traits may change within and across individuals over time (Damon & Lerner, 2008). The current dissertation tested whether child sleep problems at eight years of age predicted changes in weight indicators from eight to nine years during middle childhood. Theoretical frameworks suggest that changes in sleep behavior (e.g., restricted sleep) may serve as an initial stressor that lead to a cascade of other biological (hormonal) and psychosocial changes, which in turn leads to changes in weight (typically weight gain; Miller & Cappuccio, 2007; Spiegel, Tasali, Penev, & Van Cauter, 2004). However, there is evidence that there are likely bidirectional relationships (another key tenant in developmental systems theories) between sleep problems and weight indicators both concurrently and longitudinally (Fatima Doi, & Mamun, 2016; Magee & Hale, 2012).

Timing may also be important when examining links among traits. Middle childhood (approximately ages six to 12) may be an important period, as it seems to be a distinctive period that falls between other sensitive developmental periods and transitions (Collins, 1984). Notably, there are clear shifts in sleep architecture that occur at the beginning and end of middle childhood (Crowley, Tarokh, & Carskadon, 2014), with older children (approximately 8 years) sleeping fewer hours per night on average and demonstrating greater daytime sleepiness compared to younger children (approximately ages four and five; Crabtree & Williams, 2009). Similarly, research indicates that there are marked changes in weight and adiposity at the beginning of middle childhood, due to the development of eating and physical activity habits (Daniels, 2006). The onset of puberty (which usually begins in the middle or end of middle childhood) may be another

factor associated with normative sleep problems and weight gain or status. Finally, research suggests that increases and/or changes in genetic and environmental influences on sleep problems (e.g., insomnia) and obesity tend to remain stable or slightly increase over childhood and adolescence (Barclay, Gehrman, Gregory, Eaves, & Silberg, 2014; Plomin, DeFries, Knopik, & Neiderhiser, 2013). Thus, the current dissertation examined links between sleep and weight indicators while considering the role of variables from multiple levels of organization like genetics, age, sex, socioeconomic status (SES), ethnicity, and puberty, as well as how relations between sleep and weight indicators may change within and across individuals over time (i.e., eight to nine years) or contexts after accounting for other variables.

One example of a developmental systems theory is the biopsychosocial and contextual model of sleep (Becker et al., 2015), which considers how various biological, psychological, and social/environmental factors independently contribute to sleep, and how these factors interact with one another to influence sleep behaviors across adolescence. However, the biopsychosocial and contextual model of sleep focuses on adolescence and largely ignores other health behaviors that may contribute to sleep, such as weight or adiposity. Thus, there is significant opportunity for improvement (additions and changes) in current models outlining contributions to sleep such as the biopsychosocial and contextual model of sleep.

The Integrative Model of Health Behaviors. The Integrative Model of Health Behaviors (Fishbein, 2000) is a large theoretical model with greater clinical focus than developmental systems theories. The model explains contributions to health behaviors, while informing intervention and prevention efforts to reduce risky health behaviors (Fishbein, 2000). While the model was initially used to explain the spread of HIV (via health behaviors and decisions) and potential points of intervention for the disease, broad tenants of the integrative model can be applied to other health behaviors and subsequent disorders or diseases. First, health behaviors in the integrative health model are generally comprised of actions, a target, context, and a time frame (Fishbein, 2000). Relevant to the current dissertation and using increases in weight indicators or status as an outcome, the action may be eating, the target may be food, the context may be within the home or at school (or both), and the time period may be specific time frames

(e.g., middle childhood) or across development. Building upon this, the integrative model of health behaviors holds that any changes in action, target, context or time can produce change in a health behavior or disease (Fishbein, 2000). The current dissertation not only assessed relations between sleep and change in weight indicators, but also considered various timing and contextual factors that may affect these associations. Third, there are significant individual differences in internal and external variables that likely contribute to a health behavior (Fishbein, 2000). The current dissertation tested whether children in higher weight status groups (overweight or obese) may have different genetic influences on weight than other children, accounting for between-person variability in weight.

An example of a model related to the Integrative Model of Health Behaviors is the Individual Differences Model of Stress-induced Eating, which aims to clarify etiology and causes of obesity (Greeno & Wing, 1994). The individual differences model proposes that there are differences between obese and non-obese individuals regarding stress responses, such that obese individuals are more likely to eat in response to stress compared to non-obese individuals (Greeno & Wing, 1994). One proposed explanation for differences between obese and non-obese individuals is that individuals who have problems with "restraint" tend to over-eat (particularly when stressed), resulting in weight gain and obesity (Greeno & Wing, 1994). This "restraint" concept is relevant to the current dissertation as it may be related to an individual's temperament, specifically levels of EC and self-regulation (the extent to which individuals can willfully control behaviors, attention, and cognition; Eisenberg, Hofer, Sulik, & Spinrad, 2014). Indeed, studies demonstrate that there is variability in self-regulation within the population (Kochanska & Knaack, 2003), suggesting that self-regulation and EC may be ideal aspects of temperament to assess in relation to individual differences in eating behaviors and obesity. However, the Individual Differences Model of Stress-induced Eating is somewhat narrow and focuses primarily on how stress influences eating behaviors, rather than other person-level factors (e.g., lifestyle, demographic, etc.) that may contribute to sleep and/or obesity. Aim 1 of the current dissertation tested whether EC moderated associations between sleep and weight indicators, acting as a possible protective factor and reducing negative effects of poor sleep on weight outcomes. Aim 2

of the current dissertation examined whether EC may be a key psychosocial factor that shares overlapping genetic or environmental factors with sleep and weight indicators.

Building a New Theoretical Model Linking Sleep and Weight Indicators

There is no theoretical or conceptual model in psychology that addresses associations and pathways between sleep problems and weight indicators or status in childhood. However, we can combine the best characteristics of existing theoretical frameworks to create a comprehensive framework with which to test associations between normative sleep problems and weight indicators in childhood. Figure 1 outlines a proposed theoretical model that combines key aspects of developmental systems theories and the integrative model of health behavior and serves as rationale and description of how sleep problems and weight indicators may be associated in the current dissertation. The current dissertation does not test or address all pathways in the proposed model. As such, pathways of interest are numbered and discussed in more detail below.

Biological vulnerabilities. Biological vulnerabilities (Path 1) may contribute to relations between child sleep and weight indicators, including physiological, hormonal, or genetic factors. Strong theoretical evidence suggests that endocrine and hormone processes underlie links between sleep and weight, such that sleep problems may prompt changes in hormones levels (specifically leptin, ghrelin, and insulin) and problems with glucose uptake and metabolism (Miller & Cappuccio, 2007; Spiegel et al., 2004), which may lead to increased adiposity (Miller & Cappuccio, 2007; Spiegel et al., 2004). One empirical study also showed that greater sleep duration variability and shorter sleep duration were associated with greater changes in insulin levels and health risk during middle childhood (Spruyt, Molfese, & Gozal, 2011).

Relevant to the current dissertation, genetic factors are another prominent biological pathway that may account for relationships between sleep and weight indicators or status. The sleep-wake cycle is constitutionally-based and one of the earliest biological rhythms to develop and regulate (Peirano, Algarin, & Uauy, 2003), suggesting that sleep contains genetic influences. Indeed, cross-sectional studies indicate that subjective and objective reports of various sleep parameters such as sleep duration and daytime sleepiness are moderately heritable (30-70%;

e.g., Gregory, Rijsdijk, & Eley, 2006). Like sleep, adiposity and weight are biologically-based and contain genetic influences (Kopelman, 2000), with recent studies showing 60-70% of the variance in BMI and 40-60% of variance in WC is accounted for by additive genetics in middle childhood (see Fernandez, Klimentidis, Dulin-Keita, & Casazza, 2012; Wardle, Carnell, Haworth, & Plomin, 2008). The current dissertation used a behavior genetic framework to test links between sleep, weight indicators, and EC in middle childhood.

Environmental factors. Environmental factors (Path 2) likely directly impact links between child sleep and weight indicators. For example, both broad environmental contexts (e.g., family, physical home environment, school), as well as specific environmental factors (e.g., family eating behaviors, food availability, sleep environment; Spruijt-Metz, 2011) may influence associations between sleep and weight in childhood. The "Sleep in America" Poll also reported that child sleep difficulties in a given week could be attributed to things like evening activities (34%) or ambient or direct light (8%; National Sleep Foundation, 2014) among other factors. Empirical literature also indicates that household chaos and/or disruptions surrounding bedtime have been linked to greater likelihood of sleep disruptions at night in middle childhood (Fiese, Winter, Sliwinski, & Anbar, 2007). The current dissertation estimated broad environmental or contextual effects on links between sleep, weight indicators, and EC through behavior genetic models.

Psychosocial factors. Psychological factors (Path 3) also likely influence links between sleep and weight in childhood. Of interest to the current dissertation, self-regulatory EC is theorized to influence behavioral, cognitive, and emotional processes, including sleep-wake cycles and feeding behavior in the first few years of life (Calkins, Perry, & Dollar, 2016). Self-regulation may continue to influence sleep and weight in middle childhood, as studies have shown that poor self-regulation early in life contributes to sleep problems and weight gain and risk for obesity later in childhood (Graziano, Calkins, & Keane, 2010; Van den Bergh & Mulder, 2012). Thus, both phenotypic and behavior genetic associations between EC, sleep, and weight indicators in middle childhood were tested in the current dissertation.

Demographic and lifestyle factors. The bottom portion of the conceptual model provides multiple examples of demographic and health and lifestyle factors (Path 4) that may have direct, indirect, and/or interactive effects on relations between normative sleep problems and weight in childhood. For example, studies have shown that low socioeconomic status (SES; Biggs, Lushington, Martin, van den Heuvel, & Kennedy, 2013; Breitenstein, Doane, & Lemery-Chalfant, in press; O'Dea, Dibley, & Rankin, 2014; Wisniewski & Chernausek, 2009), poor diet (Franckle et al., 2015; Kjeldsen et al., 2014), low physical activity (Carson, Tremblay, Chaput, & Chastin, 2016), ethnicity (Biggs et al., 2013; Wisniewski & Chernausek, 2009), sex (Biggs et al., 2013; El-Sheikh, Bagley, Keiley, & Erath, 2014), and puberty (Laberge et al., 2001) may be potential moderators of associations between sleep and weight indicators. The current dissertation included many of these factors in analytic models to account for individual differences particularly in sex, race/ethnicity, time of assessment, SES, pubertal development, and prior weight indicator scores (e.g., earlier BMI scores).

Development and change over time. Finally, Path 5 is a broad representation of time, such that it delineates how biological, psychological, social, and contextual factors are contributing to links between normative sleep problems and weight indicators continuously across childhood (and the lifespan). The current dissertation addressed Path 5 by testing prospective associations between objective and subjective sleep at eight years and weight indicators and status at nine years of age (Aim 1).

Defining Sleep Problems and Weight Indicators

Definitions of (normative) sleep problems, objective and subjective sleep indicators, weight and obesity differ and change considerably across studies and theoretical frameworks. Within the current dissertation, normative sleep problems in middle childhood were defined broadly as children experiencing non-clinical levels of sleep disruptions including restricted sleep, poor sleep quality, trouble with sleep onset, and increased daytime sleepiness (National Sleep Foundation, 2014; Sadeh et al., 2000; Smaldone et al., 2007). Objective sleep indicators were defined as facets of sleep (quantity, quality, variability) measured using wrist-based

accelerometers or actigraph watches for the current dissertation. References to subjective sleep indicators and problems described parent-reports of child sleep duration and quality.

Regarding weight, the current dissertation considered multiple weight indicators and weight status. Specifically, body mass index scores (BMI) accounted for height and weight when estimating overall body mass, whereas waist circumference (WC) estimated visceral or abdominal adipose fat tissue (Vivier & Thompkins, 2008). Percent body fat provided a measure of the proportion of body fat to an individual's total body weight (Tanita, 2016). Importantly, BMI, WC, and percent body fat did not directly measure adipose tissue, but rather provided a proxy for adipose body tissue or fat (Vivier & Thompkins, 2008).

Researchers and clinicians also utilize these various proxy measures of adiposity to classify individuals into specific groups (often for comparison purposes) as a way of better understanding how adiposity may be linked to child developmental outcomes. Researchers and clinicians typically classify children (and adults) into four different weight categories: underweight, normal or healthy weight, overweight, and obese (Centers for Disease Control, 2015). Children are often placed in one of these four categories based on measurements or scores for BMI, WC, and percent body fat, accounting for sex and age (Centers for Disease Control, 2015). The current dissertation used these weight status categories within Aims 1 and 2, with children placed in weight status categories based on age- and sex-specific percentiles for BMI, WC, and percent body fat.

Developmental Importance of Examining Sleep and Weight in Middle Childhood

As noted, middle childhood serves as distinctive developmental period (Collins, 1984), characterized by shifts in sleep architecture and timing, increases (and possibly decreases) in adiposity, pubertal development, and potential changes in genetic and environmental influences on traits like sleep and weight (Barclay et al., 2014; Crabtree & Williams, 2009; Crowley et al., 2014; Daniels, 2006; Plomin et al., 2013). Thus, examining various normative sleep problems and indicators during middle childhood is critical, as childhood sleep problems have also been associated with numerous child developmental outcomes (Smaldone et al., 2007) including poor mental and physical health (Chaput et al., 2016; Smaldone et al., 2007), more anxiety and

depressive symptoms (Gregory, Eley, O'Connor, Rijsdijk, & Plomin, 2005; Smaldone et al., 2007), increased problem behaviors and ADHD (Sadeh, Gruber, & Raviv, 2002; Smaldone et al., 2007), and poorer grades and less positive school experiences (see Astill, Van der Heijden, Van IJzendoorn, & Van Someren, 2012; Curcio, Ferrara, & De Gennaro, 2006; Smaldone et al., 2007).

Weight indicators and status may be one facet aspect of poor physical health that is related to sleep (see Astill et al., 2012; Chaput et al., 2016; Smaldone et al., 2007), making it essential to further examine associations between child sleep problems and weight indicators and weight status. In particular, there is a growing obesity epidemic within the child population (both nationally and worldwide), with approximately 33% of children and adolescents (ages 2-19) in the United States classified as either overweight, and 17% of these children also meeting cutoff scores for obesity in 2010 (Ogden, Carroll, Kit, & Flegal, 2012; Ogden, Carroll, Kit, & Flegal, 2014; Pulgaron, 2013). Additionally, considerable research has shown that numerous negative health outcomes in childhood and adulthood are associated with high adiposity and risk for obesity (via BMI scores), including greater risk for morbidity and mortality (Vivier & Thompkins, 2008), physical health problems such as Type 2 diabetes mellitus, asthma or breathing problems (Ayer, Charakida, Deanfield, & Celermajer, 2015; Howe et al., 2014; Park, Falconer, Viner, & Kinra, 2012; Pulgaron, 2013), lower self-esteem, greater anxiety and depressive symptoms (Pulgaron, 2013; see Vivier & Thompkins, 2008), and cognitive deficits (Liang, Matheson, Kaye, & Boutelle, 2013). Overall, middle childhood serves as a unique period of development during which alterations in associations between sleep and weight indicators may occur and should be more closely examined.

Prior Literature Linking Sleep Problems and Weight Indicators

Cross-sectional studies. Recent reviews and meta-analyses have illustrated that short subjective sleep duration was associated with higher BMI scores, increased risk for obesity, and increased weight gain both cross-sectionally and longitudinally (Cappuccio et al., 2008; Magee & Hale, 2012). Specifically, cross-sectional empirical studies have shown that shorter parent-reported and actigraphy-based sleep duration (including later bedtimes and waketimes; Ekstedt,

Nyberg, Ingre, Ekblom, & Marcus, 2013) are associated with risk for obesity and/or higher BMI scores (Chaput, Brunet, & Tremlay, 2006; Ekstedt et al., 2013; Martinez et al., 2014; Nixon et al., 2008; von Kries, Toschke, Wurmser, Sauerwald, & Koletzko, 2002), greater WC (Chaput et al., 2006), and higher percent body fat (Nixon et al., 2008; von Kries et al., 2002). Notably, associations between short sleep duration and risk for being overweight or obese remain after controlling for multiple demographic and lifestyle factors, such as parent obesity, parent education, sex, age, screen time, physical activity level, eating behaviors, and birth height and weight (Chaput et al., 2006; Martinez et al., 2014; Nixon et al., 2008; von Kries et al., 2002).

Regarding other objective sleep indicators, one meta-analysis also found that poor sleep quality was concurrently associated with greater risk for being overweight or obese, independent of sleep duration (Fatima et al., 2016). Empirical studies also show that greater objective sleep duration variability is associated with obesity (Spryut et al., 2011), and lower objective sleep efficiency predicted higher zBMI scores after controlling for various risk factors such as parent education, SES, family structure, and stressful life events (Bagley & El-Sheikh, 2013).

Longitudinal studies. Far fewer longitudinal studies have tested links between child sleep and weight indicators. However, longitudinal research suggests children who obtain more sleep at night on average (via parent-report), have earlier bedtimes, and later wake times show lower BMI scores and lower risk of being obese five years later after accounting for initial BMI and demographic factors (Snell, Adam, & Duncan, 2007). Similarly, longer objective sleep duration in early childhood was associated with lower zBMI scores, lower percent body fat, and approximately a 60% reduced risk for being classified as overweight or obese at age seven after adjusting for BMI and body fat at age three and demographic factors (Carter, Taylor, Williams, & Taylor, 2011). On the other hand, children who were consistently short sleepers (via parent-report) in early childhood showed higher BMI scores and greater risk of being overweight or obese children at age six compared to children who were persistently long sleepers after accounting for numerous confounding factors (Touchette et al., 2008).

Overall, cross-sectional and longitudinal studies show that shorter objective and subjective sleep duration, quality, and later sleep timing are associated with greater BMI scores

increased risk for being overweight or obese. However, few studies assess multiple objective and subjective measures of sleep or multiple weight indicators or test associations between sleep and weight over time while adjusting for a number of demographic and lifestyle factors.

The Role of Effortful Control in Associations Among Sleep and Weight Indicators

As outlined in the conceptual model (Figure 1, Path 3), EC may be an important psychosocial factor that accounts for or modulates associations between normative sleep problems and weight indicators in middle childhood. Within the current dissertation, EC was defined as a broad, regulatory dimension of temperament that typically includes executive functioning (e.g., planning, decision making, and detecting errors), as well as inhibitory control, attentional focusing, persistence, and low intensity pleasure (Eisenberg et al., 2014; Rothbart & Bates, 2008). EC is also the extent to which individuals can *willfully* activate a subdominant response to stimuli and inhibit a dominant response to stimuli (Eisenberg et al., 2014). Characteristics of EC and broad self-regulation like attentional focusing and willful behavioral inhibition begin to appear around 12 months of age and steadily increase across childhood, with sharp increases at the beginning of middle childhood (ages five to six; Eisenberg et al., 2014; Kochanska & Knaack, 2003). A recent review identified EC as a crucial factor influencing child (and adult) development, such that children with higher EC show better emotional regulation and understanding, greater socioemotional awareness and empathy, higher school liking and academic achievement, and better adjustment overall (Eisenberg et al., 2014).

Studies have also shown that EC is independently associated with normative sleep problems and weight indicators in middle childhood. Lower levels of broad self-regulation have been independently associated with poor sleep, shorter sleep duration, and higher BMI scores both concurrently and longitudinally in middle childhood (Graziano et al., 2010; Graziano, Kelleher, Calkins, Keane, & Brien, 2013; Hughes, Power, O'Connor, & Fisher, 2015; Williams & Sciberras, 2016). For example, one recent longitudinal study found that children with average to high self-regulation exhibited diminished parent-reported sleep problems over time (69% of children; Williams, Nicholson, Walker, & Berthelsen, 2016), whereas children with lower self-regulatory skills showed increased sleep problems across early and middle childhood (31%).

Regarding various components of self-regulation and weight indicators, Hughes and colleagues (2015) found that lower eating self-regulation was associated with higher zBMI scores in preschoolers, but broad self-regulation (accounting multiple aspects of self-regulation) was not related to zBMI scores. Another study found that children with lower emotional self-regulation and inhibitory control (component of self-regulation) at two years of age were more likely to be classified as obese at about five years of age (Graziano et al., 2010).

Overall, EC and broad self-regulation are linked independently with sleep, BMI scores and risk for obesity concurrently and longitudinally, but it is less clear whether specific aspects of self-regulation like EC are associated with sleep and weight, as no studies to my knowledge have tested various facets of sleep, weight indicators, and EC in a model together. Furthermore, while direct relations between EC, sleep, and weight indicators have been established, links between sleep and weight may vary according to child EC levels. For example, it is possible that poor sleep predicts increased BMI, WC, and percent body fat, but only for children with low levels of EC and poor self-regulation more broadly. As such, dysregulated behaviors, emotions, or cognitions would help explain links between poor sleep and increased weight and adiposity for particular children with low EC. Thus, Aim 1 of the current dissertation tested whether child EC at eight years moderates associations between sleep at eight years and weight indicators at nine years.

The Role of Genetics in Associations Among Sleep and Weight Indicators

Based on prior literature showing independent links between sleep, weight indicators, and EC, it may also be critical to examine associations between these traits within a genetically-informed design. The twin method is one way of exploring the contributions of genetic and environmental factors on developmental outcomes in the population by comparing monozygotic (MZ; identical) and dizygotic twins (DZ; fraternal; Plomin et al., 2013). Comparing MZ and DZ twins shows the 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 segregating DNA, while any behavioral differences between DZ twins may be

attributed to both genetic and environmental factors, as DZ twins share roughly 50% of their genetic composition (Plomin et al., 2013).

Using these assumptions regarding genetic composition, the ACE model can be used to estimate differences between MZ and DZ twins on variances in additive genetic, shared environmental, and non-shared environmental factors that contributes to a phenotypic trait (see Figure 2; Neale & Cardon, 1992; Plomin et al., 2013). The additive genetic (A) portion of the model accounts for the likelihood that multiple genes influence a phenotype (Neale & Cardon, 1992; Plomin et al., 2013). The proportion of additive genetic contribution to a behavior will differ between MZ and DZ twin pairs (MZ set to 1.0, DZ set to 0.5), 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 for a set of twins and may influence a phenotype to make twins more similar to one another. Shared environmental factors are assumed to equal to 100% (or 1.0) for both MZ and DZ twin pairs raised together (Neale & Cardon, 1992; Plomin et al., 2013). 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, and thus make twins more different from one another (Neale & Cardon, 1992; Plomin et al., 2013). The E component is important for measuring contextual factors that twins may experience uniquely, such as schooling or peer groups, as well as measurement error.

Prior research indicates genetic and environmental influences on various sleep parameters, weight indicators, and EC in twin samples of children in middle childhood, with sleep parameters and problems as much as 70% heritable, and daytime sleepiness (indicator of sleep quality) being 30 to 55% heritable (Breitenstein, Doane, Clifford, & Lemery-Chalfant, 2018; Gregory et al., 2006; Moore et al., 2009). Twins studies estimating genetic and environmental influences on weight indicators are more consistent, showing that BMI is between 60 and 70% heritable (Maes, Neale, & Eaves, 1997; Fernandez et al., 2012; Wardle et al., 2008), and about 40% of the variance in WC is accounted for by unique genetic and environmental factors, while the remaining 60% can be explained by overlapping additive genetic contributions with BMI scores (Wardle et al., 2008). Finally, EC and other aspects of temperament show between 20 to

60% heritability (Saudino & Micalizzi, 2015), although empirical studies suggest EC is between 50 and 70% heritable in middle childhood, with some consistency across multiple reporters as well (Lemery-Chalfant, Doelger, & Goldsmith, 2008; Lemery-Chalfant, Kao, Swann, & Goldsmith, 2013; Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009). Overall, Aim 2 of the current dissertation used univariate ACE models to estimate contributions to sleep, weight indicators, and EC for twins in a large, multiethnic and socioeconomically diverse sample of children in middle childhood.

Quantitative Behavioral Genetic Models

Beyond simple ACE modeling, other quantitative behavioral genetic models can estimate unique and shared genetic and environmental contributions to sleep, weight indicators, and EC (Neale & Maes, 2004; Plomin et al., 2013). Three quantitative behavioral genetic models relevant to the current dissertation are the Multivariate Cholesky Decomposition, the Independent Pathway Model, and the Liability Threshold Model, with each model answering slightly different questions regarding genetic and environmental associations between traits.

Using a Multivariate Cholesky Decomposition model can help researchers determine the extent to which genetic or environmental factors influence a given trait after accounting for genetic and environmental influences shared with other traits (see Figure 3; Clifford, Lemery-Chalfant, & Goldsmith, 2015; Neale & Maes, 2004). As depicted in Figure 3, the multivariate Cholesky decomposition can estimate unique additive genetic (A3), shared environmental (C3) and nonshared environmental (E3) influences on weight indicators such as BMI scores. Further, the multivariate Cholesky decomposition accounts for additive genetic (A1 and A2), common environmental (C1 and C2), and nonshared environmental (E1 and E2) influences on weight indicators (e.g., BMI) that are shared with sleep (e.g., sleep duration) and EC, respectively.

The Independent Pathway Model is similar to the Multivariate Cholesky Decomposition, but tests whether there is a common underlying set of genes (As), a single shared environmental factor (Cs) or a common nonshared environmental factor (Es) that accounts for associations among traits. (see Figure 4; Clifford et al., 2015; Neale & Maes, 2004). In addition, the independent pathway model estimates unique additive genetic (A1, A2, and A3), shared

environmental (C1, C2, and C3), and nonshared environmental factors (E1, E2, and E3) that contribute to a particular trait, independent of other traits or behaviors. For example, we can test whether there is an underlying additive genetic factor that explains links between sleep, weight indicators, and EC (As), or whether a single shared environmental or psychosocial factor (Cs) such as general dysregulation can explain associations between sleep, weight indicators, and EC.

Finally, Liability Threshold Models allow researchers to use categorical data to test the extent to which genetic and environmental influences on a trait may differ across groups of individuals (Figure 5; Plomin et al., 2013). For example, the Liability Threshold Model can use the assumptions of a typical ACE model and estimate differences in additive genetic, shared environmental, and nonshared environmental factors between groups on physical health traits like weight (e.g., weight status group). The Liability Threshold Model is particularly useful in that it can inform clinical research and interventions, given that the model utilizes categorical data which is often used with psychological and physical health diagnoses.

Regarding genetic and environmental associations between sleep and weight indicators, there are no studies to our knowledge testing these associations with child samples. However, at least one set of studies using adult twins found that shorter self-reported sleep duration was associated with higher BMI scores, and these associations were accounted for entirely by common environmental effects (Watson, Buchwald, Vitiello, Noonan, & Goldberg, 2010). A related study found that shorter self-reported sleep duration predicted greater BMI scores, as well as higher heritability of BMI scores particularly for participants who reported longer sleep duration (Watson et al., 2012). These findings suggest that restricted sleep may provide an opportunity or context that allows for greater genetic expression of BMI or weight more broadly, whereas longer sleep duration may restrict genetic expression of BMI or weight.

Intersections between Psychosocial Factors, Sleep, and Weight Indicators

A few empirical studies have been conducted that examine whether biological, psychological or contextual factors influence associations between sleep problems and weight indicators. One study found that sleep problems at eight years predicted depressive symptoms at

10 years in a longitudinal sample of twins, and stability in sleep problems over time were primarily explained by genetic influences (46%), although stability in depressive symptoms was accounted for by shared environmental factors primarily (Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009). Associations between sleep problems and depressive symptoms across middle childhood were primarily accounted for by genetic factors (Gregory et al., 2009). Another study by Faith et al. (2012) found phenotypic associations between self-regulation and eating behaviors in a small sample of twins in middle childhood, such that poor self-regulation was linked to increased eating and greater percent body fat, with these relationships being particularly strong for Hispanic and African American children and girls in the study. Furthermore, the authors found that the greatest proportion of the variance in poor self-regulatory eating was accounted for by shared environmental factors, with the remaining variance primarily attributed to nonshared environmental influences (Faith et al., 2012). Overall, there are few studies examining biopsychosocial and contextual intersections of sleep problems and weight in middle childhood; however, future studies should aim to test phenotypic associations among multiple domains of functioning (like EC), sleep problems, and weight indicators to clarify these relationships, as well as test these associations within a behavior genetics framework to determine whether shared genes or the environment account for links among sleep, weight indicators, and EC.

The Present Study

Aim 1. The first aim of the current dissertation had two parts: Aim 1a tested whether objective and subjective sleep at eight years of age was associated with concurrent objective weight indictors (BMI, WC, percent body fat, risk for being classified as overweight/obese). Aim 1b examined whether objective and subjective sleep at eight years predicted objective weight indicators one year later in middle childhood, controlling for prior weight indicators and significant demographic factors at eight years. Furthermore, Aim 1a and 1b both tested whether EC at eight years moderated associations between sleep at eight years and weight indicators concurrently and longitudinally. While substantial research has established associations between subjective and objective sleep duration and BMI scores or risk for being overweight or obese, there are still a number of gaps in the literature regarding these links. First, most studies have only examined

cross-sectional (rather than longitudinal) associations between sleep duration and BMI or risk for obesity. Further, there are mixed results regarding cross-sectional and longitudinal findings, with some studies showing associations between sleep and weight both concurrently and over time (e.g., see review by Magee & Hale, 2012), while others have found no or mixed associations between sleep and weight, particularly as age increases (Patel & Hu, 2008). Thus, it is less clear whether there are associations between sleep and weight indicators, after controlling for prior weight and other important demographic factors, particularly when using sleep to predict subsequent weight (Snell et al., 2007). Additionally, associations between sleep and weight indicators may be stronger during particular developmental periods such as middle childhood.

Second, many studies examine subjective sleep (rather than objective sleep or multi-method sleep assessment), which may be problematic. Studies suggest anywhere from 20 minutes to almost an hour difference between actigraphy reports of sleep duration and parent-reported sleep duration, with parents consistently reporting longer sleep duration (Martinez et al., 2014; Nixon et al., 2008). Research also indicates that various aspects of sleep, as well as objective versus subjective sleep estimates, may have differential relations with developmental outcomes (see Patel & Hu, 2008; Tremaine, Dorrian, & Blunden, 2010), making it pertinent to examine multiple facets of subjective and objective measures of sleep.

Furthermore, most studies have only collected data for sleep duration, BMI scores, and risk for being overweight or obese (via BMI scores). As such, there is little information about how other sleep parameters (e.g., sleep efficiency, sleep midpoint time variability) and additional weight indicators (WC and percent body fat) may be related to one another. Scant research suggests that lower sleep efficiency and later bedtimes and wake times is associated with higher BMI scores and increased risk for being classified as overweight or obese (Bagley & El-Sheikh, 2013; Ekstedt et al., 2013; see meta-analysis by Fatima et al., 2016). However, no study to my knowledge has examined sleep midpoint variability specifically (which accounts for fluctuations in bedtime and waketime) in relation to weight indicators or risk for obesity.

Finally, few studies to my knowledge have tested whether important family- or person-level factors may strengthen or weaken links between sleep and weight indicators during middle

childhood. Child self-regulation has been associated with sleep duration and quality (Hughes et al., 2015; Williams & Sciberras, 2016), as well as BMI and risk for obesity in childhood (e.g., Graziano et al., 2010; Granziano et al., 2013), suggesting that specific facets of regulation like EC, may serve as important moderators of links between sleep and weight indicators.

Given these limitations in the literature, the current dissertation used cross-sectional data as well as a short-term longitudinal design to examine whether a) different aspects of sleep and EC were associated with weight indicators concurrently, and b) multiple sleep parameters and child EC predicted changes in weight indicators from eight to nine years of age. The first aim used actigraphy-based estimates of sleep duration, sleep efficiency (proxy for sleep quality), and sleep midpoint variability, as well as parent-reported sleep duration and daytime sleepiness (proxy for sleep quality) to test differential relations between sleep and weight outcomes both cross-sectionally and longitudinally. Additionally, the current dissertation used objectively measured BMI scores, WC, percent body fat, and weight status (based on all weight indicators). Based on prior literature, I predicted that shorter objective and subjective duration, lower objective sleep efficiency and subjective daytime sleepiness, and greater objective sleep midpoint variability at eight years would be linked with higher BMI scores, greater WC, higher percent body fat, and greater risk for being classified as overweight or obese at eight years (Aim 1a). I also expected that shorter objective and subjective duration, lower objective sleep efficiency and subjective daytime sleepiness, and greater objective sleep midpoint variability at eight years would be linked with higher BMI scores, greater WC, higher percent body fat, and greater risk for being classified as overweight or obese at nine years (Aim 1b). For both Aim 1a and b, I also hypothesized that there would be stronger negative associations between objective and subjective sleep and weight indicators and status for children with lower levels of EC, whereas links between sleep and weight indicators would be attenuated for children with average and high levels of EC.

Aim 2. Prior literature has demonstrated independent associations between EC and various aspects of sleep and weight indicators, and other studies show moderate to high heritability of various sleep parameters, weight indicators, and EC in middle childhood. As such,

Aim 2a of the current dissertation estimated unique additive genetic, shared environmental and nonshared environmental influences on sleep, weight indicators and EC (Univariate Cholesky Decompositions), as well as tested whether the covariance between various sleep, weight, and EC indicators is primarily explained by additive genetic, shared environmental and nonshared environmental factors (Bivariate or Multivariate Cholesky Decompositions). Based on prior empirical studies with twin children (Breitenstein et al., 2018; Fernandez et al., 2012; Gregory et al., 2006; Moore et al., 2009; Saudino & Micalizzi, 2015; Wardle et al., 2008), I expected the greatest proportion of the variance in objective and subjective sleep duration, objective sleep efficiency, and child EC to be accounted for by additive genetic factors. I also predicted that the greatest proportion of the variance in parent-reported daytime sleepiness to be accounted for primarily by shared environmental factors, with some contribution from additive genetic factors. Finally, I expected that the greatest proportion of the variance in BMI, WC, and percent body fat to be accounted for by additive genetic factors.

Furthermore, Aim 2b of the current dissertation tested whether there were shared and unique additive genetic, shared environmental, and nonshared environmental influences on child objective and subjective sleep, weight indicators, and EC at eight years of age using two multivariate quantitative behavioral genetic models (Multivariate Cholesky Decomposition and Independent Pathway Model; see Figures 3 and 4). I expected mostly shared additive genetic influences to account for links between sleep, weight indicators, and EC in children, given the moderate to strong additive genetic influence detected on various sleep problems, weight indicators, and EC in prior empirical studies of twins (Fernandez et al., 2012; Gregory et al., 2006; Wardle et al., 2008; Lemery-Chalfant et al., 2013). However, I also expected to find shared environmental associations between sleep, weight, and EC, given prior research in adults showing links between sleep duration and BMI scores are accounted for primarily by environmental effects (Watson et al., 2010; Watson et al., 2012).

Aim 2c of the current dissertation used a third behavior genetic model, the Liability Threshold Model, to determine whether genetic and environmental influences on weight differed according to weight status groups at eight years of age (see Figure 5). Specifically, I tested

whether different weight status groups showed greater additive genetic influences (or shared or nonshared environmental contributions) by comparing children who are underweight, overweight, and obese to children who fell within the normal range of the population at eight years. At least one study showed that adult twins who reported shorter self-reported sleep duration were more likely to have higher BMI scores, as well as higher heritability of BMI scores particularly for adults who reported longer sleep duration (Watson et al., 2012). These findings and theory concerning gene by environment interactions suggest that genetic expression of traits and characteristics may be highly dependent on the environment in which the trait is expressed, with some environments (e.g., supportive, nurturing, environments with greater resources) allowing for greater expression of genetic influence on a particular trait and other environments restricting genetic expression of the same trait (e.g., poorer, less supportive environments with fewer resources; Rutter, 2003; Price & Jaffee, 2008). Given this theory and prior findings, I predicted children who fell in the overweight and obese weight status groups would show higher heritability of obesity compared to children in the normal and underweight groups.

CHAPTER 2

METHOD

Participants

Participants were children from the Arizona Twin Project (ATP; Lemery-Chalfant, Clifford, McDonald, O'Brien, & Valiente, 2013), a large ongoing, longitudinal twin-panel study. Primary caregivers completed a questionnaire at the baseline assessment, which occurred when the twins were approximately 12 months old. The current dissertation included families who agreed to participate in two follow-up assessments: one when twins were approximately eight years old (data collected 2016-2018) and a second assessment when twins were nine years old (data collected 2017-2019). The full sample (12 months of age) consisted of 582 twins (291 families), including both monozygotic (MZ) and dizygotic (DZ) twin pairs (MZ = 26%, same-sex DZ = 35%, opposite-sex DZ = 33%; unknown zygosity = 6%). Twin group sizes were similar to that of what is found in the broader population. The sample was evenly split between males and females (male = 50.5%), and was ethnically diverse with 56.5% European American, 25.1% Latino, 5.2% Asian

American, 4.2% African American, 1.0% Native American, 1.6% Native Hawaiian families, and 6.3% multiethnic or unknown ethnicity. Families in the sample also demonstrated a broad range of socioeconomic status (SES; range = under $20,000 to over $150,000) at all the baseline assessment (Median = $80,000-$100,000). Regarding income-to-needs, 10.4% of the sample was living in poverty (score of <1), 20.9% were near the poverty line (score of 1-2), 23.9% were lower middle class (score of 2-3), and 44.8% were middle to upper class (score of 3+).

For the eight-year assessment, families from the full sample and new families with children born in the same years/cohort as the full sample were recruited into the study. Thus, the eight-year assessment included 608 twins (304 families; *M* = 8.52 years, *SD* = .63; data collected from 2016-2017). Of these families, 89 (29.6%) were MZ twin pairs, 117 (38.9%) were same-sex DZ twins, 95 (31.6%) were opposite-sex DZ families, and 3 (1.0%) were of unknown zygosity. Similar to the full sample, the eight-year sample was 49.2% male and ethnically diverse (approximately 56.6% European American, 24.8% Latino, 3.6% Asian American, 4.0% African American, 2.6% Native American, 1.0% Native Hawaiian families, and 8.0% multiethnic or unknown ethnicity). The majority of primary caregivers reported either completing college degree (36.8%; 33.3% for spouse/partner), two or more years of graduate school (3.3%; 3.5% for spouse/partner), or a completed graduate or professional degree (22.5%; 20.2% for spouse/partner), with the remaining reporting some college (27.5%; 26.7% for spouse/partner), a high school degree or equivalent (9.3%; 14.7% for spouse/partner), or less than a high school degree (.7%; 1.6% for spouse/partner) and unknown education level for .7% of primary caregivers (15.1% for spouse/partner). Like at the baseline assessment, families in the eight-year assessment showed a broad range of socioeconomic status (SES; range = under $20,000 to over $150,000), and had similar proportions of the sample in each income-needs-ratio category (living in poverty = 7.6% near the poverty line 19.4%; lower middle class = 13.2%; middle to upper class = 42.8%; 17.1% missing).

Procedure

Parents of twins were recruited through birth records in Arizona when the twins were approximately 12 months of age, and primary caregivers (94.6% mothers) completed interviews

via telephone regarding mother's pregnancy and twins' development, zygosity, temperament, and health. Primary caregivers were contacted again when twins were approximately eight years of age and offered the opportunity to participate in an intensive assessment of child sleep, physical and mental health, daily practices, cognitive functioning, and academic achievement, consisting of two home visits separated by a week-long study protocol. At eight years, over 70% of children completed the study week during the school year, with the remaining families completing procedures when children were out of school (e.g., vacation, summer break, etc.)

At each home visit for the eight-year assessment, experimenters collected questionnaires, biological measures (height, weight indicators), conducted cognitive tasks with the twins, and administered a parent-child interaction and an interview assessing the home environment. At each family's first home visit, study staff also trained the primary caregiver (94.1% mothers) for the week-long study protocol, in which the twins wore wrist-based accelerometers (actigraph watches) for seven nights and eight days, and primary caregivers completed online daily diaries via smartphone or computer (90.9%), paper (7.2%), or both (1.5%; .4% missing diary data). Primary caregivers also reported child bedtimes and wake times on a daily assessment table as an additional report of child sleep used for cross-validation when cleaning actigraphy sleep data.

Primary caregivers were contacted approximately one year later when twins were nine years old and offered the opportunity to participate in one home visit consisting of assessments of child and parent pain as well as physical and mental health (N = 278). At home visits for the nine-year assessment, experimenters collected questionnaires, biological measures (height, weight, WC, and percent body fat), conducted interviews with the twins regarding behaviors and emotions, and administered a cold-pressor task with parents and children to assess pain threshold and sensitivity. Sleep was not the focus of the nine-year assessment; as such, objective measures of sleep were not collected.

Families part of the full sample (beginning at 12 months of age) who lived outside the state of Arizona were also invited to participate in the eight-year assessment (N = 40 families); however, materials and assessments that typically occurred in home visits, including sleep

assessment and biological measurements, were not collected from these families. Analyses were conducted including and excluding families who completed the out-of-state protocol, and results were similar such that significant main effects and interactions (and non-significant results) were the same across analyses. As such, families who completed the out-of-state protocol were included in the final analytic sample for the current dissertation.

Measures

Objective Sleep. Objective sleep indicators were assessed and scored using the Micro Motionlogger actigraph wrist watch (Ambulatory Monitoring, Inc., Ardsley, NY). The Micro Motionlogger contains an accelerometer, which captures even small movement throughout the waking day and during sleep periods. Each twin wore an actigraph watch on their non-dominant wrist for seven nights and eight days (M = 6.83 nights, SD = .62). Researchers and study staff scored objective sleep data using the Action-W2 program (version 2.7.1), which uses a validated algorithm to measure sleep (Oakley, 1997). Researchers used the Sadeh algorithm to assess sleep (Sadeh, Hauri, Kripke, & Lavie, 1995; Sadeh, Sharkey, & Carskadon, 1994), with movement measured in one-minute epoch using a zero-crossing mode. Utilizing one-minute epochs and based on significant movement after at least 20 minutes of inactivity, the Sadeh algorithm calculates a variety of sleep parameters. Research suggests that actigraphy is reliable when measuring five more nights of sleep (Acebo et al., 1999), and actigraphy sleep measurement has been validated against concurrent polysomnography (Sadeh et al., 1995). Three sleep variables were used in the current dissertation: sleep duration, sleep efficiency, and sleep midpoint time variability. Nighttime sleep duration represented the total number of hours and minutes asleep each night on average (not counting wake bouts). Sleep efficiency represented the percentage of time asleep each night (excluding wake bouts) based on the total amount of time in bed on average. Sleep midpoint time variability (or midpoint variability) was calculated as the within-person standard deviation estimate of sleep midpoint time of night (time halfway between bedtime and waketime) across the study week on average.

Additionally, study staff cross-checked objective actigraphy sleep periods with parent-reported bedtimes and wake times from daily assessment tables and daily sleep diaries that were

completed by the primary caregiver as an additional sleep-period compliance measure to identify significant outliers and equipment malfunction. Compliance for the eight-year assessment was high, with only 9.8% (*N* = 48) of actigraphy data missing due to loss of actigraph watch or water damage (*N* = 4), watch mechanical malfunction (*N* = 15), children not wearing the watch but participating in other parts of the study week (*N* = 7), and the number of families who participated only in the questionnaire and home visit portion of the study (*N* = 22). Furthermore, of the families who had actigraphy data, 87.3% (*N* = 428) wore the watch for seven or more nights, 9.6% (*N* = 47) had six nights of data, .8% (*N* = 4) had five nights of data, 1.4% (*N* = 7) had four nights of data, and .8% (*N* = 4) had three nights of data. If an individual has fewer than five nights of actigraphy data, this may provide a poor estimation of regular sleep (Acebo et al., 1999). Thus, I conducted exploratory analyses excluding participants with fewer than five nights of sleep (*N* = 11) to determine whether results were similar compared to analyses including all children with available objective sleep data. Results excluding children with fewer than five nights of sleep did not differ from results including all children with available objective sleep data; As such, all cases were included in analyses.

Subjective Sleep. Subjective sleep duration and quality were measured using items from the primary caregiver-report of the Child Sleep Habits Questionnaire at eight years (CSHQ; Owens, Spirito, & McGuinn, 2000). The current dissertation used questions regarding total sleep duration (1 item) and daytime sleepiness (7 items; α = .35). Parent-reported sleep duration (i.e., *Typically, Twin A/B sleeps ___ hours at night.*) represented the total number of hours and minutes each twin slept at night on average. The daytime sleepiness scale assessed difficulty waking up in the morning and frequency of falling asleep during daytime activities (e.g., *Twin A/B has a hard time getting out of bed in the morning*). Daytime sleepiness items were summed to form a single score where higher scores reflected greater daytime sleepiness. Subjective sleep duration and quality served as predictors in phenotypic and behavior genetic models.

Weight Indicators. BMI, WC, and percent body fat were used as outcomes in phenotypic and behavior genetic models. Height, weight, WC, and percent body fat were collected a home visits when twins were eight and nine years old. Height was measured twice

with a tape measure to the nearest half inch at eight years (one measure per visit) and once at nine years of age. At eight years, height measurements were averaged across the two visits to give a single measure of height for each child and the single assessment of height was used for each child at the nine-year assessment.

Weight in pounds was measured using an FDA-approved full body composition scale for children (Tanita Child Scale; Tanita, 2016) at eight and nine years of age. Weight was measured twice at each home visit (four times across two visits) when twins were eight years old, and a third assessment was collected at each visit if the first two weight estimates showed any discrepancies (six possible total measures of weight per child). At nine years, weight was measured twice during the home visit, with a possible third assessment collected if the first two weight estimates showed discrepancies. At eight and nine years, weight estimates were averaged to give each child a single weight estimate at each time point. BMI scores were derived from measures of height and weight taken at home visits at eight and nine years. BMI scores were calculated using the Centers for Disease Control (2015) child BMI formula: weight (kg) / [height (m)]^2.

Percent body fat was also measured using Tanita Child Scale (2016), which utilizes a form of bio-electrical impedance analysis (BIA), a gold-standard in assessing body fat (Davis, 2008). Percent body fat was collected when child weight was assessed, such that percent body fat was assessed up to three times per home visit at eight years (six possible total measures), and up to three times at the nine-year assessment. Percent body fat estimates were averaged across or within visits to provide a single percent body fat score for each twin at each time point.

WC was assessed in inches once at each home visit (two measures total) using a Gulick tape measure (one of the recommended methods for estimating visceral adiposity) at the natural waistline approximately two inches below the lowest rib (Davis, 2008). The Gulick tape measure has a metal counterweight on one end that is activated when placing equal pressure around the body when assessing WC. Experimenters asked children to remove bulky clothing before assessing WC and were trained to have the Gulick tape measure resting directly on the child's clothing with no space between the body or clothing and the tape measure (Davis, 2008). One

end of the Gulick tape measure was pulled around the body to meet the end of the tape with the counterweight, and the counterweight was pulled until it reached a marker signaling equal and constant pressure against the body. WC measures were averaged across visits for each twin to create a single WC score at each time point.

Weight Status. Weight status at eight and nine years (underweight, healthy, overweight, and obese) was calculated using three weight indicators: BMI, WC and percent body fat. The current dissertation used age- and sex-specific centile cut-off scores for each weight indicator. For BMI, children were classified as underweight if scores were less than the 5th percentile, normal or health weight if scores were between the 5th and less than the 85th percentile, overweight if scores were between the 85th and less than the 95th percentile, and obese if scores were greater than the 95th percentile. Children were classified as underweight for WC if scores were less than the 3rd percentile, normal or health weight if scores were between the 3rd and less than the 85th percentile, overweight if scores were between the 85th and less than the 97th percentile, and obese if scores were greater than the 97th percentile. For percent body fat, children were classified as underfat for WC if scores were less than the 2nd percentile, normal or health fat percent if scores were between the 2nd and less than the 85th percentile, overfat if scores were between the 85th and less than the 95th percentile, and obese if scores were greater than the 95th percentile.

Per practices in the pubertal literature (Davison, Susman, & Birch, 2003), children were assigned to a weight status group if they met criteria for a specific weight status category on at least two of the three weight indicators, allowing me to account for weight status using multiple indicators. When combining weight status groups for each child (if they met criteria for the category on at least two of the three weight indicators), 5.4% of children were underweight, 73.8% of children were normal or healthy, 13.3% of children were overweight, and 7.5% of children were obese at eight years of age. At nine years of age, 11.2% of children were underweight, 69.2% of children were normal or healthy, 11.2% of children were overweight, and 8.3% of children were obese. Weight status served as an outcome in phenotypic analyses and behavior genetic analyses (Aim 2b).

Child EC. Child EC at eight and nine years was assessed using the Temperament in Middle Childhood Questionnaire (TMCQ; Simonds, 2006; Putnam & Rothbart, 2006). The current dissertation used the attentional focusing (14 items; α = .90), inhibitory control (7 items; α = .68), and activation control (8 items; α = .69) scales of the TMCQ, which were used to create a higher-order composite of EC (Putnam & Rothbart, 2006). All scale items are rated on a seven-point Likert scale, from 1 (*Extremely Untrue*) to 7 (*Extremely True*) ("NA" was also an option). Items were reverse scored if necessary, and higher scores on the scales indicated more of a certain behavior. In the current dissertation, EC was a moderator in phenotypic models and a predictor in behavioral genetic models.

Zygosity. Zygosity was assessed at 12 months (α = .95) via primary-caregiver reports using the Zygosity Questionnaire for Young Twins (ZQYT; Goldsmith, 1991), a 32-item measure that differentiates between MZ and DZ twins using parent report of birth and observable differences in physical appearance between the twins. Studies have shown parent-reported zygosity (using the ZQYT) is between 93% and 98% accurate in characterizing twin zygosity compared to genotyping, making questionnaires a reliable alternative (Goldsmith, 1991; Forget-Dubois et al., 2003). Families who did not report or participate at the 12-month assessment completed the ZQYT at eight or nine years of age. Zygosity (or twin number) was included in models to account for twin interdependence, as well as to form groups for MZ and DZ twins in quantitative behavioral genetic analyses (i.e., additive genetic paths set to equal 1.0 within MZ cotwin pairs, and 0.5 for DZ cotwin pairs).

Covariates. The following demographic variables at eight years were included in all phenotypic analyses: Age, sex (female = 1), twin ethnicity (European American = 1, all other ethnicities = 0), SES composite (includes income-to-needs ratio, primary caregiver education level, and secondary caregiver education level), whether the child completed the study week during the school year (school break = 1), and parent-reported pubertal development scores (Pubertal Development Scale; Petersen, Crockett, Richards, & Boxer, 1988; α = .96 for males and females). Relevant weight indicators from eight years of age (BMI scores, WC, and percent body fat) also served as covariates in phenotypic models predicting weight indicators at nine

years. Significant covariates (based on zero-order correlations) were included in phenotypic models, and the effects of sex and age were regressed out of variables in quantitative behavior genetic analyses.

Statistical Approach

Preliminary Analyses. Demographic information regarding the sample was collected, including percentages for participant sex, race/ethnicity, zygosity, diary completion, whether study week was completed during the summer or school year, primary caregiver education level, and income-to-needs ratio at eight years, and weight status groups at the eight- and nine-year assessments (See Table 1). Descriptive statistics including raw means, standard deviations, minimums, maximums, skew, and kurtosis for all objective and subjective sleep, weight indicators, EC, and covariates were conducted (See Table 2). Univariate outlier analyses, skew, and kurtosis were examined to determine whether any variables had significant outliers that may bias estimates. Variables with significant outliers and significant skew and/or kurtosis were first windorized to ± 3 *SD*s. If variables still exceeded the recommended cutoff for positive or negative skew (2.00) or kurtosis (7.00; Muthén & Kaplan, 1985), they were transformed either by squaring the scores of the variable (if negatively skewed) or logarithmically transforming the variable (if positively skewed) to approximate a normal distribution of the data for the variable. Parent-reported daytime sleepiness at eight years, BMI at eight years, and WC at nine years all contained significant outliers, as well as skew and kurtosis that exceeded the recommended cutoffs (Muthén & Kaplan, 1985). As such, parent-reported daytime sleepiness at eight years, BMI at eight years, and WC at nine years were windorized at ± 3 *SD*s to approximate a more normal distribution of each variable and reduce skew and kurtosis. Windorizing variables successfully dispelled significant positive skew and kurtosis for BMI at eight years and WC at nine years; however, parent-reported daytime sleepiness remained positively skewed and kurtotic after windorizing at ± 3 *SD*s. Thus, parent-reported daytime sleepiness at eight years was also logarithmically transformed (after being windorized) to approximate a normal distribution of the data. Overall, windorized and/or transformed parent-reported daytime sleepiness at eight years,

BMI at eight years, and WC at nine years were used in phenotypic analyses, and raw forms of all other variables were used as they were normally distributed.

Zero-order correlations between predictor, outcome, and covariate variables were conducted (See Table 3), and twin intra-class correlations (ICCs; see Table 4) were reported to show the extent to which sleep, weight indicators, and EC were heritable and provide a basis and context for the univariate, bivariate, and multivariate behavior genetic models (Aim 2). Twin ICCs were conducted in SPSS 24 (IBM), as twin ICCs are typically conducted in a twin-level file by testing bivariate correlations while excluding either Twin 1 or 2's data from the bivariate correlation. If MZ twin ICCs are higher than DZ ICCs, this suggests a genetic influence on individual differences in particular traits (higher heritability). Approximately equal or similar MZ and DZ ICCs indicate influence of the shared and nonshared environment, as well as capturing measurement error, on a particular trait (lower heritability).

Aim 1a and b. Mixed model and multivariate logistic regression analyses were conducted in Mplus 7 (Muthén & Muthén, 1998-2012; version 7.11) using the *complex* command and full information maximum likelihood with robust standard errors (MLR; recommended when using *complex* command) to account for twin interdependence, non-normality, and missing data. Mixed model regression analyses examined associations between a) objective and subjective sleep at eight years and weight indicators at eight years of age, as well as whether EC moderated these associations, and b) objective and subjective sleep at eight years and weight indicators at nine years of age, as well as whether EC moderated these associations. Multivariate logistic regression analyses tested whether objective and subjective sleep at eight years predicted odds of being overweight or obese at both eight and nine years of age, and if links between sleep and weight indicators differed by child EC level at eight years. When used as predictors in mixed model or multivariate logistic regression analyses, objective and subjective sleep indicators and EC were centered at zero, and unstandardized beta estimates, standard errors, confidence intervals, and *p*-values were reported. For significant interactions in mixed model regression analyses, simple slopes were plotted at 1 *SD* below and above the mean of EC at eight years

using the simple slopes technique for 2-way interactions with nested data outlined by Preacher and colleagues (2006).

Aim 2a. Univariate and Bivariate Cholesky Decomposition models (i.e., ACE models) were conducted in OpenMx (Boker et al., 2011), an R-based program that estimates genetic and environmental variance and covariance (e.g., between sleep duration and BMI scores) using structural equation models with maximum likelihood estimation and allowance for missing data. With all quantitative behavior genetic models, additive genetic influences on a phenotype were set to correlate 1.0 for MZ twins and .5 for DZ twins. Shared environmental influences affect MZ and DZ twins (reared together) to the same degree regardless of genetic relatedness and were set to correlate 1.0 across twins for both MZ and DZ groups. Nonshared environmental variance encompasses all non-genetic factors that reduce phenotypic covariance between cotwins, including measurement error, and were uncorrelated across MZ and DZ cotwins. Significance of all A and C parameters in each quantitative behavior genetic model were tested by systematically dropping the A parameter, C parameter, and then both A and C parameters from the model and comparing the fit of full and reduced models. Because the E parameter contains measurement error, it was not dropped from any models. Full and reduced models were compared using the -2 log likelihood chi-squared test of fit (-2LL or χ^2), as well as chi-square different tests (or log likelihood tests; indicated by Δ -2LL) which compared model fit of nested models. Non-significant *p*-values for the -2LL difference test indicated that a reduced model did not fit the data significant worse compared to the full model with all paths estimated (better model fit). In contrast, significant *p*-values for the -2LL difference test indicated that the reduced model fit the data significantly worse compared to the full model with all paths estimated. Akaike's Information Criterion (AIC; Akaike, 1974), which penalizes models with a larger number of parameters, was also used to assess model fit. Lower AIC values indicated better model fit. Overall, -2LL, degrees of freedom, AIC, change in degrees of freedom, change in -2LL and the *p*-value were reported for each full and best-fitting (full or reduced) behavior genetic model.

Appropriate Univariate and Bivariate Cholesky Decompositions were conducted with various objective and subjective sleep variables, weight indicators, and EC at eight years to

decompose the covariance between three traits into unique and shared A, C, and E components by examining cross-twin cross-trait covariances separately for MZ and DZ twin groups (multivariate example, Figure 3). Univariate behavior genetic models were conducted (See Figure 4) first with all sleep, weight, and EC variables at eight years of age to provide a basis for multivariate models. Second, Bivariate Cholesky Decompositions were estimated to demonstrate genetic and environmental contributions to the variance for individual phenotypes, as well as decompose any covariance shared between two phenotypes. Finally, if there was no phenotypic association or a weak association (correlations less than .20 will be reviewed) between sleep, weight indicators, and EC conducted in Aim 1, it may not be appropriate to conduct behavior genetic models. As such, zero-order correlations and phenotypic results from Aim 1 directed the total number and type of Univariate and Bivariate Cholesky Decompositions that were conducted in Aim 2a.

Aim 2b. The Multivariate (trivariate) Cholesky Decomposition estimated unique additive genetic (A3), shared environmental (C3) and nonshared environmental (E3) influences on weight indicators such as BMI. In the same model, additive genetic (A1 and A2), common environmental (C1 and C2), and nonshared environmental (E1 and E2) influences on weight indicators like BMI that are *shared* with objective and subjective sleep parameters (e.g., sleep duration) and EC were also estimated. For comparison, Independent Pathway Models were fit to determine whether there was a common underlying set of genes (A_s), a single shared environmental factor (C_s) or a common nonshared environmental factor (E_s) that accounts for associations among traits (Figure 4). In the same model, unique additive genetic (A1, A2, and A3) and nonshared environmental factors (E1, E2, and E3) that contribute to a particular trait, independent of other traits or behaviors, was estimated. For example, we can test whether there is an underlying additive genetic factor that explains links between objective sleep duration, BMI, and EC (A_s), or whether a single shared environmental or psychosocial factor (C_s or E_s) such as general dysregulation can explain associations between sleep duration, BMI, and EC. Multivariate behavior genetic models were only conducted for significantly correlated sleep variables and weight indicators to test the extent to which sleep or weight indicators share the same or different genetic etiologies.

Aim 2c. Finally, one Liability Threshold Model was fit to test the extent to which additive genetic and environmental influences on a weight status may differ across groups of individuals (Plomin et al., 2013; Figure 5). The Liability Threshold Model uses the same assumptions as the typical ACE model and estimates differences in additive genetic, shared environmental and nonshared environmental factors between weight status group (i.e., whether additive genetic influences are stronger or weaker for different groups). However, given that the Liability Threshold Model does not utilize continuous data, degrees of freedom are less than what is required to estimate model fit. To account for this, Liability Threshold Models traditionally constrain the total variance estimated (V) to 1, such that A + C + E = 1 allowing models to be estimated. This approach also assumes and estimates the data as normally distributed. Overall, the Liability Threshold Model was conducted in two major steps. First, the full univariate model was fit, including estimating and calculating thresholds and a predicted correlations matrix for MZ twins. For the univariate model, child weight status was obtained from Aim 1, and the liability threshold model contained cut points between each of the weight status groups, such that underweight to healthy weight = 0, healthy weight to overweight = 1, and overweight to obese = 2. Thresholds and correlations were calculated in OpenMx when fitting the univariate model, and two thresholds were estimated given that there are three categories or groups in the data. Second, multiple submodels were tested by constraining thresholds across twins and zygosity groups and systematically dropping A parameters, C parameters, and then both A and C parameters, and the full model was compared to the reduced models to determine the best fitting model (just as with other quantitative behavior genetic models).

CHAPTER 3

RESULTS

Preliminary Analyses

Descriptive Statistics. Means, standard deviations, minimums, maximums, skewness and kurtosis for all key variables are presented in Table 2. On average, parents reported that children slept approximately nine hours and 39 minutes each night (*SD* = 52 minutes), whereas objective sleep duration measurement showed that children slept about eight hours and five

minutes each night (*SD* = 44 minutes). Children also showed adequate sleep quality, spending about 90% of their time in bed each night sleeping. Regarding weight indicators, children showed an average BMI score of about 16.86 (*SD* = 2.94 points; windorized BMI: *M* = 16.79, *SD* = 2.68 points) at eight years and 17.40 at nine years of age (*SD* = 3.28 points), which falls within the normal or healthy range for both males and females at these ages. Children also demonstrated an average WC of 22.82 inches (*SD* = 3.00 inches) at eight years and 23.19 inches at nine years of age (*SD* = 4.08 inches; windorized WC: *M* = 23.09, *SD* = 3.61 inches), and an average percent body fat of 20.23 (*SD* = 6.45 percentage points) at eight years and 20.38 at nine years of age (*SD* = 7.36 percentage points).

Correlations. Zero-order correlations for the analytic sample are presented in Table 3. Parent-reported sleep duration was negatively associated with daytime sleepiness, sleep midpoint variability, and BMI and percent body fat at eight years of age. Conversely, parent-reported sleep duration was positively associated with objective sleep duration and EC at eight years. Objective nighttime sleep duration was positively associated with objective sleep efficiency, sleep midpoint variability, and EC at eight years. Both objective sleep duration and efficiency were significantly negatively correlated with BMI, WC, percent body fat, and weight status at eight and nine years of age. All weight indicators and weight status were significantly positively correlated with each other at eight and nine years of age, as well as correlated across time points suggesting high stability in weight estimates from eight to nine years of age for children. EC was negatively related to BMI at eight and nine years and WC at nine years of age.

Regarding demographic variables, females showed longer objective sleep duration, greater sleep efficiency, greater percent body fat at eight and nine years of age, and greater EC at eight years compared to male children. European American/White participants in the study showed greater parent-reported and objective sleep duration, lower sleep midpoint variability, lower BMI and percent body fat at eight and nine years of age, and smaller WC at eight years compared to non-European American/White participants. Participant who completed their study week during a school break (rather than the school year) showed lower sleep midpoint variability. Children who demonstrated greater pubertal development or higher pubertal stage at eight years

of age showed longer objective sleep duration and greater efficiency, higher EC at eight years, and greater BMI, WC, percent body fat and were classified as overweight/obese at both eight and nine years compared to children who were not as far along in pubertal development. Finally, children from higher SES backgrounds at eight years also showed longer parent-reported and objective sleep duration, greater sleep efficiency, lower midpoint variability, greater EC, and lower BMI, WC, percent body fat, and were classified as underweight or normal weight (rather than overweight/obese) at both eight and nine years of age.

Twin ICCs. Twin intra-class correlations (ICCs) were conducted to examine whether identical twins were more similar to each other than fraternal twins (see Table 4 for complete twin ICCs on key study variables). Twin ICCs indicated that MZ twins were more similar particularly on objective sleep duration (ICC = .84) and sleep efficiency (ICC = .84) at eight years compared to DZ twins (sleep duration: ICC = .46; sleep efficiency: ICC = .46). MZ twins were also more similar on EC at eight years (ICC = .73) than DZ twins (ICC = .43). Finally, MZ twins were considerably more similar on BMI (MZ = .92; DZ = .30), WC (MZ = .90; DZ = .35), and percent body fat estimates (MZ = .93; DZ = .29) compared to DZ twins at both eight and nine years of age (BMI at nine years: MZ = .81; DZ = .37; WC at nine years: MZ = .84; DZ = .48; Percent body fat at nine years: MZ = .85; DZ = .38).

Aim 1a Results

Parent-reported Sleep Duration and Weight Indicators. The interaction between parent-reported sleep duration and EC at eight years was marginally significant in predicting concurrent BMI (*b* = .46*,* {95% CI, -.03, .96}, *SE* = .25, *p* = .07). Simple slopes were probed at ±1 *SD* of EC at eight years to test the interaction, but there were no significant differences in associations between parent-reported sleep duration and BMI at eight years based on high, mean, and low levels of child EC (all *p*s > .05). However, in the model with parent-reported sleep duration at eight years predicting concurrent BMI, greater EC (*b* = -.55*,* {95% CI, -1.04, -.07}, *SE* = .25, *p* = .03) and higher SES (*b* = -.49*,* {95% CI, -.92, -.05}, *SE* = .22, *p* = .03) were associated with lower BMI scores, and greater pubertal development was associated with higher BMI scores at eight years (*b* = 2.49*,* {95% CI, 1.23, 3.75}, *SE* = .64, *p* < .001).

Parent-reported sleep duration and the interaction between parent-reported sleep duration and EC at eight years did not predict concurrent WC, percent body fat or weight status. However, there was as significant main effect of pubertal status in all models, with greater pubertal development was associated with larger WC (b = 2.94, {95% CI, 1.49, 4.39}, SE = .74, p < .001), greater percent body fat (b = 5.82, {95% CI, 2.95, 8.68}, SE = 1.46, p < .001), and a seven-fold increase in the odds of being classified as overweight/obese at eight years (b = 2.00, {95% CI, 1.11, 3.00}, SE = .48, p < .001). In the percent body fat model, higher SES was associated with lower percent body fat (b = -1.38, {95% CI, -2.40, -.36}, SE = .52, p < .01). European American/White participants also showed lower percent body fat compared to children from other racial/ethnic backgrounds (b = -1.65, {95% CI, -3.15, -.14}, SE = .77, p = .03), and females showed significantly higher percent body fat at eight years (b = 1.69, {95% CI, .45, 2.93}, SE = .63, p < .01).

Daytime Sleepiness and Weight Indicators. The interaction between parent-reported daytime sleepiness and EC at eight years marginally predicted concurrent percent body fat at the trend level (b = -2.48, {95% CI, -3.15, -.14}, SE = 1.51, p = .10). Simple slopes were probed at ± 1 SD of EC at eight years to test the interaction, but there were no significant differences in associations between parent-reported daytime sleepiness and percent body fat at eight years based on high, mean, and low levels of child EC (all ps > .05). However, in the percent body fat model, higher SES was associated with lower percent body fat (b = -1.37, {95% CI, -2.41, -.32}, SE = .53, p = .01), and greater pubertal development was associated with greater percent body fat at eight years (b = 5.86, {95% CI, 2.97, 8.76}, SE = 1.48, p < .001). European American/White participants also showed lower percent body fat compared to children from other racial/ethnic backgrounds (b = -2.00, {95% CI, -3.42, -.57}, SE = .73, p < .01), and females showed significantly higher percent body fat at eight years (b = 1.70, {95% CI, .44, 2.96}, SE = .64, p < .01).

Similar to parent-reported sleep duration, neither parent-reported daytime sleepiness nor the interaction between daytime sleepiness and EC at eight years predicted concurrent BMI, WC, or weight status. There was a significant main effect of pubertal status in all models, such that

greater pubertal development was associated with higher BMI (*b* = 2.49*,* {95% CI, 2.49, 3.76}, *SE* = .65, *p* < .001), larger WC (*b* = 2.92*,* {95% CI, 1.46, 4.39}, *SE* = .75, *p* < .001), and a seven-fold increase in the odds of being classified as overweight/obese at eight years (*b* = 1.95*,* {95% CI, 1.01, 2.89}, *SE* = .48, *p* < .001). Additionally, greater SES was associated with lower BMI (*b* = -.41, {95% CI, -.93, -.02}, *SE* = .30, *p* = .04), completing the study week during a school break was associated with a 70% increase in the odds of being classified as overweight or obese (*b* = .53, {95% CI, .01, 1.05}, *SE* = .27, *p* = .05), and greater daytime sleepiness was associated with 72% reduced odds of being classified as overweight/obese at eight years (*b* = -1.33, {95% CI, -3.15, -.14}, *SE* = .59, *p* = .02).

Objective Sleep Duration and Weight Indicators. Longer objective nighttime sleep duration was associated with lower BMI (*b* = -.53, {95% CI, 2.49, 3.76}, *SE* = .18, *p* < .01), smaller WC (*b* = -.51,{95% CI, -.91, -.11}, *SE* = .20, *p* < .01), lower percent body fat (*b* = -1.42, {95% CI, -2.29, -.56}, *SE* = .44, *p* < .001), and 38% reduced odds of being classified as overweight/obese at eight years (*b* = -.49, {95% CI, -.86, -.11}, *SE* = .19, *p* = .01). Greater pubertal development was associated with higher BMI (*b* = 2.67*,* {95% CI, 1.37, 3.97}, *SE* = .66, *p* < .001), larger WC (*b* = 3.12*,* {95% CI, 2.49, 3.76}, *SE* = .76, *p* < .001), greater percent body fat (*b* = 6.34*,* {95% CI, 1.64, 4.60}, *SE* = 1.52, *p* < .001), and over a seven-fold increase in the odds of being classified as overweight/obese at eight years (*b* = 2.10*,* {95% CI, 1.14, 3.07}, *SE* = .65, *p* < .001).

Furthermore, the BMI model showed that greater EC (*b* = -.44*,* {95% CI, -.91, .03}, *SE* = .65, *p* = .06) and SES (*b* = -.43*,* {95% CI, -.88, .02}, *SE* = .65, *p* = .06) were marginally associated with lower BMI scores at eight years. Regarding percent body fat model, higher SES was associated with lower percent body fat (*b* = -1.21*,* {95% CI, -2.24, -.18}, *SE* = .53, *p* = .02). European American/White participants also showed lower percent body fat compared to children from other racial/ethnic backgrounds (*b* = -1.60*,* {95% CI, -2.99, -.20}, *SE* = .71, *p* = .03) and females showed significantly higher percent body fat at eight years (*b* = 1.87*,* {95% CI, .61, 3.14}, *SE* = .65, *p* < .01). Finally, completing the study week during a school break was associated with

a 71% increase in the odds of being classified as overweight or obese (*b* = .54, {95% CI, .01, 1.07}, *SE* = .27, *p* = .05).

Objective Sleep Efficiency and Weight Indicators. Greater objective sleep efficiency was associated with lower BMI (*b* = -.07, {95% CI, -.12, -.02}, *SE* = .03, *p* < .01), smaller WC (*b* = -.08, {95% CI, -.13, -.03}, *SE* = .03, *p* < .01), lower percent body fat (*b* = -.16, {95% CI, .01, 1.07}, *SE* = .06, *p* < .01), and 5% reduced odds of being classified as overweight/obese at eight years (*b* = -.05, {95% CI, -.27, -.04}, *SE* = .02, *p* = .02). Greater pubertal development was associated with higher BMI (*b* = 2.70, {95% CI, 1.40, 3.99}, *SE* = .66, *p* < .001), larger WC (*b* = 3.18, {95% CI, 1.71, 4.66}, *SE* = .75, *p* < .001), greater percent body fat (*b* = 6.37, {95% CI, 3.42, 9.32}, *SE* = 1.51, *p* < .001), and an eight-fold increase in the odds of being classified as overweight/obese at eight years (*b* = 2.11, {95% CI, 1.14, 3.08}, *SE* = .49, *p* < .001).

Furthermore, greater EC was associated with lower BMI (*b* = -.49, {95% CI, -.96, -.02}, *SE* = .24, *p* = .04). Higher SES (*b* = -.39, {95% CI, -.85, .07}, *SE* = .23, *p* = .09) and older age at eight years (*b* = .46, {95% CI, -.01, .92}, *SE* = .24, *p* = .06) were marginally associated with lower BMI scores at eight years. Greater EC was also associated with marginally smaller WC at eight years (*b* = -.47, {95% CI, -1.03, .09}, *SE* = .28, *p* = .10). Regarding percent body fat model, higher SES was associated with lower percent body fat (*b* = -1.15, {95% CI, -2.20, -.11}, *SE* = .53, *p* = .03). European American/White participants also showed lower percent body fat compared to children from other racial/ethnic backgrounds (*b* = -1.88, {95% CI, -3.30, -.47}, *SE* = .72, *p* < .01) and females showed significantly higher percent body fat (*b* = 1.79, {95% CI, .52, 3.06}, *SE* = .65, *p* < .01), and marginally lower WC at eight years (*b* = -.58, {95% CI, -1.19, .02}, *SE* = .31, *p* = .06). Finally, completing the study week during a school break was associated with a 71% increase in the odds of being classified as overweight or obese (*b* = .53, {95% CI, .01, 1.06}, *SE* = .27, *p* = .05) and marginally larger WC (*b* = .59, {95% CI, -.11, 1.29}, *SE* = .36, *p* = .10).

Objective Sleep Midpoint Variability and Weight Indicators. Neither objective sleep midpoint variability nor the interaction between midpoint variability and EC at eight years predicted concurrent BMI, WC, percent body fat or weight status. There was a significant main effect of pubertal status in all models, such that greater pubertal development was associated

with higher BMI (*b* = 2.40*,* {95% CI, 1.09, 3.71}, *SE* = .67, *p* < .001), larger WC (*b* = 2.86*,* {95% CI, 1.36, 4.37}, *SE* = .77, *p* < .001), greater percent body fat (*b* = 5.72, {95% CI, 2.74, 8.71}, *SE* = 1.52, *p* < .001), and over a six-fold increase in the odds of being classified as overweight/obese at eight years (*b* = 1.87*,* {95% CI, .93, 2.81}, *SE* = .48, *p* < .001). Greater EC predicted lower BMI (*b* = -.54, {95% CI, -1.02, -.05}, *SE* = .25, *p* = .03) and marginally smaller WC at eight years (*b* = -.52, {95% CI, -1.09, .05}, *SE* = .29, *p* = .07). Older age was also associated greater BMI at eight years (*b* = .52, {95% CI, .07, .97}, *SE* = .23, *p* = .03). Higher SES was associated with lower BMI (*b* = -.53, {95% CI, -.99, -.08}, *SE* = .23, *p* = .02) and percent body fat at eight years of age (*b* = -1.41*,* {95% CI, -2.48, -.35}, *SE* = .54, *p* < .01). European American/White participants also showed lower percent body fat compared to children from other racial/ethnic backgrounds (*b* = -2.01*,* {95% CI, -3.48, -.54}, *SE* = .75, *p* < .01) and females showed significantly higher percent body fat (*b* = 1.68*,* {95% CI, .40, 2.97}, *SE* = .65, *p* = .01) and smaller WC at eight years of age (*b* = -.63*,* {95% CI, -1.24, -.02}, *SE* = .31, *p* = .04). Finally, completing the study week during a school break was associated with a 71% increase in the odds of being classified as overweight or obese (*b* = .54, {95% CI, .01, 1.07}, *SE* = .27, *p* = .05).

Aim 1b Results

Parent-reported Sleep Duration and Weight Indicators. The interaction between parent-reported sleep duration and EC at eight years predicted BMI at nine years (*b* = .34*,* {95% CI, .01, 67}, *SE* = .17, *p* < .05). Simple slopes were probed at ±1 *SD* of EC at eight years to test the interaction (Figure 6), but there were no significant differences in associations between parent-reported sleep duration at eight years and BMI at nine years based on high, mean, and low levels of child EC (all *p*s > .05). In the same model, completing the study week during a school break predicted lower BMI at nine years (*b* = -.47*,* {95% CI, -.81, -.13}, *SE* = .17, *p* < .01), and greater BMI at eight years predicted greater BMI at nine years (*b* = 1.15*,* {95% CI, 1.05, 1.25}, *SE* = .05, *p* < .001). However, parent-reported sleep duration and the interaction between parent-reported sleep duration and EC at eight years did not predict WC, percent body fat or weight status at nine years. Rather, greater EC at eight years predicted smaller WC at nine years (*b* = -1.01*,* {95% CI, -1.55, -.48}, *SE* = .27, *p* < .001), and greater WC (*b* = .80*,* {95% CI, .67, .93},

SE = .07, *p* < .001) and pubertal development (*b* = 1.86, {95% CI, .46, 3.26}, *SE* = .71, *p* < .001) at eight years predicted greater WC at nine years. Similarly, greater percent body fat at eight years predicted greater percent body fat at nine years (*b* = 1.02, {95% CI, .94, 1.10}, *SE* = .04, *p* < .001), and greater likelihood of being overweight/obese at eight years prospectively predicted overweight/obesity status at nine years of age (*b* = 4.61, {95% CI, 3.49, 5.74}, *SE* = .57, *p* < .001). Finally, completing the study week during a school break at eight years was associated with 69% reduced odds of being classified as overweight or obese (*b* = -1.18, {95% CI, -2.30, -.06}, *SE* = .57, *p* = .04).

Daytime Sleepiness and Weight Indicators. Parent-reported daytime sleepiness and the interaction between parent-reported daytime sleepiness and EC at eight years did not predict BMI, WC, percent body fat or likelihood of being classified as overweight/obese at nine years of age. In the BMI model, completing the study week during a school break (*b* = -.44, {95% CI, -.78, -.10}, *SE* = .17, *p* = .01) and older age at eight years predicted marginally lower BMI at nine years (*b* = -.40, {95% CI, -.82, .02}, *SE* = .21, *p* = .06). Greater pubertal development at eight years was associated with larger WC at nine years (*b* = 1.92, {95% CI, .51, 3.33}, *SE* = .72, *p* = .01). In addition, greater BMI, WC, percent body fat, and likelihood of being overweight/obese at eight years predicted greater BMI, WC, percent body fat, and likelihood of being overweight/obese at nine years, respectively (BMI: *b* = 1.15, {95% CI, 1.06, 1.26}, *SE* = .05, *p* < .001; WC: *b* = .79, {95% CI, .66, .93}, *SE* = .07, *p* < .001; Percent body fat: *b* = 1.03, {95% CI, .95, 1.11}, *SE* = .04, *p* < .001; Overweight/obesity status: *b* = 4.75, {95% CI, 3.58, 5.92}, *SE* = .60, *p* < .001).

Objective Sleep Duration and Weight Indicators. The interaction between objective sleep duration and EC at eight years predicted BMI at nine years (*b* = .46, {95% CI, .08, .84}, *SE* = .19, *p* = .02), such that greater objective sleep duration at eight years predicting lower BMI scores for children with low EC (*b* = -.29, *SE* = .13, *p* = .02), but not average (*b* = -.05, *SE* = .09, *p* = ns) or high EC at eight years (*b* = .20, *SE* = .15, *p* = ns; Figure 7). In the same model, greater BMI at eight years predicted greater BMI at nine years (*b* = 1.15, {95% CI, 1.05, 1.25}, *SE* = .05, *p* < .001). Older age (*b* = -.36, {95% CI, -.77, .05}, *SE* = .21, *p* = .09) and being female predicted marginally lower BMI scores (*b* = -.27, {95% CI, -.60, .05}, *SE* = .17, *p* < .10). However, objective

sleep duration and the interaction between objective sleep duration and EC at eight years did not predict WC, percent body fat or weight status at nine years. Completing the study week during a school break at eight years predicted lower BMI (*b* = -.44, {95% CI, -.79, -.10}, *SE* = .17, *p* = .01) and marginally lower odds of being overweight or obese at nine years (*b* = -1.04, {95% CI, -.2.14, .05}, *SE* = .56, *p* = .06). Greater EC at eight years predicted smaller WC at nine years (*b* = -1.00, {95% CI, -1.55, -.45}, *SE* = .28, *p* < .001), and greater pubertal development at eight years predicted greater WC at nine years (*b* = 1.94, {95% CI, .52, 3.37}, *SE* = .73, *p* < .01). Additionally, greater WC, percent body fat, and likelihood of being overweight/obese at eight years predicted greater WC, percent body fat, and likelihood of being overweight/obese at nine years, respectively (WC: *b* = .79, {95% CI, .66, .92}, *SE* = .07, *p* < .001; Percent body fat: *b* = 1.01, {95% CI, .93, 1.09}, *SE* = .04, *p* < .001; Overweight/obesity status: *b* = 4.70, {95% CI, 3.51, 5.89}, *SE* = .61, *p* < .001).

Objective Sleep Efficiency and Weight Indicators. Objective sleep efficiency and the interaction between sleep efficiency and EC at eight years did not predict BMI, percent body fat, or likelihood of being classified as overweight/obese at nine years of age. However, completing the study week during a school break predicted lower BMI (*b* = -.45, {95% CI, -2.19, .04}, *SE* = .18, *p* = .01) and marginally lower odds of being overweight or obese (*b* = -1.08, {95% CI, -.79, .06}, *SE* = .58, *p* = .06). Older age at eight years predicted marginally lower BMI at nine years (*b* = -.37, {95% CI, -.79, .06}, *SE* = .17, *p* = .09). Greater EC at eight years predicted smaller WC at nine years (*b* = -.99, {95% CI, -1.55, -.44}, *SE* = .28, *p* < .001), and greater pubertal development at eight years predicted greater WC at nine years (*b* = 2.01, {95% CI, .51, 3.52}, *SE* = .77, *p* < .01). In addition, greater BMI, WC, percent body fat, and likelihood of being overweight/obese at eight years predicted greater BMI, WC, percent body fat, and likelihood of being overweight/obese at nine years, respectively (BMI: *b* = 1.15, {95% CI, 1.05, 1.25}, *SE* = .05, *p* < .001; WC: *b* = .79, {95% CI, .65, .92}, *SE* = .07, *p* < .001; Percent body fat: *b* = 1.02, {95% CI, .93, 1.10}, *SE* = .04, *p* < .001; Overweight/obesity status: *b* = 4.71, {95% CI, 3.15, 5.89}, *SE* = .61, *p* < .001).

Objective Sleep Midpoint Variability and Weight Indicators. The interaction between objective sleep midpoint variability and EC at eight years marginally predicted BMI at nine years (*b* = -.86*,* {95% CI, .65, .92}, *SE* = .49, *p* = .08), such that greater objective midpoint variability at eight years predicted higher BMI scores for children with low EC (*b* = 2.19*, SE* = 1.08, *p* = .04), but not average (*b* = .49, *SE* = .81, *p* = ns) or high EC at eight years (*b* = -1.22, *SE* = 1.06, *p* = ns). In the same model, greater BMI at eight years predicted greater BMI at nine years (*b* = 1.16*,* {95% CI, .65, .92}, *SE* = .05, *p* < .001), and older age at eight years predicted marginally lower BMI at nine years (*b* = -.38*,* {95% CI, .65, .92}, *SE* = .22, *p* = .08). Similarly, the interaction between sleep midpoint variability and EC at eight years predicted percent body fat at nine years (*b* = -3.15*,* {95% CI, -5.70, -.61}, *SE* = 1.30, *p* = .02), such that greater midpoint variability at eight years predicted greater percent body fat for children with low EC (*b* = 2.26*,* {95% CI, .65, .92}, *SE* = .07, *p* = .05; Figure 8), but not average (*b* = .49, {95% CI, -1.10, 2.07}, *SE* = .81, *p* = ns) or high EC at eight years (*b* = -1.04, {95% CI, .65, .92}, *SE* = .07, *p* = ns). In the same model, greater percent body fat at eight years predicted greater percent body fat at nine years (*b* = 1.03*,* {95% CI, .95, 1.10}, *SE* = .04, *p* < .001). Objective sleep midpoint variability and the interaction between midpoint variability and EC at eight years did not predict WC or the of likelihood of being classified as overweight/obese at nine years of age. However, greater EC at eight years predicted smaller WC at nine years (*b* = -1.00*,* {95% CI, -1.55, -.45}, *SE* = .28, *p* < .001), and greater pubertal development at eight years predicted greater WC at nine years (*b* = 1.78*,* {95% CI, .35, 3.22}, *SE* = .73, *p* = .02). Completing the study week during a school break predicted marginally lower odds of being overweight/obese at nine years (*b* = -.96, {95% CI, -2.06, .14}, *SE* = .56, *p* = .09). Finally, greater WC and likelihood of being overweight/obese at eight years predicted greater WC and likelihood of being overweight/obese at nine years, respectively (WC: *b* = .80*,* {95% CI, .67, .93}, *SE* = .07, *p* < .001; Overweight/obesity status: *b* = 4.68*,* {95% CI, 3.53, 5.83}, *SE* = .59, *p* < .001).

Aim 2a Results

Sleep Indicators. The full univariate ACE model for parent-reported sleep duration at eight years was a good fit for the data, -2LL(565) = 1112.35, AIC = -17.65 (see Tables 5 for full fit

statistics). Using the ACE model fit, the standardized variance components were estimated, such that the greatest proportion of the variance in parent-reported sleep duration was accounted for by the shared environmental factor (c^2 = .66), with the little remaining variance accounted for by additive genetic (a^2 = .21) and non-shared environmental contributions (e^2 = .13; see Table 6 for full estimates).

Similarly, the full univariate ACE model for parent-reported daytime sleepiness at eight years was a good fit for the data, -2LL(575) = -72.73, AIC = -1222.73 (see Tables 5 for full fit statistics). Using the ACE model fit, the greatest proportion of the variance in parent-reported daytime sleepiness was accounted for by the shared environmental factor (c^2 = .66), with the little remaining variance accounted for by additive genetic (a^2 = .27) and non-shared environmental contributions (e^2 = .07; see Table 6 for full estimates).

The full univariate ACE model for objective nighttime sleep duration at eight years was a good fit for the data, -2LL(455) = 889.95, AIC = -20.05 (see Tables 5 for full fit statistics). However, the reduced AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may fit the data best, -2LL(456) = 897.03, AIC = -21.44, Δ -2LL = .60, p = .44. The standardized variance components for nighttime sleep duration were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in nighttime sleep duration was accounted for by additive genetic factors (a^2 = .81), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .19; see Table 6 for full estimates).

Likewise, the full univariate ACE model for objective sleep efficiency at eight years was a good fit for the data, -2LL(455) = 2819.34, AIC = 1909.22 (see Tables 5 for full fit statistics). However, the reduced AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may also fit the data well, -2LL(456) = 2821.43, AIC = 1909.43, Δ -2LL = 2.21, p = .14. The standardized variance components for sleep efficiency were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in sleep efficiency was accounted for by additive genetic factors (a^2 = .79), with remaining variance

accounted for by the non-shared environmental contribution (e^2 = .21; see Table 6 for full estimates).

Finally, the full univariate ACE model for objective sleep midpoint variability at eight years was a good fit for the data, -2LL(455) = -41.80, AIC = -951.80 (see Tables 5 for full fit statistics). However, the reduced CE model did not fit significantly worse than the full ACE model, suggesting that the CE model may also fit the data well, -2LL(456) = -40.61, AIC = -952.61, Δ -2LL = 1.19, p = .27. The standardized variance components for sleep midpoint variability were estimated based on the reduced CE model, and showed that the greatest proportion of the variance in sleep midpoint variability was accounted for by shared environmental factors (c^2 = .77), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .23; see Table 6 for full estimates).

EC. The full univariate ACE model for EC at eight years was also a good fit for the data, -2LL(513) = 723.29, AIC = -302.71 (see Tables 5 for full fit statistics). However, the reduced AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may also fit the data well, -2LL(514) = 723.68, AIC = -304.32, Δ -2LL = .39, p = .53. The standardized variance components for EC were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in EC was accounted for by additive genetic factors (a^2 = .76), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .24; see Table 6 for full estimates).

Weight Indicators. Twin intra-class correlations for weight indicators suggest large differences between DZ same-sex twins and DZ opposite-sex twins, such that the magnitude of genetic or environmental effects (quantitative sex-limitation model) *or* the actual genetic and environment effects (i.e., variance accounted for by genetics or environment; qualitative sex-limitation model) may differ between males and females on weight indicators. Given these estimates, multiple Univariate Cholesky Decomposition Sex-limitation Models were conducted for BMI, WC, and percent body fat at eight years of age to determine whether genetic and environmental influences on each weight indicators differed by sex and the best fitting model. Specifically, a general scalar sex-limitation model was conducted, which allows the proportion of

variance accounted for by A, C, and E components to change based on a scalar (i.e., k) for males and females and the total variance to differ across males and females (Bartels, 2016). The general scalar sex-limitation model also suggests differences in both the magnitude and nature of genetic and environmental influences on a trait for males and females. A non-scalar, sex-limitation model was also conducted, which allows the proportion of variance accounted for by A, C, E, and the total variance to differ across males and females, so all parameters are estimated separately (Bartels, 2016). In contrast to the general scalar sex-limitation model, the non-scalar model specifically estimates magnitude differences in genetic effects between males and females (Bartels, 2016). Finally, A and C paths were dropped for scalar and non-scalar sex-limitation models to determine the best fit for the data.

The full univariate general scalar sex-limitation ACE model for objective BMI scores at eight years was a good fit for the data, -2LL(465) = 2106.05, AIC = 1176.05 (see Tables 7 for full fit statistics). However, the reduced general scalar sex-limitation AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may also fit the data well, -2LL(467) = 2108.77, AIC = 1174.77, Δ -2LL = 2.72, p = .25. The standardized variance components for BMI for males were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in BMI was accounted for by additive genetic factors (a^2 = .95), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .05; see Table 8 for full estimates). The standardized variance components for BMI for females estimated based on the reduced AE model showed that the greatest proportion of the variance in BMI was accounted for by additive genetic factors (a^2 = .90), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .10; see Table 8 for full estimates).

Similarly, full univariate general scalar sex-limitation ACE model for WC at eight years was a good fit for the data, -2LL(462) = 2217.20, AIC = 1293.20 (see Tables 7 for full fit statistics). However, the reduced general scalar sex-limitation AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may also fit the data well, -2LL(464) = 2217.91, AIC = 1289.91, Δ -2LL = .71, p = .07. The standardized variance components for WC for males were estimated based on the reduced AE model, and showed that the greatest

proportion of the variance in WC was accounted for by additive genetic factors (a^2 = .95), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .05; see Table 8 for full estimates). The standardized variance components for WC for females estimated based on the reduced AE model showed that the greatest proportion of the variance in WC was accounted for by additive genetic factors (a^2 = .88), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .12; see Table 8 for full estimates).

The full univariate general scalar sex-limitation ACE model for percent body fat at eight years was a good fit for the data, -2LL(441) = 2797.80, AIC = 1915.80 (see Tables 7 for full fit statistics). However, the reduced general scalar sex-limitation AE model did not fit significantly worse than the full ACE model, suggesting that the AE model may also fit the data well, -2LL(443) = 2799.83, AIC = 1913.83, Δ -2LL = 2.03, p = .36. The standardized variance components for percent body fat for males were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in percent body fat was accounted for by additive genetic factors (a^2 = .95), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .05; see Table 8 for full estimates). The standardized variance components for percent body fat for females estimated based on the reduced AE model showed that the greatest proportion of the variance in percent body fat was accounted for by additive genetic factors (a^2 = .90), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .10; see Table 8 for full estimates).

Given that best-fitting models indicate differences in the magnitude and nature of genetic and environmental effects between males and females for each weight indicator, DZ opposite-sex twins were excluded from all Univariate and Bivariate Cholesky Decomposition Models using weight indicators. When excluding DZ opposite-sex twins from analyses, the reduced AE model for BMI at eight years age did not fit significantly worse than the full ACE model, -2LL(322) = 1429.27, AIC = 785.27, Δ -2LL = .59, p = .44 (see Table 5 for full fit statistics). The standardized variance components for BMI were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in BMI was accounted for by additive genetic factors (a^2 = .93), with remaining variance accounted for by the non-shared environmental contribution (e^2 =

.07; see Table 6 for full estimates). Similarly, the reduced AE model for WC at eight years age did not fit significantly worse than the full ACE model, -2LL(319) = 1501.95, AIC = 863.95, ∆ -2LL = 1.13, *p* = .29 (see Table 5 for full fit statistics). The standardized variance components for WC were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in WC was accounted for by additive genetic factors (a^2 = .92), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .08; see Table 6 for full estimates). Finally, the reduced AE model for percent body fat at eight years age did not fit significantly worse than the full ACE model, -2LL(308) = 1925.67, AIC = 1309.67, ∆ -2LL = .01, *p* = .93 (see Table 5 for full fit statistics). The standardized variance components for percent body fat were estimated based on the reduced AE model, and showed that the greatest proportion of the variance in percent body fat was accounted for by additive genetic factors (a^2 = .92), with remaining variance accounted for by the non-shared environmental contribution (e^2 = .08; see Table 6 for full estimates).

Objective Sleep Indicators Bivariate Models. A Bivariate Cholesky Decomposition of objective sleep duration and efficiency at eight years revealed the AE-ACE model to be the best fitting model, after also dropping the C contribution to the covariance between the two phenotypes (see Table 9 for fit statistics and Table 10 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .80), with the remaining variance accounted for by the nonshared environmental contributions (E11 = .20). The covariance between objective sleep duration and efficiency was accounted for primarily by additive genetic factors, explaining 37% of the total variance in sleep efficiency, with the nonshared environment also explaining 14% of the total variance in sleep efficiency. After accounting for objective sleep duration, the variance in sleep efficiency was accounted for by additive genetics (A22 = .14), the shared environment (C22 = .26), and the nonshared environment (E22 = .08). Genetic and environmental correlations showed that genetic influences on objective sleep duration were highly correlated with genetic environmental influences on sleep efficiency at .85.

The bivariate model for objective sleep duration and midpoint variability at eight years revealed the full ACE-ACE model to be the best fitting model, after also dropping the A contribution to the covariance between the two phenotypes (see Table 9 for fit statistics and Table 10 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .72), with the remaining variance divided between the shared (C11 = .09) and nonshared environmental contributions (E11 = .19). The covariance between objective sleep duration and midpoint variability was accounted for primarily by the shared environment, explaining 26% of the total variance in parent-reported sleep duration, but the nonshared environment also explained 1% of the total variance in midpoint variability. After accounting for objective sleep duration, the variance in midpoint variability was accounted for by shared environmental (C22 = .44) and additive genetic factors (A22 = .11), with little contribution of the nonshared environment (E22 = .19). Genetic and environmental correlations showed that shared environmental influences on objective sleep duration were correlated with shared environmental influences on midpoint variability at .61, and nonshared environmental influences on objective sleep duration were correlated with nonshared environmental influences on midpoint variability at .08.

The bivariate model for objective sleep duration and parent-reported sleep duration at eight years revealed the full ACE-ACE model to be the best fitting model, after also dropping the A contribution to the covariance between the two phenotypes (see Table 9 for fit statistics and Table 10 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .70), with the remaining variance divided between the shared (C11 = .10) and nonshared environmental contributions (E11 = .20). The covariance between objective and parent-reported sleep duration was accounted for primarily by the shared environment, explaining 65% of the total variance in parent-reported sleep duration, and the nonshared environment also explained 1% of the total variance in parent-reported sleep duration. After accounting for objective sleep duration, the variance in parent-reported sleep duration was accounted for by additive genetic factors (A22 = .21) and little remaining contribution of the shared (C22 = .01) and nonshared environment (E22 = .12). Genetic and environmental

correlations showed that shared environmental influences on objective sleep duration were correlated with shared environmental influences on parent-reported sleep duration at .59, and nonshared environmental influences on objective sleep duration were correlated with nonshared environmental influences on parent-reported sleep duration at .15.

The bivariate model for parent-reported sleep duration and objective sleep midpoint variability at eight years revealed the full ACE-ACE model to be the best fitting model, after also dropping the A and E contributions to the covariance between the two phenotypes (see Table 9 for fit statistics and Table 10 for standardized variance components). Parent-reported sleep duration was primarily influenced by shared environmental factors (C11 = .62), with the remaining variance divided between additive genetic (A11 = .21) and nonshared environmental contributions (E11 = .17). The covariance between parent-reported sleep duration and objective midpoint variability was entirely accounted for by the shared environment, explaining 5% of the total variance in sleep midpoint variability. After accounting for parent-reported sleep duration, the variance in sleep midpoint variability was accounted for by shared environmental (C22 = .57), additive genetic, (A22 = .18), and nonshared environmental factors (E22 = .20). Genetic and environmental correlations showed that shared environmental influences on parent-reported sleep duration were correlated with shared environmental influences on sleep midpoint variability at .30.

Objective Sleep Duration and Weight Indicator Bivariate Models. A Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep duration and BMI at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 12 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .58), with the remaining variance accounted for by shared (C11 = .22) and nonshared environmental contributions (E11 = .20). The covariance between objective sleep duration and BMI was accounted entirely by additive genetic factors, explaining 6% of the total variance in BMI. After accounting for objective sleep duration, the variance in BMI was accounted for by additive genetics (A22 = .86) and the nonshared

environment (E22 = .08). Genetic and environmental correlations showed that genetic influences on objective sleep duration were correlated with genetic environmental influences on BMI at .26.

Similarly, the Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep duration and WC at eight years revealed the AE-AE model to be the best fitting model, after also dropping the E contribution to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 12 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .80), with the remaining variance accounted for by the nonshared environmental contributions (E11 = .20). The covariance between objective sleep duration and WC was accounted entirely by additive genetic factors, explaining 3% of the total variance in WC. After accounting for objective sleep duration, the variance in WC was accounted for by additive genetics (A22 = .89) and the nonshared environment (E22 = .08). Genetic and environmental correlations showed that genetic influences on objective sleep duration were correlated with genetic environmental influences on WC at .16.

Similarly, the Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep duration and percent body fat at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 12 for standardized variance components). Objective sleep duration was primarily influenced by additive genetic factors (A11 = .58), with the remaining variance accounted for by shared (C11 = .22) and nonshared environmental contributions (E11 = .20). The covariance between objective sleep duration and percent body fat was accounted entirely by additive genetic factors, explaining 10% of the total variance in percent body fat. After accounting for objective sleep duration, the variance in percent body fat was accounted for by additive genetics (A22 = .83) and the nonshared environment (E22 = .07). Genetic and environmental correlations showed that genetic influences on objective sleep duration were correlated with genetic environmental influences on percent body fat at .32.

Objective Sleep Efficiency and Weight Indicator Bivariate Models. A Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep efficiency and BMI at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C

and E contributions to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 13 for standardized variance components). Objective sleep efficiency was primarily influenced by shared environmental factors (C11 = .47), with the remaining variance accounted for by additive genetic factors (A11 = .32) and nonshared environmental contributions (E11 = .21). The covariance between objective sleep efficiency and BMI was accounted entirely by additive genetic factors, explaining 14% of the total variance in BMI. After accounting for objective sleep efficiency, the variance in BMI was accounted for by additive genetics (A22 = .79) and the nonshared environment (E22 = .07). Genetic and environmental correlations showed that genetic influences on objective sleep efficiency were correlated with genetic environmental influences on BMI at .39.

Similarly, the Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep efficiency and WC at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 13 for standardized variance components). Objective sleep efficiency was primarily influenced by shared environmental factors (C11 = .43), with the remaining variance accounted for by additive genetic factors (A11 = .36) and nonshared environmental factors (E11 = .21). The covariance between objective sleep efficiency and WC was accounted entirely by additive genetic factors, explaining 9% of the total variance in WC. After accounting for objective sleep efficiency, the variance in WC was accounted for by additive genetics (A22 = .83) and the nonshared environment (E22 = .08). Genetic and environmental correlations showed that genetic influences on objective sleep efficiency were correlated with genetic environmental influences on WC at .32.

Similarly, the Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective sleep efficiency and percent body fat at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 11 for fit statistics and Table 13 for standardized variance components). Objective sleep efficiency was primarily influenced by shared environmental factors (C11 = .46), with the remaining variance accounted for by additive genetic factors (A11 = .33) and

nonshared environmental contributions (E11 = .21). The covariance between objective sleep efficiency and percent body fat was accounted entirely by additive genetic factors, explaining 17% of the total variance in percent body fat. After accounting for objective sleep efficiency, the variance in percent body fat was accounted for by additive genetics (A22 = .76) and the nonshared environment (E22 = .07). Genetic and environmental correlations showed that genetic influences on objective sleep efficiency were correlated with genetic environmental influences on percent body fat at .43.

EC and Sleep Indicator Bivariate Models. A Bivariate Cholesky Decomposition of EC and objective sleep duration at eight years revealed the AE-ACE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 14 for fit statistics and Table 15 for standardized variance components). EC was primarily influenced by additive genetic factors (A11 = .75), with the remaining variance accounted for by the nonshared environment (E11 = .25). The covariance between EC and objective sleep duration was accounted entirely by additive genetic factors, explaining 3% of the total variance in sleep duration. After accounting for EC, the variance in objective sleep duration was accounted for by additive genetics (A22 = .67), shared environment (C22 = .10), and the nonshared environment (E22 = .20). Genetic and environmental correlations showed that additive genetic influences on EC were correlated with additive genetic environmental influences on objective sleep duration at .20.

A Bivariate Cholesky Decomposition of EC and objective sleep efficiency at eight years revealed the AE-ACE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 14 for fit statistics and Table 15 for standardized variance components). EC was primarily influenced by additive genetic factors (A11 = .75), with the remaining variance accounted for by the nonshared environment (E11 = .25). The covariance between EC and objective sleep efficiency was accounted for primarily by additive genetic factors, explaining 1% of the total variance in sleep efficiency. After accounting for EC, the variance in objective sleep efficiency was accounted for by additive genetics (A22 = .57), shared environment (C22 = .20), and the nonshared environment (E22 =

.22). Genetic and environmental correlations showed that genetic influences on EC were correlated with genetic environmental influences on objective sleep efficiency at .15.

A Bivariate Cholesky Decomposition of EC and parent-reported sleep duration at eight years revealed the ACE-ACE model to be the best fitting model, after also dropping the A contribution to the covariance between the two phenotypes (see Table 14 for fit statistics and Table 15 for standardized variance components). EC was primarily influenced by shared environmental factors (C11 = .16), with the remaining variance accounted for by additive genetic factors (A11 = .36) and the nonshared environment (E11 = .48). The covariance between EC and parent-reported sleep duration was accounted for by the shared environment, explaining 5% of the total variance in parent-reported sleep duration, as well as the nonshared environment, which explained 1% of the total variance in parent-reported sleep duration. After accounting for EC, the variance in parent-reported sleep duration was accounted for by additive genetics (A22 = .22), shared environment (C22 = .63), and the nonshared environment (E22 = .10). Genetic and environmental correlations showed that shared environmental influences on EC were correlated with shared environmental influences on parent-reported sleep duration at .28, and nonshared environmental influences on EC were correlated with nonshared environmental influences on parent-reported sleep duration at .12.

EC and Weight Indicator Bivariate Models. A Bivariate Cholesky Decomposition of EC and objective BMI at eight years revealed the AE-AE model to be the best fitting model, after also dropping the C and E contributions to the covariance between the two phenotypes (see Table 14 for fit statistics and Table 15 for standardized variance components). EC was primarily influenced by additive genetic factors (A11 = .73), with the remaining variance accounted for by the shared (C11 = .01) and nonshared environment (E11 = .26). The covariance between EC and BMI was accounted for entirely by additive genetic factors, explaining 2% of the total variance in BMI. After accounting for EC, the variance in BMI was accounted for by additive genetics (A22 = .91) and the nonshared environment (E22 = .07). Genetic and environmental correlations showed that genetic influences on EC were correlated with genetic environmental influences on BMI at .12.

Weight Indicator Bivariate Models. A Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective BMI and percent body fat at eight years revealed the AE-AE model to be the best fitting model, after also dropping the C contribution to the covariance between the two phenotypes (see Table 16 for fit statistics and Table 17 for standardized variance components). BMI was primarily influenced by additive genetic factors (A11 = .93), with the remaining variance accounted for by the nonshared environment (E11 = .07). The covariance between BMI and percent body fat was accounted for primarily by additive genetic factors, explaining 80% of the total variance in percent body fat, with the remaining covariance accounted for by nonshared environmental factors, which explained 5% of the total variance in percent body fat. After accounting for BMI, the variance in percent body fat was accounted for by additive genetics (A22 = .13) and the nonshared environment (E22 = .02). Genetic and environmental correlations showed that genetic influences on BMI were correlated with genetic environmental influences on percent body fat at .87.

A Bivariate Cholesky Decomposition (excluding DZ opposite-sex twins) of objective BMI and WC at eight years revealed the AE-AE model to be the best fitting model, after also dropping the C contribution to the covariance between the two phenotypes (see Table 16 for fit statistics and Table 17 for standardized variance components). BMI was primarily influenced by additive genetic factors (A11 = .92), with the remaining variance accounted for by the nonshared environment (E11 = .08). The covariance between BMI and WC was accounted for primarily by additive genetic factors, explaining 70% of the total variance in WC, with the remaining covariance accounted for by nonshared environmental factors, which explained 5% of the total variance in WC. After accounting for BMI, the variance in WC was accounted for by additive genetics (A22 = .21) and the nonshared environment (E22 = .04). Genetic and environmental correlations showed that genetic influences on BMI were correlated with genetic environmental influences on WC at .93.

A Bivariate Cholesky Decomposition of objective WC and percent body fat at eight years revealed the ACE-AE model to be the best fitting model, after also dropping the C contribution to the covariance between the two phenotypes (see Table 16 for fit statistics and Table 17 for

standardized variance components). WC was primarily influenced by additive genetic factors (A11 = .76), with the remaining variance accounted for by the shared (C11 = .16) and nonshared environments (E11 = .08). The covariance between WC and percent body fat was accounted for primarily by additive genetic factors, explaining 79% of the total variance in percent body fat, with the remaining covariance accounted for by nonshared environmental factors, which explained 3% of the total variance in percent body fat. After accounting for WC, the variance in percent body fat was accounted for by additive genetics (A22 = .14) and the nonshared environment (E22 = .04). Genetic and environmental correlations showed that genetic influences on WC were correlated with genetic influences on percent body fat at .93, and nonshared environmental influences on WC were correlated with nonshared environmental influences on percent body fat at .60.

Aim 2b Results

As specified, if there are no phenotypic associations or weak correlations between sleep, weight indicators, and EC conducted in Aim 1a, it may not be appropriate to conduct multivariate Cholesky decomposition models. Given that there were weak zero-order correlations and no significant phenotypic associations (from Aim 1a) among sleep, weight indicators and EC, Multivariate Cholesky Decompositions may be unstable and were not appropriate to fit within the current dissertation.

Aim 2c Results

Finally, one Liability Threshold Model was fit to test the extent to which additive genetic and environmental influences on a weight status may differ across groups of individuals (Plomin et al., 2013). The Liability Threshold Model was conducted without DZ opposite-sex twins given significant differences between males and females in univariate sex-limitation models for all weight indicators. The best fitting model was one in which the thresholds were equated across twin 1 and twin 2 as well as across zygosity groups, and all of the variances were constrained to be 1. The full and best fitting model showed that overweight/obese status was primarily influenced by additive genetics (A11 = .46), but there were also significant contributions from the shared (C11 = .42) and nonshared environment (E11 = .12).

CHAPTER 4

DISCUSSION

The current dissertation supports a growing body of literature demonstrating associations between various objective and subjective sleep and weight indicators in middle childhood and addresses the need for understanding longitudinal associations and change over time in the links between sleep and weight indicators (Miller, Lumeng, & LeBourgeois, 2015). This dissertation also builds on the current literature by exploring how key person-level variables like EC may modulate associations between sleep and weight indicators, and the extent to which both genetic and environmental influences on associations among sleep, EC, and weight indicators in middle childhood. The findings are in concordance with aspects of developmental systems theories which hold that changes within and between individuals occur at multiple levels of organization over time (Damon & Lerner, 2008). These tenets are also entrenched in more specific theories regarding the development of sleep and weight problems across the lifespan (Becker et al., 2015; Fishbein, 2000; Greeno & Wing, 1994), as well as research that shows fluctuations in genetic and environmental influences on traits over time (Barclay et al., 2014; Plomin et al., 2013). In the current dissertation, I tested whether normative sleep problems and EC at eight years concurrently and prospectively predict weight indicators and status at nine years and estimated unique and shared covariance among non-clinical sleep problems, weight indicators, and effortful control at eight years of age. The current findings suggest that while there are no cross-sectional interactions between sleep and EC when predicting weight indicators at eight years of age, interactions between unique sleep and EC at eight years of age predict greater changes in various weight indicators from eight to nine years of age. Furthermore, the findings suggest differential genetic and environmental influences on objective and subjective sleep indicators, as well as considerable additive genetic contribution to each weight indicator that differed by sex. The current findings also indicate that most of the associations between sleep and weight indicators can be explained by additive genetic contributions and links between weight indicators are entirely explained by additive genetic influences. However, covariance between various aspects of sleep was dependent on whether sleep was parent-report or objectively collected.

Aim 1a Findings and Interpretation

Prior studies have shown that shorter parent-reported and actigraphy-based sleep duration are associated with greater concurrent risk for obesity, higher BMI scores, greater WC, and higher percent body fat after accounting for multiple demographic and lifestyle factors, such as parent obesity, parent education, sex, age, screen time, physical activity level, eating behaviors, and birth height and weight (Chaput et al., 2006; Ekstedt et al., 2013; Martinez et al., 2014; Nixon et al., 2008; von Kries et al., 2002). Furthermore, poor objective and subjective sleep quality have been concurrently linked with greater risk for being overweight or obese and higher zBMI (Bagley & El-Sheikh, 2013; Fatima et al., 2016), and greater objective sleep duration variability has been associated with obesity (Spryut et al., 2011). The current dissertation extends these findings in a number of ways. Regarding interactions between objective and subjective and EC predicting concurrent weight indicators and status, my hypotheses were not supported; there were no significant interactions between any objective and subjective sleep parameters and EC when predicting concurrent weight indicators or status. These null results suggest that cross-sectional associations between objective and subjective sleep and weight indicators and status do not vary based on child level of EC, and that children with low EC do not show stronger links between sleep and weight indicators at eight years of age.

Rather, the current findings suggest that there are associations between particular sleep and weight indicators across all children, regardless of their level of EC. Specifically, I found significant main effects of objective sleep duration and efficiency on concurrent BMI, WC, percent body fat, and risk for being classified as overweight or obese, which supports my hypotheses and numerous previous studies. Additionally, recent findings with a subsample of the population used in the current dissertation show that longer sleep duration at eight years was associated with lower BMI for all children, regardless of early-life SES level (Breitenstein et al., in press). These findings also fit within biological and endocrine models of weight gain and increased adiposity that suggest various stressors, including poor or restricted sleep, may prompt changes in hormones levels and glucose uptake and metabolism which may lead to increased body fat and weight gain (Miller & Cappuccio, 2007; Spiegel et al., 2004). These findings indicate that children who

experience shortened sleep duration and poorer sleep quality based on actigraphy may also demonstrate higher BMI, larger WC, greater percent body fat, and increase risk for being classified as overweight or obese (Breitenstein et al., in press), and changes in hormones and metabolism may be one possible mechanism that accounts for these links.

However, I did not find significant main effects of parent-reported sleep duration, daytime sleepiness or sleep midpoint variability on any of the weight indicators at eight years. This was counter to my hypotheses and some prior literature showing relations between parent- or self-reported sleep quantity and quality and various weight indicators and risk for obesity (Chaput et al., 2006; Fatima et al., 2016; Martinez et al., 2014; von Kries et al., 2002). Yet, these results align with some prior literature showing that there are differential associations between objective and subjective sleep and various outcomes (e.g., Shochat, Cohen-Zion, & Tzischinsky, 2014; Tremaine et al., 2010), as I found significant main effects of objective sleep duration and efficiency but not sleep midpoint variability or parent-reported sleep duration and quality. In the current dissertation, I observed an almost 1.5-hour difference in average parent-reported sleep duration and objective sleep duration, such that parents reported longer sleep duration than was detected by actigraphy. This finding replicates other studies comparing parent-reported sleep duration to actigraphy-based sleep duration that show parents tend to report significantly longer sleep duration compared to actigraphy estimates of sleep duration by as much as one hour (Martinez et al., 2014; Nixon et al., 2008).

It is notable that child nighttime sleep duration is determined using a single item, which asked primary caregivers to report how many hours and minutes each child slept at night on average. It is likely that primary caregivers based their estimate of child sleep duration on the time each child gets into bed each night and gets out of bed each morning, but no clear guidelines or clarification was provided for primary caregivers when reporting average nighttime sleep duration. In comparison, actigraphy-based sleep duration estimates “true” sleep time, with bedtime marked when children are in bed and trying to go to sleep (i.e., no more active movement, lights are out) and waketime marked when children get out of bed, demonstrate moderate activity and lights are turned on (as recorded by the actigraphy watch). Actigraphy-based sleep estimates also exclude

waking periods throughout the night. As such, differential cross-sectional associations between objective and subjective sleep duration and weight indicators and status in middle childhood may be a result of parent overreporting of child nighttime sleep duration, but it is also possible that lack of main effects for parent-reported sleep duration is a product of how subjective sleep duration was assessed and conceptualized in the current dissertation.

My hypothesis that greater sleep midpoint variability at eight years would be associated with higher scores on all weight indicators at eight years was also unsupported. This was also surprising, as prior studies show that earlier bedtimes and waketimes are associated with lower BMI scores and reduced risk for being classified as obese in middle childhood (Anderson, Sacker, Whitaker, & Kelly, 2017; Ekstedt et al., 2013). However, sleep midpoint variability accounts for variability in both bedtime and waketime from night to night across a week. It is possible that children have greater variability in bedtimes across a typical week or particularly on weekend days, but children experience much less variability in waketimes across a week, as waketimes in this developmental period are often restricted by early school start times and caregiver work schedules (Crowley et al., 2014). As such, estimates of midpoint variability in middle childhood are likely much lower and constrained by consistent waketimes compared to midpoint variability estimates that may be observed in infant, emerging adult or adult samples, and this may explain lack of concurrent significant findings.

Similar to significant main effects for objective sleep indicators, greater EC at eight years was associated with lower BMI and marginally lower WC at eight years in multiple models. These findings suggest that children who demonstrate higher regulation of their thoughts, behaviors, emotions, and cognitions at eight years of age may be able to better able to regulate or modulate thoughts and behaviors related to weight gain and increased body fatness, resulting lower BMI and slightly less visceral body fat at eight years. Indeed, theory regarding sleep and emotion regulation suggests bidirectional links between the constructs such that sleep dysregulation and restriction lead to deficits in various domains of regulation, and poor regulation more broadly may predict subsequent sleep problems (Dahl, 1996). This theoretical framework may extend to links between regulation and weight indicators as well, given that some prior empirical studies that

lower eating self-regulation was associated with higher concurrent zBMI scores in preschoolers (Hughes et al., 2015). In the same study, however, broad self-regulation (measured with executive functioning, inhibitory control, and emotion regulation) was not related to zBMI scores (Hughes et al., 2015), suggesting that there may not be relations between EC and some aspects of weight; rather, it may depend which facet of self-regulation is being examined in relation to weight indicators. On the other hand, prior studies show longitudinal associations between EC and weight indicators (similar to findings from the current dissertation), such that greater broad and emotional self-regulation at two years predicted higher BMI scores and greater risk for obesity at 5.5 years (Graziano et al., 2010). A related study also found that lower self-regulation or difficulties at two years predicted higher BMI scores and obesity at 10 years, as well as more eating and body image concerns at 10 years (Graziano et al., 2013). Thus, I may have found significant main effects of self-regulation on weight indicators if I had examined other self-regulation or EC scales (e.g., impulsivity, inhibitory control, activation control, attentional focusing) in relation to weight indicators in middle childhood. Furthermore, findings from prior empirical studies (and the current dissertation) suggest that relations between EC and weight may not be present in cross-sectional analyses (e.g., Hughes et al., 2015), but that EC earlier in development predicts scores on weight indicators and status later in development (e.g., Graziano et al., 2010; Graziano et al., 2013).

Finally, the current findings generally show that a number of demographic and lifestyle factors are also associated with childhood weight indicators. Specifically, children further along in pubertal development demonstrated greater BMI, larger WC, greater percent body fat and increased risk of being classified as overweight or obese is supported by some empirical work on pubertal timing and weight gain (Daniels, 2006; Davison et al., 2003). European American/White participants also demonstrated lower percent body fat similar to past research showing that Latino and African American children have greater adiposity and poorer sleep compared to their European American/White counterparts (Biggs et al., 2013; Wisniewski & Chernausek, 2009). Females showed higher percent body fat in multiple models, which supports a host of literature showing that females show greater adiposity both before and after the onset of puberty (Daniels,

2006; Davison et al., 2003; Wisniewski & Chernausek, 2009). Completing the study week during a school break (rather than during the school year) was linked with about a 70% increase in odds of being classified as overweight or obese. Finally, greater SES was associated with lower BMI and percent body fat, which mirror findings showing that children from low SES backgrounds experience shorter sleep duration (Biggs et al., 2013; Breitenstein et al., in press; O'Dea et al., 2014). Interactive effects of SES with objective and subjective sleep on weight indicators have also been established with a subsample of the population used in the current dissertation, showing that associations between sleep and weight indicators differed based on children's early level of SES (Breitenstein et al., in press). Specifically, greater sleep duration predicted lower percent body fat for children with low early SES, greater sleep efficiency predicted lower BMI, smaller WC and lower percent body fat for children with low and average early SES, and greater parent-reported sleep problems predicted larger WC specifically for children with low early SES (Breitenstein et al., in press). Importantly, greater sleep duration and efficiency predicted the lowest odds of being classified as overweight or obese particularly for children from low early SES backgrounds (Breitenstein et al., in press). These findings also highlight the importance of various demographic and lifestyle factors on health behaviors across development and demonstrate that it may be critical to test some of these demographic and lifestyle factors as moderating and mediating factors when examining links between sleep and weight indicators.

Overall, there were no significant interactions between objective and subjective sleep and EC when predicting concurrent weight indicators and status, suggesting no differences in associations between sleep and weight indicators based on child EC levels. Alternatively, I may not have detected significant interactions between sleep and EC on weight indicators and status because there is significant contributions from or variability that was accounted for by other contextual factors (or demographic/lifestyle factors; Biggs et al., 2013; Breitenstein et al., in press; Daniel, 2006; Davison et al., 2003; Wisniewski & Chernausek, 2009). However, there were numerous main effects for objective sleep quantity and quality, EC, and demographic and lifestyle factors that support prior literature and indicate that better sleep and self-regulation as associated

with lower BMI, WC, percent body fat, and risk for being classified as overweight or obese (Breitenstein et al., in press; Hughes et al., 2015; Martinez et al., 2014; Nixon et al., 2008).

Aim 1b Findings and Interpretation

Longitudinal research suggests children who obtain longer parent-reported and objective sleep at night on average, have earlier bedtimes, and later wake times showed lower BMI scores, percent body fat, and lower risk of being obese (Bagley & El-Sheikh, 2015; Carter et al., 2011; Snell et al., 2007). Additionally, children who demonstrated short parent-reported sleep in early childhood showed higher BMI scores and greater risk of being overweight or obese children in middle childhood (Touchette et al., 2008). Some of the longitudinal phenotypic findings from the current dissertation support these previous studies, as a number of my hypotheses regarding interactions between objective sleep duration, midpoint variability and EC at eight years predicting weight indicators at nine years were supported. Specifically, greater objective sleep duration at eight years predicted greater decreases in BMI from eight to nine years, particularly for children who showed low EC at eight years. This finding indicates that when children obtain greater sleep quantity, children with lower EC showed the greatest decreases in BMI from eight to nine years of age. Furthermore, this suggests that children with low EC may experience the greatest benefit from obtaining longer sleep duration, and that obtaining more hours of sleep per night on average may lead to greater decreases in BMI across middle childhood. Importantly, relations between objective sleep duration at eight years and changes in BMI from eight to nine years were not significant for children with average or high EC levels, suggesting that higher levels of EC may protect against the negative effects of short sleep and children with high EC may better regulate themselves even when obtaining fewer hours of sleep at night.

I also found that greater sleep midpoint variability at eight years predicted greater increases in percent body fat at nine years, particularly for children with low EC, which supported my hypothesis. This finding suggests that when children show greater variability in bedtimes and waketimes on average, children with lower EC showed the greatest increases in percent body fat from eight to nine years of age. Furthermore, children with low EC may experience the greatest risk from varying bedtimes and waketimes, and that having greater fluctuation in bedtimes and

waketimes on average may lead to greater increases in percent body fat across middle childhood. As noted earlier, children waketimes are more restricted by early school start times and caregiver work schedules (Crowley et al., 2014), so it is possible that high variability in bedtimes from night to night on average may be driving effects on percent body fat. Furthermore, these findings suggest that broad, underlying dysregulation may account for links between sleep midpoint variability, percent body fat and low EC, such that children with low EC may have more difficulty regulating their sleep (including falling asleep and staying asleep), their eating behaviors, and their thoughts, emotions, and behaviors. Indeed, one study showed that poor self-regulation in middle childhood was linked to greater eating and greater percent body fat concurrently (Faith et al., 2012). Another study showed that children with average to high levels of self-regulation in middle childhood also exhibited fewer parent-reported sleep problems over time, whereas children with lower self-regulatory skills experienced increases in sleep problems across early and middle childhood (Williams et al., 2016). Thus, higher levels of EC may protect against the negative effects of short sleep and more variability in sleep schedules and increases in BMI and percent body fat.

Collectively, these interactive effects fit with some literature showing that children who demonstrate greater sleep duration variability and shorter sleep duration are more likely to consume sugar, sugary drinks, energy-dense foods, and fewer vegetables (Franckle et al., 2015; Kjeldsen et al., 2014), indicating that when children experiencing greater variability in sleep schedules more broadly, these children also tend to eat more calorie-dense and sugary foods that are likely to increase body fatness. Specifically, it is possible that shorter sleep and greater variability in bedtimes and waketimes (and a less regular sleep schedule more generally) at eight years of age acts as a stressor for children, which leads to increases in ghrelin (hormone signaling hunger) and reduction in leptin (hormone signaling satiety), glucose uptake, and metabolism (Miller & Cappuccio, 2007; Spiegel et al., 2004). These changes in hormones that signal hunger and satiety may lead children to eat more food, specifically high calorie or sugar foods, which may lead to increased body fat over time (Miller & Cappuccio, 2007; Spiegel et al., 2004). It is also likely that children with restricted sleep and greater variability in bedtime and

waketime on average are awake more hours of the day compared to children with more rigid sleep schedules, and that being awake more hours of the day provides some children additional opportunities to eat many types of food including foods that may increase percent body fat.

Another possible explanation for associations between longer sleep duration and lower BMI and greater sleep midpoint variability and increased percent body fat is increased autonomy and decision-making regarding sleep schedules, exercise, eating, and other lifestyle factors during middle childhood. Regarding sleep duration and timing, research shows that autonomy at bedtime and more broadly may be associated with higher sleep quality, lower sleep duration variability, and lower odds of having a late bedtime (e.g., Doane et al., 2019; Erath & Tu, 2011; Spilsbury et al., 2005). Thus, while parents may still primarily dictate sleep schedules, food choice, activity and other day to day activities during middle childhood, children are increasingly expected to take on more responsibility and are allowed to make some decisions for themselves during this developmental period and this responsibility may impact their sleep quantity and timing. As such, greater autonomy and decision-making may account for associations among longer sleep duration, lower BMI, and low EC, as well as links between high sleep midpoint variability, increased percent body fat and low EC.

Finally, it is also possible that increases in midpoint variability and percent body fat for children with low EC detected in the study stem from other stressors not assessed in the current dissertation (see Figure 1). For example, variability in bedtime and waketime, as well as increases in adiposity, may be driven by aspects of parenting and the home environment such as parenting styles, family schedules (daily and related to mealtime), food choices, whether children share rooms or beds with other family members, and level of engagement in physical activity. Indeed, studies have shown that children with fathers whose parenting styles were characterized as either permissive or disengaged had greater odds of being in a higher weight status (e.g., overweight or obese; Wake, Nicholson, Hardy, & Smith, 2007). Regarding sleep, one study found that for every one-hour decrease in parent-reported sleep duration, there was a 40% increase in risk or odds or being obese and that greater parenting stress was linked with shorter subjective sleep duration, but not with increased risk of being obese (Ievers-Landis, Storfer-Isser, Rosen,

Johnson, & Redline, 2008). Other studies have shown that families who were more engaged with one another during mealtimes, who had more positive communication during mealtimes, and who placed greater value on mealtimes tended to have children who were considered healthy or normal weight, compared to families with children who were classified as having overweight or obese status (per zBMI scores; Fiese, Hammons, & Grigsby-Toussaint, 2012). Similarly, Anderson (2012) showed that eating meals at consistent times, eating meals as a family and having household rules regarding television watching were all related to lower probability of children being obese. Thus, other psychosocial factors may explain longitudinal links between midpoint variability and percent body fat.

Despite a number of significant interactions between unique sleep indicators and EC at eight years when predicting BMI and percent body fat at nine years, there were no significant interactions between sleep and EC at when predicting risk for being overweight or obese cross-sectionally or longitudinally. This finding did not support my hypothesis and was surprising, as many numerous meta-analyses have shown cross-sectional association between short sleep duration and increased risk for obesity (Chen et al., 2008; Marshall, 2008; Patel & Hu, 2008). The null longitudinal finding regarding relations between sleep indicators and risk for being classified as overweight or obese adds to the current literature and suggests that poor sleep may not incur increased risk for obesity over short time spans such as one year. However, if longitudinal associations between sleep and risk for obesity were measured over longer periods of time or across developmental periods, it is possible that poor sleep may predict increased risk for obesity. Further, given that the measure of overweight/obesity in analyses accounts for percentiles and scores on BMI, WC, and percent body fat, we would expect this variable to be the most robust assessment of weight and body fatness in the current dissertation. However, recent findings with a subsample of the population used in the current dissertation demonstrated greater sleep duration and efficiency predict the lowest odds of being classified as overweight or obese particularly for children from low early SES backgrounds (Breitenstein et al., in press). These significant findings occurred in addition to individual links between objective and subjective sleep and weight indicators (Breitenstein et al., in press). Differences in these findings and results from

the current dissertation may be a function of sample size or the fact that overweight/obesity status was calculated accounting for BMI, WC, and percent body fat in the current dissertation, rather than just BMI centile cutoffs like is used in most prior studies (e.g., Breitenstein et al., in press). Thus, the lack of significant interactions and main effects on risk for being classified as overweight or obese suggest that we must be cautious when interpreting and making broad conclusions about the phenotypic findings in the current dissertation, as they may represent relations that are significant only for small groups of children or individuals or may depend on sample size and how overweight/obesity status is computed.

Finally, similar to cross-sectional findings, there were a number of consistent main effects of various demographic and lifestyle factors on weight indicators at nine years of age. First, greater BMI, WC, percent body fat, and likelihood of being classified as overweight or obese at eight years each predicted BMI, WC, percent body fat, and likelihood of being classified as overweight or obese at nine years of age, respectively. These significant main effects indicate high stability in weight indicators and status over time and are expected. Greater EC at eight years predicted smaller WC at nine years, suggesting that greater self-regulation may promote better health and lower visceral body fat. Being further along in pubertal development at eight years predicted greater WC at nine years, which corresponds with some literature showing links between pubertal timing and increased adiposity (Davison et al., 2003). Children who completed the study week during a school break at eight years of age had lower BMI and reduced odds of being overweight or obese at nine years.

Overall, longer objective sleep duration at eight years predicted greater decreases in BMI from eight to nine years, and greater sleep midpoint variability at eight years predicted increases in percent body fat from eight to nine years of age. Importantly, both of these associations were only significant for children who show low EC at eight years, suggesting that when children experience short sleep duration and greater variability in bedtimes and waketimes, children with low EC may also show the greatest increases in BMI and percent body fat from eight to nine years of age. On the other hand, these findings indicate that children with low EC may experience the greatest benefits from attaining longer sleep duration and more stable bed and waketimes

from day to day. However, it is still unclear which aspects of sleep and health to target for intervention, as well as who may benefit most from these interventions, given that findings show no significant association between specific facets of sleep and risk for being classified as overweight or obese.

Aim 2a Findings and Interpretation

Prior research indicates moderate to high heritability for various sleep parameters (Breitenstein et al., 2018; Gregory et al., 2006; Moore et al., 2009), weight indicators (Maes, Neale, & Eaves, 1997; Fernandez et al., 2012; Wardle et al., 2008), and EC (Lemery-Chalfant et al., 2008; Lemery-Chalfant et al., 2013; Mullineaux et al., 2009) in twin samples of children in middle childhood. These strong additive genetic influences on sleep, weight indicators and EC reported in other twin samples, as well as prior evidence of phenotypic links between sleep, weight indicators and self-regulation (e.g., Graziano et al., 2010; Graziano, et al., 2013; Hughes et al., 2015; Williams & Sciberras, 2016), indicates that there may be shared underlying additive genetic influences on various aspects of sleep, weight indicators, and EC in middle childhood. Numerous quantitative behavior genetic findings from the current dissertation support and contribute new information to the literature regarding genetic and environmental influences on various sleep and weight indicators, as well as their associations.

First, I found that the greatest proportion of the variance in parent-reported sleep duration and daytime sleepiness at eight years of age were accounted for by shared environmental factors, with the remaining variance accounted for by additive genetic and nonshared environmental factors. Specifically, I found that only about 20% of the reason why individuals differ from one another on parent-reported sleep duration during middle childhood can be explained by additive genetics. Similarly, about 27% of the reason why individuals differ from one another on daytime sleepiness during middle childhood can be explained by additive genetics. While my hypothesis regarding daytime sleepiness was supported, my hypothesis that most of the variance in parent-reported sleep duration would be explained by additive genetic factors was not supported. However, some prior studies have reported slightly lower additive genetic influences on parent-reported sleep duration similar to what was detected in the current

dissertation (see Brescianini et al., 2011; Gregory et al., 2009; 30-46%). Greater shared environmental influence on parent-reported sleep duration and daytime sleepiness may capture high similarity in parent reports of sleep duration and sleep quality for each child (parent-report bias) but may also represent numerous factors in twins' home or sleep environment that make them more alike on sleep duration. For example, twins in the same family may have similar daily and sleep schedules and may share a room or even a bed, all of which may make their sleep duration and level of daytime sleepiness more similar to their co-twin.

In contrast with parent-reported sleep duration and quality, the greatest proportion of the variance in actigraphy-based sleep duration and efficiency was accounted for by additive genetic factors, with the remaining variance attributed to nonshared environmental factors. Thus, findings suggest that objective sleep quantity and quality are highly heritability during middle childhood in the current sample, with the estimates for sleep duration (.81) and efficiency (.79) being slightly higher than those reported in prior studies with this age group (e.g., Gregory et al., 2006; Moore et al., 2009). These findings also suggest that I found that about 80% of the reason why individuals differ from one another on objective sleep duration and efficiency during middle childhood can be explained by additive genetics, indicating that sleep quantity and quality hold strong genetic underpinnings. Interestingly, objective sleep midpoint variability demonstrated no additive genetic influence; the greatest proportion of the variance in sleep midpoint variability was accounted for by shared environmental factors similar to parent-reported sleep duration and daytime sleepiness. As previously noted, child bedtimes and waketimes are heavily influenced and restricted by school start times, parent work schedules, and family routines more broadly in middle childhood (Anderson, 2012; Crowley et al., 2014; Fiese et al., 2012). As such, it is likely that these environmental factors contribute to high similarity in bedtime and waketime variability from night to night. However, this is the first study to my knowledge to test the heritability of sleep midpoint variability, which contributes to a growing body of literature that calls for the examination of other sleep indicators beyond sleep duration (e.g., Patel & Hu, 2008).

My hypothesis that the greatest proportion of the variance in EC would be accounted for by additive genetic factors was supported, with the higher heritability estimate for EC (.72) falling

within the same range as estimates reported in other studies on children during middle childhood (e.g., Lemery-Chalfant et al., 2008; Lemery-Chalfant et al., 2013; Mullineaux et al., 2009). Furthermore, about 72% of the reason why individuals differ from one another on parent-reported EC duration during middle childhood can be explained by additive genetics. Finally, my hypothesis that the greatest proportion of the variance in BMI, WC, and percent body fat would be accounted for by additive genetic factors was supported. Twin intra-class correlations for weight indicators were quite different between DZ same-sex twins and DZ opposite-sex twins, suggesting that there may be sex differences for weight indicators during middle childhood. When testing genetic and environmental influences on each weight indicator separately for males and females, I found that the greatest proportion of the variance in BMI, WC, and percent body was accounted for by additive genetic factors for both males and females, but that additive genetic contributions to each weight indicator were slightly higher for males compared to females. Overall, my findings suggest that over 90% of the reason why both males and females differ from one another on BMI, WC, and percent body fat during middle childhood can be explained by additive genetics, highlighting strong genetic influences and underpinnings for weight indicators that have been reported in the literature (Plomin et al., 2013). While not initially predicted, significant sex differences in the magnitude and nature of the genetic influences on BMI, WC, and percent body fat may fit with some literature showing phenotypic differences between male and females on weight indicators. Specifically, prior studies show that males tend to have reduced adiposity and fat mass across at the end of middle childhood (and as they progress through puberty), while females show greater free-fat, fat mass, likelihood of being overweight or obese, and greater percent body fat in middle childhood (Daniels, 2006; Davison et al., 2003). Further, research shows that females tend to have earlier timing in terms of pubertal development (e.g., Davison et al., 2003), which may account for difference in weight indicators. However, additive genetic estimates for all weight indicators across male and females were still slightly higher than those reported in prior studies for BMI and WC (Maes et al., 1997; Fernandez et al., 2012; Wardle et al., 2008).

Overall, I found that many key variables in the current dissertation showed high additive genetic influence, including objective sleep quantity and quality, all weight indicators, and parent-reported EC, which supports and extends the current literature. However, parent-reported sleep duration and quality, as well as objective sleep midpoint variability, demonstrated greater environmental influences suggesting that factors in the home or sleep environment (e.g., family routines and schedules, parent report bias) may explain why children show similarities to one another on these aspects of sleep rather than underlying genetic factors.

Bivariate Sleep Models. While numerous studies have documented univariate genetic and environmental contributions to sleep, weight indicators, and EC in middle childhood (e.g., Breitenstein et al., 2018; Gregory et al., 2006; Lemery-Chalfant et al., 2013; Wardle et al., 2008), far fewer studies have tested genetic and environmental influences on associations between various sleep, weight indicators and EC. Two recent studies of adults have found that shorter self-reported sleep duration was associated with higher BMI scores, with these associations accounted for entirely by common environmental effects and higher heritability of BMI scores for participants obtaining short sleep compared to participants who reported longer sleep duration (Watson et al., 2010; Watson et al., 2012). However, no studies to my knowledge have examined genetic and environmental influences on associations between sleep, weight indicators and EC in children or during middle childhood. As such, findings from the current dissertation provide considerable new information and address a number of gaps in the literature.

First, my hypothesis that additive genetics would primarily explain links between sleep duration and efficiency was supported. I found that the covariance between objective sleep duration and efficiency at eight years of age was primarily accounted for by shared additive genetic factors, with nonshared environmental factors also partially explaining links between objective sleep quantity and quality. Furthermore, additive genetic influences on sleep duration were highly correlated with additive genetic influences on sleep efficiency (r_g = .85), suggesting that some of the same genes may be influencing objective sleep duration and efficiency. Indeed, it is possible that common sets of genes may contribute to multiple aspects of sleep and drive similarity between aspects of sleep like sleep duration and quality, as other previous studies have

also shown that sleep duration and efficiency are highly correlated (phenotypically) in middle childhood (e.g., Bagley & El-Sheikh, 2013; Bagley, Kelly Buckhalt, & El-Sheikh, 2015). However, it is notable that objective sleep duration is used to compute sleep efficiency in the current dissertation, which may also contribute to high correlations and overlap between sleep quantity and quality both genetically and phenotypically.

In contrast and contrary to my hypothesis, almost all of the covariance between objective sleep duration and sleep midpoint variability and between parent-reported sleep duration and midpoint variability was accounted for by shared environmental contributions. While there were strong, unique additive genetic contributions for sleep duration and high common environmental influences on sleep midpoint variability in the bivariate model, shared environmental factors still primarily explained links between sleep duration and midpoint variability. Mirroring univariate models, parent-reported sleep duration and midpoint variability showed high unique shared environmental influence, and all of the covariance between parent-reported sleep duration and midpoint variability can be accounted for by factors in the common environment. Thus, some aspect(s) of the twins' shared environment accounts for the association between objective and parent-reported sleep duration and midpoint variability. Indeed, genetic and environmental correlations also suggest that the common environmental factors influencing sleep duration are shared with the common environmental factors influencing sleep midpoint variability (r_c = .61). However, environmental correlations between parent-reported sleep duration and midpoint variability were much lower (r_c = .30) and the covariance between (.05) between parent-reported sleep duration and midpoint variability was weak and indicates less overlap between factors in the shared environment linking the two sleep indicators. Yet, parent-imposed bedtimes and waketimes and school start times are strong, possible explanations for links between sleep duration and midpoint variability. As previously noted, family routines and schedules may influence bed and wake times, which contribute to both sleep midpoint variability and sleep duration (Anderson, 2012; Fiese et al., 2012). It is also notable that correlations indicate that longer objective and parent-reported sleep duration are associated with lower sleep midpoint variability. This inverse associations also suggests that consistency or regularity of daily

schedules (or sleep schedules) in particular may explain associations between sleep duration and midpoint variability. Finally, genetic and environmental variance and covariance between sleep indicators does not account for potential gene x environment interactions. It is possible that environmental factors (like family routines and schedules) increase or decrease additive genetic influences on (or genetic expression related to) objective and parent-reported sleep, midpoint variability and associations between these sleep indicators. Thus, despite all of the covariance between a) objective and parent-reported sleep duration and b) parent-reported sleep duration and midpoint variability being accounted for by shared environmental factors, it is possible that gene x interactions exist for these links between different aspects of sleep.

I also found that while there were strong, unique additive genetic contributions for objective sleep duration and high common environmental influences on parent-reported sleep duration, shared environmental factors primarily explained links between objective and parent-reported sleep duration at eight years of age, with some of the covariance between objective and parent-reported sleep duration explained by nonshared environmental factors. This finding suggests that some aspect(s) of the twins' shared and nonshared environments accounts for the association between objective and parent-reported sleep duration, with genetic and environmental correlations also indicating that the common environmental factors influencing objective sleep duration are shared with the common environmental factors influencing parent-reported sleep duration (r_c = .59). As expected, nonshared environmental factors contributing to objective sleep duration were weakly correlated with nonshared environmental factors influencing parent-reported sleep duration (r_e = .15). As with associations between sleep duration and midpoint variability, it is likely that links between objective and parent-reported sleep duration are driven by parenting practices and family schedules, with similarity in daily schedules driving high shared environmental covariance. Additionally, although objective and parent-reported sleep duration represent slightly different constructs (based on how they were measured) in the current dissertation and constitute different reporters, it is logical that they should be influenced by similar factors in the environment and demonstrate a positive correlation.

Bivariate Sleep and Weight Models. My hypotheses regarding genetic and environmental associations between objective sleep duration and weight indicators were all supported. All of the final, best fitting Bivariate Cholesky Decomposition models linking objective sleep duration and BMI, WC, and percent body fat at eight years of age were highly similar and showed that all of the covariance between objective sleep duration and each weight indicator was explained by shared additive genetic factors, although the magnitude of the covariance differed across these models. Specifically, 10% of the total variance in percent body fat was explained by objective sleep duration, whereas only 3% of the variance in WC and 6% of the variance in BMI was explained by objective sleep duration. Further, additive genetic influences on sleep duration were highly correlated with additive genetic influences on BMI (r_g = .26), WC (r_g = .16), and percent body fat (r_g = .32), suggesting that some of the same genes may be influencing objective sleep duration and weight indicators or adiposity. Importantly, these findings differ from at least two prior studies that show associations between sleep duration and BMI in particular are accounted for entirely by common environmental effects (Watson et al., 2010; Watson et al., 2012). However, these prior studies were conducted with a sample of adult twins that was primarily Caucasian (89%), whereas our findings apply to ethnically diverse, young twins living in the same home. Additionally, prior studies assessed self-reported sleep duration, while significant correlations in our study called for examining links between objective sleep duration and weight indicators. Thus, variation in developmental stage, context (i.e., living in the same home or not) at the time of assessment, and measurement of sleep duration (subjective vs. objective) between prior studies and the current dissertation may explain differences in results.

Similarly, my hypotheses regarding genetic and environmental associations between objective sleep efficiency and weight indicators were supported. Bivariate Cholesky Decomposition models linking objective sleep efficiency and BMI, WC, and percent body fat at eight years of age demonstrated that the covariance between objective sleep duration and each weight indicator was solely explained by shared additive genetic factors, with the magnitude of the covariance slightly shifting across these models. Specifically, 17% of the total variance in percent body fat was explained by objective sleep efficiency, whereas 9% of the variance in WC

and 14% of the variance in BMI was explained by objective sleep efficiency. Further, additive genetic influences on sleep efficiency were highly correlated with additive genetic influences on BMI (r_g = .39), WC (r_g = .32), and percent body fat (r_g = .43), suggesting that some of the same genes may be influencing objective sleep efficiency and weight indicators or adiposity. It is also noteworthy that the additive genetic covariance and correlations between sleep efficiency and weight indicators were larger than additive genetic covariance and correlations between sleep duration and each weight indicator in bivariate models, suggesting slightly stronger genetic links between sleep efficiency and weight indicators compared to relations between sleep duration and weight indicators. While much of the current literature reports links between sleep duration and weight indictors like BMI, these findings highlight the importance of sleep quality and other weight indicators like WC and percent body fat that may more directly measure adiposity. Specifically, no prior studies have examined possible genetic links between sleep quality and weight indicator. Additionally, these findings show that sleep quantity and quality are associated with multiple measures of adiposity at a genetic or biological level, not just a phenotypic or behavioral level.

Indeed, theory outlines biological and endocrine links between short sleep and increased adiposity (Miller & Cappuccio, 2007; Spiegel et al., 2004). However, beyond theoretical hypotheses, new lines of research suggest that specific genes such as the *Clock* gene are responsible for maintaining circadian rhythms and sleep patterns as well as alterations in metabolism (Laposky, Bass, Kohsaka, & Turek, 2007; Vitaterna, 1994). Studies with mice have demonstrated that mutations in the *Clock* gene lead to significant alterations in sleep, activity and eating that result in less sleep, increased eating, lower leptin levels, and obesity (Laposky et al., 2008; Naylor et al., 2000; Turek, 2005). Importantly, genes like the *Clock gene* that regulate both sleep and metabolism may not be present only in the brain, but have also been documented in various areas of the body such as adipose tissue, suggesting wide spread effects of this gene on multiple aspects of health (Laposky et al., 2007). Thus, this line of research and findings from the current dissertation suggest that there are common, underlying genetics that may contribute to and explain links between various aspects of sleep and weight indicators. These findings also provide a foundation for future studies to examine candidate genes like the *Clock* gene, and its

influence on circadian patterns, sleep, and metabolism in humans at different stages of development, such as childhood, to determine how specific genes or sets of genes modulate multiple aspects of health and well-being like sleep and weight.

Bivariate EC, Sleep, and Weight Models. My hypotheses that additive genetics would primarily explain links between EC and sleep duration and efficiency were supported. I found that the covariance between EC and objective sleep duration, as well as between EC and objective sleep efficiency at eight years of age, was entirely accounted for by shared additive genetic factors. However, the additive genetic covariance (sleep duration A21 = .03; sleep efficiency A21 = .01) and correlations between EC and sleep duration and efficiency were relatively small (sleep duration r_g = .20; sleep efficiency r_g = .15), suggesting that while common, underlying genes may explain links between EC and sleep quantity and quality, these findings should not be given much weight and genetic links between EC and weight indicators may be weak. Bivariate findings for associations between EC and BMI were similar to genetic and environmental links between EC and sleep duration and efficiency; the covariance between EC and BMI at eight years of age was solely accounted for by additive genetic factor, although additive genetic covariance (A21 = .02) and correlation between EC and BMI (r_g = .12) was quite small. This again indicates that while common, underlying genes may explain links between EC and BMI, these findings should be interpreted cautiously.

In terms of genetically-influenced factors that may account for links between EC and sleep, as well as between EC and BMI, there are a number of possibilities. Regarding genetic links between EC and sleep quantity and quality, it is possible that general dysregulation (as influenced by multiple genes) may explain associations between EC and objective sleep. Prior studies show that self-regulation and sleep regulation or dysregulation both have moderate to high additive genetic influence (Lemery-Chalfant et al., 2013; Saudino & Micalizzi, 2015); thus, it is possible that one set of genes regulates multiple aspects of behavior and/or contributes to both of these characteristics or qualities.

In contrast, my hypothesis that additive genetics would primarily explain links between EC and parent-reported sleep duration was not supported, as I found that the covariance

between EC and parent-reported sleep duration, primarily accounted for by shared environmental factors (A21 = .05) with a small contribution of nonshared environmental factors (E21 = .01). While counter to my hypothesis, these findings may fit with at least one study of twins assessed during middle childhood that found that greatest proportion of the variance in poor self-regulatory eating was accounted for by shared environmental factors, with the remaining variance primarily attributed to nonshared environmental influences (Faith et al., 2012). Thus, the current findings and those from Faith et al. (2012) suggest that some aspect(s) of the twins' shared and nonshared environments may account for links between EC and parent-reported sleep duration. As with links between EC and objective sleep duration, it is possible that general regulation or dysregulation may explain associations between EC and parent-reported sleep duration. For example, children who are able to better regulate their thoughts, behaviors and emotions more broadly are likely able to better regulate their sleep, including falling asleep, staying asleep, or going to bed when instructed, whereas children who have difficulty with regulating thoughts, behaviors and emotions may struggle to regulate various aspects of sleep (Dahl, 1996). At least one study has shown that increased emotional intensity and lower emotional regulation before bedtime predicts shorter sleep duration and greater sleep disturbances in middle childhood (including sleep duration; El-Sheikh & Buckhalt, 2005). Additionally, research shows that EC in toddlerhood is linked to children's ability to regulate other emotions such as anger, joy, and restraint (Kochanska, Murray, & Harlan, 2000), suggesting that emotion regulation ability or parents' fostering of emotion regulation may serve as a common environmental factor that links EC and parent-reported sleep duration in middle childhood. Furthermore, household schedules and routines may explain links between EC and parent-reported bedtime in middle childhood. At least one recent study found that toddlers whose parents reported that they had regular bedtimes and mealtimes also showed higher emotional self-regulation (Anderson et al., 2017). Furthermore, the same study found that lower emotional self-regulation and less regular bedtimes in toddlerhood predicted increased odds of obesity in middle childhood, suggesting links new links between EC, sleep timing and scheduling and weight status.

Bivariate Weight Models. My hypotheses regarding genetic and environmental associations between various weight indicators were all supported. All of the final, best fitting Bivariate Cholesky decomposition models linking a) BMI and WC, b) WC and percent body fat, and c) BMI and percent body fat at eight years of age were highly similar and showed that all of the covariance between weight indicators in each model was explained by shared additive genetic factors, although the magnitude of the covariance slightly differed across these models. Specifically, 80% of the total variance in percent body fat was explained by BMI, whereas only 70% of the variance in WC was explained by BMI and 79% of the variance in percent body fat was explained by WC. Further, additive genetic influences on BMI were highly correlated with additive genetic influences on WC (r_g = .87) and percent body fat (r_g = .93), and additive genetic influences on WC were highly correlated with additive genetic influences on percent body fat (r_g = .93), suggesting that some of the same genes may be influencing BMI, WC, and percent body fat. Indeed, I expected high shared additive genetic influence on associations between all weight indicators in the current dissertation given that all three weight indicators were proxy measures for adiposity and should be highly related to one another given that some of the same measurements contribute to scores on BMI, WC, and percent body fat. For example, a form of body mass is used to score both BMI and percent body fat, and WC and percent body fat both estimate some form of actual body fatness rather than just overall body mass. As such, common sets of genes almost certainly contribute to links between weight indicators. However, remaining differences between individuals on weight indicators and associations between various weight indicators can be attributed to nonshared environmental factors, or characteristics that make twins more different from one another. Thus, associations between weight indicators in the current dissertation may also be explained by differences in lifestyle factors like activity level, food intake (both amount and type), sedentary time and behavior, and other factors like metabolism possibly. These findings also support results from an adult twin study that show moderate nonshared environmental influences on bivariate associations between self-reported sleep duration and BMI (Watson et al., 2010; Watson et al., 2012); however, these results have not been examined or shown in child samples before the current dissertation.

Overall, my findings show that many of the associations between sleep indicators, sleep and weight indicators, and among weight indicators can be attributed to shared additive genetic factors, suggesting that common, underlying sets of genes explain these relations. Further, links between EC and objective sleep indicators and BMI were explained by additive genetic factors, although these relations were weak and should be interpreted with caution. Parent-reported sleep duration and sleep midpoint variability showed strong shared environmental covariance with other sleep indicators and EC suggesting that factors in twins' shared environments like family and daily schedules may contribute to associations between sleep duration and sleep midpoint variability, and their links with other sleep parameters and EC. Finally, it is critical to note that while many bivariate associations between sleep, weight indicators and EC are explained by additive genetic influences, these models and associations do not capture gene x environment interactions, which further elucidate under which environmental conditions genes are more or less likely to be expressed. As such, associations among sleep, weight and EC indicators may change depending on the context or environment, as well as the extent to which these health behaviors are influenced by sets of genes.

Aim 2b Findings and Interpretation

Given that there were not significant correlations or phenotypic associations among sleep, weight indicators and EC at eight years of age, Multivariate Cholesky Decompositions were not fit and do not warrant interpretation.

Aim 2c Findings and Interpretation

Finally, my hypothesis that children classified as overweight or obese at eight years of age would show higher additive genetic influence on weight status compared to children classified as normal/healthy weight or underweight was not supported. Rather, the best-fitting Liability Threshold Model constrained all paths and cut points to be the same across twin and zygosity groups. Thus, the model showed that contributions to overweight or obesity status was almost evenly split between additive genetic and shared environmental influences, suggesting lower heritability than BMI, WC, or percent body fat alone, and much lower heritability for obesity than has been reported in other samples of children and adults in previous literature (see Plomin

et al., 2014). These findings suggest that when children demonstrate greater weight status and more adiposity, genetic influences on weight may actually be restricted or have less genetic expression. This finding is counter to results with adult samples of twins showing that restricted sleep may provide an opportunity or environment that allows for greater genetic expression of BMI or weight more broadly, whereas longer sleep duration may restrict genetic expression of BMI or weight (Watson et al., 2010; Watson et al., 2012). Additionally, aspects of twins' shared environment may heavily contribute to their weight status, holding just as much importance as additive genetic influences. As previously noted, lifestyle factors like activity level, food intake (both amount and type), and sedentary time and behavior may all serve as common factors in the home and family environment that contribute to weight status in middle childhood.

Strengths, Limitations, and Future Directions

The current dissertation is characterized by a number of strengths both conceptually and methodologically. The current dissertation utilized a longitudinal sample of twins recruited through state birth records, making this a community sample of socioeconomically and ethnically diverse families. This is highly valuable as many twin studies have been conducted with ethnically homogeneous samples of European American children or adults and estimates of genetic and environmental influences on traits likely vary according to population or sample composition (Plomin et al., 2013). The current dissertation also employs multimethod assessment of multiple aspects of sleep and repeated assessment of objective weight indicators, allowing me to control for prior scores on weight indicators in models and show change in weight over time and control for the high stability of weight indicators over time. Furthermore, the use of a twin sample allows for elucidating genetic and environmental influences on particular traits, as well as genetic and environmental influences on associations between traits and behaviors, which can help identify where to best direct intervention efforts for traits like sleep and weight.

Despite addressing numerous gaps in the current literature, the current dissertation has a number of limitations. First, only partial data were available at the nine-year assessment. As such, the sample size at the nine-year assessment was not large enough to allow for longitudinal analyses to be conducted within quantitative behavior genetic models, limiting all quantitative

behavior genetic models to the eight-year assessment. Additionally, with more complete data from the nine-year assessment, it is possible that phenotypic analyses may slightly change and reveal different associations between sleep, EC, and weight indicators. Thus, analyses should be examined again with the larger sample to determine whether results hold with additional data. Second, sleep was only assessed at the eight-year assessment, making it difficult to characterize the actual direction of effects between sleep and weight indicators. The current dissertation draws on theoretical and prior empirical findings that show sleep problems likely precede increase in weight and body fat, but without multiple longitudinal measurements of sleep and weight, direction of effects and bidirectional associations cannot be determined. Furthermore, longitudinal relations between sleep, EC, and weight indicators were only assessed across one year, making this a short-term longitudinal study. While the phenotypic analyses in the current dissertation provide valuable information, it is still unclear how sleep, weight, and EC are associated with one another over longer periods of time and into early adolescence.

Additionally, the current dissertation relied on multivariate regression tests for phenotypic analyses. While regression analyses fit the aims of the current dissertation, they assume linear associations between sleep, EC, and weight over time. Testing moderation and mediation of psychosocial (i.e., EC) and other demographic factors in the current dissertation would further clarify pathways and mechanisms in associations between sleep and weight indicators. Further, utilizing longitudinal growth modeling with these analyses would allow modeling of sleep and weight trajectories over time and give a more nuanced picture of changes in sleep and weight across middle childhood. In addition, while I was able to conduct univariate and bivariate quantitative behavior genetic models in the current dissertation to estimate genetic and environmental influences on associations between sleep, EC, and weight, phenotypic correlations were not high enough or significant to warrant more conducting more complex behavior genetic links among sleep, EC, and weight.

Finally, the current dissertation examined specific associations between unique sleep and weight indicators. While I was able to detect significant associations between various sleep and weight indicators, high correlations among some aspects of sleep in the sample and all of the

weight indicators may warrant creating a latent variable to characterize optimal or poor sleep, as well as a single indicator of weight. Using this approach may make findings more robust and provide a clearer, overall picture of associations between sleep and weight in childhood. On the other hand, other sleep parameters and weight indicators may also be important to test when considering broad associations between sleep problems and weight, such as sleep latency (time taken to fall asleep), sleep start time (bedtime) variability, wake time variability, and total body composition. Future studies should test these points in an effort to provide a more complete picture regarding the associations between sleep problems and weight in childhood.

Conclusions

When children experience short sleep duration and greater variability in bedtimes and waketimes, children with low EC may also show the greatest increases in BMI and percent body fat (respectively) from eight to nine years of age. Further, these findings suggest that children with low EC may experience the greatest benefits from attaining longer sleep duration and greater regularity in bed and waketimes from day to day. My findings also showed greater environmental influences on parent-reported sleep duration and quality, as well as objective sleep midpoint variability, suggesting that factors in the home or sleep environment (e.g., family routines and schedules, parent report bias) may explain why twins' similarities on various aspects of sleep. Similarly, associations between parent-reported sleep duration and sleep midpoint variability and other sleep indicators and EC were primarily accounted for by shared environmental factors, suggesting that factors in twins' shared environments like family and daily schedules explain these links. In contrast, I found high additive genetic influence on objective sleep quantity and quality, all weight indicators, and parent-reported EC. Further, many of the associations between sleep indicators, sleep and weight indicators, and among weight indicators were entirely accounted for by shared additive genetic factors, suggesting that common, underlying sets of genes explain these relations.

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APPENDIX A

TABLES

Table 1

Summary of Demographic Information for Full Analytic Sample

Demographic Summary	*n*	%
Sex		
Male	299	49.2%
Female	309	50.8%
Twin ethnicity		
European American	342	56.6%
Hispanic/Latino	150	24.8%
Asian American	22	3.6%
African American	24	4.0%
Native American	16	2.6%
Native Hawaiian	6	1.0%
Multiethnic or Unknown	48	8.0%
Zygosity[a]		
Monozygotic (MZ)	178	29.6%
Same-sex dizygotic (DZ_{ss})	234	38.9%
Opposite-sex dizygotic (DZ_{os})	190	31.6%
Diary completion[b]		
Paper	44	7.2%
Electronic	553	91.9%
Both	9	1.5%
Summer participation[c]		
Completed study week during school year	372	71.0%
Completed study week during summer or break	152	29.0%
Primary caregiver education level[d]		
Some or less than high school	4	0.7%
High school graduate/GED	56	9.3%
Some college	166	27.5%
College degree	222	36.8%
Some graduate education	20	3.3%
Graduate or professional degree	136	22.5%
Secondary caregiver education level[e]		
Some or less than high school	8	1.6%
High school graduate/GED	76	14.7%
Some college	138	26.7%

College degree	172	33.3%
Some graduate education	18	3.5%
Graduate or professional degree	104	20.2%
Income-to-needs Ratio[f]		
Living in poverty (score of < 1)	46	7.6%
Near the poverty line (score of 1-2)	118	19.1%
Lower middle class (score of 2-3)	80	13.5%
Middle to upper class (score of 3+)	260	42.8%
Weight status at 8-year assessment[g]		
Underweight	26	5.4%
Normal/Healthy weight	354	73.8%
Overweight	64	13.3%
Obese	36	5.9%
Weight status at 9-year assessment[h]		
Underweight	31	11.2%
Normal/Healthy weight	191	69.2%
Overweight	31	11.2%
Obese	23	8.3%

Note. N = 608. Primary caregivers reported on twin sex, ethnicity, zygosity, primary caregiver education, secondary caregiver education, and total household income before taxes. Daily diaries were completed during a week-long study protocol. Zygosity was collected using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991). Whether participants completed their study week during week during an extended school break (i.e., summer or winter break) was determined by cross-checking study participation dates with school calendars for each twin pair. Income-to-needs ratios were computed by dividing total household income before taxes by the federal household income threshold (based on the number of individuals supported by the household income) for 2016-2017. Weight status at eight and nine years was computed by determining whether each participant met criteria for a specific weight status group on at least two of the three weight indictors: BMI scores, waist circumference, and percent body fat. [a]Zygosity unknown for 1.0% of participants (N = 6). [b]Daily diaries not completed by .4% of participants (N = 2). [c]Summer participation unknown for 13.8% of participants (N = 84). [d]Primary caregiver education level unknown for .7% of participants (N = 4). [e]Secondary caregiver education level unknown for 15.1% of participants. (N = 92). [f]Income-to-needs ratio unknown for 17.1% of participants (N = 104). [g]Weight status at 8 years unknown for 21.1% of participants (N = 128). [h]Weight status at 9 years unknown for 54.6% of participants (N = 332).

Table 2

Raw Means, Standard Deviations, Ranges, Skewness, and Kurtosis for Key Study Variables in Full Analytic Sample

Study Variables	*M*	*SD*	Min	Max	Skewness	Kurtosis
Parent-reported sleep duration (hours; 8 year)	9.65	.86	6.33	13.00	-.24	.61
Parent-reported daytime sleepiness (8 year)[a]	2.69	2.75	1.00	23.00	5.13	27.86
Nighttime sleep duration (hours; 8 year)	8.08	.74	4.46	10.26	-.72	1.83
Sleep efficiency (%; 8 year)	89.89	5.91	55.90	99.45	-1.37	3.75
Sleep midpoint time variability (8 year)	.58	.30	.08	1.91	1.24	2.48
Body mass index (BMI; 8 year)[b]	16.86	2.94	12.62	34.92	2.07	6.37
Waist circumference (WC; 8 year)	22.82	3.00	17.50	36.40	1.53	2.92
Percent body fat (8 year)	20.23	6.45	8.42	50.68	1.43	2.57
Effortful control composite (8 year)	3.30	.54	1.81	4.45	-.20	-.48
Body mass index (BMI; 9 year)	17.40	3.28	12.65	34.21	1.74	4.58
Waist circumference (WC; 9 year)[c]	23.19	4.08	16.00	48.50	2.17	9.90
Percent body fat (9 year)	20.38	7.36	4.10	45.83	.97	.93
Age (8 year)	8.52	.63	6.97	9.97	-.21	-.09
Pubertal status (8 year)	1.32	.27	1.00	2.40	.73	.46
Socioeconomic status composite (SES; 8 year)	.00	.66	-1.20	3.08	1.12	2.01

Note. N = 608. Primary caregivers reported on child average nighttime sleep duration, daytime sleepiness, effortful control, pubertal status, total household income before taxes, and primary and secondary caregiver education levels. Parent-reported nighttime sleep duration and daytime sleepiness were assessed using the Child Sleep Habits Questionnaire (Owens et al., 2000); nighttime sleep duration was assessed with a single item and daytime sleepiness was sum score of 7 items. Nighttime sleep duration, sleep efficiency, and sleep midpoint variability were collected from each twin using wrist-based accelerometers during a week-long study protocol. BMI, WC, and percent body fat were collected at two home visits at eight and nine years of age. Effortful control was a composite of three scales from the Temperament in Middle Child Questionnaire (Simonds, 2006; Putnam & Rothbart, 2006): activation control, inhibitory control, and attentional focusing. Pubertal status was assessed with the Pubertal Developmental Scale (Petersen et al., 1988) and mean scores were computed for each twin based on sex. Socioeconomic status was a standardized mean composite of primary caregiver highest level of education, secondary caregiver highest level of education, and income-to-needs ratio. [a]Given significant skew and kurtosis, parent-reported daytime sleepiness was windorized at 3 SDs and logarithmically transformed to estimate a more normal distribution. The windorized and logarithmically transformed variable was used for analyses. Raw scores for the non-windorized and transformed variable are reported here. [b]Given significant skew and high kurtosis, BMI (8 years) was windorized at 3 SDs to estimate a more normal distribution. The windorized variable was used for

analyses. Raw scores for the non-windorized variables are reported here. [c]Given significant skew and kurtosis, WC (9 years) was windorized at 3 SDs to estimate a more normal distribution. The windorized variable was used for analyses. Raw scores for the non-windorized variable are reported here.

Table 3

Zero-order Correlations for Key Study Variables in Full Analytic Sample

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. Parent-reported sleep duration (hours; 8 year)	-																			
2. Parent-reported daytime sleepiness (8 year)[a]	-.16*	-																		
3. Nighttime sleep duration (hours; 8 year)	.36***	-.04	-																	
4. Sleep efficiency (%; 8 year)	-.02	.02	.68***	-																
5. Sleep midpoint time variability (8 year)	-.24***	-.06	.20***	-.01	-															
6. Body mass index (BMI; 8 year)[b]	-.12*	-.06	-.19***	-.13**	.01	-														
7. Waist circumference (WC; 8 year)	-.08+	-.09+	-.16***	-.14**	-.03	.86***	-													
8. Percent body fat (8 year)	-.14**	-.04	-.18***	-.12*	.04	.91***	.78***	-												
9. Weight Status (8 year)	-.03	-.04	-.13*	-.11*	-.02	.75***	71***	.75***	-											
10. Effortful control composite (8 year)	.17***	-.10*	.18***	.13*	-.04	-.12*	-.09+	-.06	-.03	-										
11. Body mass index (BMI; 9 year)	-.04	-.01	-.19**	-.15*	.02	.93***	.82***	.84***	.69***	-.14*	-									
12. Waist circumference (WC; 9 year)[b]	-.03	-.03	-.12*	-.14*	.05	.59***	.58***	.57***	.42***	-.15*	.60***	-								
13. Percent body fat (9 year)	.08	.09	-.20***	-.13*	.04	.84***	.75***	.87***	.69***	-.08	.90***	.53***	-							
14. Weight Status (9 year)	-.01	.04	-.15*	-.13*	.06	.70***	.65***	.67***	.72***	-.10	.77***	.45***	.77***	-						
15. Sex	.04	.05	.15**	.12**	.02	.01	-.07	.17**	.05	.21***	-.04	-.03	.19***	-.01	-					
16. Race/ethnicity	.29***	-.07+	.14**	-.01	-.15***	-.13**	-.10*	-.20***	-.04	-.01	-.10*	-.09	-.17*	-.08	-.05	-				
17. Age (8 year)	-.26***	-.28***	-.22***	-.01	.24***	.15***	.08+	.10*	.03	.04	.07	.18**	.05	.01	.04	-.10*	-			
18. Summer (8 year)	.04	-.03	-.01	-.03	-.15***	-.01	.07	-.01	.06	.01	-.05	-.03	-.01	-.05	.01	.01	-.08	-		
19. Pubertal status (8 year)	-.07+	-.02	.09*	.15***	-.01	.26***	.24***	.30***	.21***	.09*	.20***	.25***	.25***	.17**	.23***	-.13**	.13***	-.05	-	
20. Socioeconomic status composite (SES; 8 year)	.09*	.28***	.14***	.13**	-.20***	-.20***	-.10**	-.20***	-.10*	.22***	-.21**	-.33***	-.16**	-.15*	.07+	.08*	-.20***	.03	-.12**	-

Note. $N = 608$. Parent-reported nighttime sleep duration and daytime sleepiness were assessed using the Child Sleep Habits Questionnaire (Owens et al., 2000). Nighttime sleep duration, sleep efficiency, and sleep midpoint variability were collected from each twin using wrist-based accelerometers during a week-long study protocol. BMI, WC, and percent body fat were collected at two home visits at eight and nine years of age. Weight status at 8 and 9 years was computed such that each participant must fall into a given weight status group (i.e., underweight, normal/healthy, overweight, obese) on least two of the three weight indicators. For analyses, weight status was further scored such that 0 = not overweight/obese and 1 = overweight/obese. Effortful control was a composite of the activation control, inhibitory control, and attentional focusing scales from the Temperament in Middle Child Questionnaire (Simonds, 2006; Putnam & Rothbart, 2006). Summer indicates whether the study week protocol was completed when children were in school (0) or on a school break (1; i.e., summer or winter break). Pubertal status was assessed with the Pubertal Developmental Scale (Petersen et al., 1988) and mean scores were computed for each twin based on sex. Socioeconomic status was a standardized mean composite of primary caregiver highest level of education, secondary caregiver highest level of education, and income-to-needs ratio. [a]Parent-reported daytime sleepiness was windorized at 3 SDs and logarithmically transformed for analyses; windorized and transformed daytime sleepiness was used for zero-order correlations. [a]BMI at 8 years and WC at 9 years were windorized at 3 SDs for analyses; windorized values were used for zero-order correlations.

Table 4

Twin Intraclass Correlations (ICC to Show MZ and DZ Twin Similarity on Predictor, Moderator, and Outcome Variables

Sleep, Effortful Control and Weight Variables	MZ	Same-sex DZ	Opposite-sex DZ
Parent-reported sleep duration (8 year)	.87	.81	.78
Daytime sleepiness (8 year)[a]	.93	.88	.62
Objective sleep duration (8 year)	.84	.47	.44
Objective sleep efficiency (8 year)	.84	.50	.43
Objective sleep midpoint variability (8 year)	.83	.83	.69
Child effortful control (8 year)	.73	.43	.42
Child body mass index (BMI; 8 year)[b]	.92	.59	.01
Child waist circumference (8 year)	.90	.60	.10
Child percent body fat (8 year)	.93	.53	.04
Child body mass index (BMI; 9 year)	.81	.64	.10
Child waist circumference (9 year)[c]	.84	.68	.28
Child percent body fat (9 year)	.85	.65	.11

Note. *N* = 608. Heritability estimates were calculated assuming full ACE models. [a]Parent-reported daytime sleepiness at 8 years was windorized to 3 *SD*s for analyses; raw scores and statistics are presented here. [b]BMI scores at 8 years were windorized to 3 *SD*s for analyses; raw scores and statistics are presented here. [c]Waist circumference at 9 years were windorized to 3 *SD*s for analyses; raw scores and statistics are presented here.

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Table 5

Full and Best-fitting Univariate Cholesky Decomposition Model Fit Statistics for Sleep, Weight Indicators, and Effortful Control

Scale	Model	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
Parent-reported sleep duration (8 year)	**ACE**	**1112.35**	**565**	**-17.65**	--	--	--
Parent-reported daytime sleepiness (8 year)	**ACE**	**-72.73**	**575**	**-1222.73**	**--**	**--**	**--**
Nighttime sleep duration (8 year)	ACE	889.95	455	-20.05	--	--	--
	AE	**890.56**	**456**	**-21.44**	**1**	**.60**	**.44**
Sleep efficiency (8 year)	ACE	2819.34	455	1909.22	--	--	--
	AE	**2821.43**	**456**	**1909.43**	**1**	**2.21**	**.14**
Sleep midpoint time variability (8 year)	ACE	-41.80	455	-951.80	--	--	--
	CE	**-40.61**	**456**	**-952.61**	**1**	**1.19**	**.27**
Body mass index (BMI; 8 year)[a]	ACE	1428.69	321	786.69	--	--	--
	AE	**1429.27**	**322**	**785.27**	**1**	**.59**	**.44**
Waist circumference (WC; 8 year)[a]	ACE	1500.83	318	864.83	--	--	--
	AE	**1501.95**	**319**	**863.95**	**1**	**1.13**	**.29**
Percent body fat (8 year)[a]	ACE	1925.66	307	1311.66	--	--	--
	AE	**1925.67**	**308**	**1309.67**	**1**	**0.01**	**.93**
Effortful control composite (8 year)	ACE	723.29	513	-302.71	--	--	--
	AE	**723.68**	**514**	**-304.32**	**1**	**.39**	**.53**

Note. Bolded models denote the best fitting models for each predictor, moderator, 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 -2LL is the change in -2 log likelihood values when dropping model parameters. *p* denotes the p-value level of significance for the chi-squared test. [a]BMI, WC, and percent body fat variance components were estimated for the sample by excluding opposite-sex DZ twin pairs.

Table 6

Full and Best-fitting Univariate ACE Model Estimates for Sleep, Weight Indicators, and Effortful Control

Scale	Model	A	C	E
Parent-reported sleep duration (8 year)	**ACE**	**.21 (.16-.39)**	**.66 (.47-.83)**	**.13 (.07-.13)**
Parent-reported daytime sleepiness (8 year)	**ACE**	**.27 (.02-1.29)**	**.66 (.55-.83)**	**.07 (.05-.09)**
Nighttime sleep duration (8 year)	ACE	.69 (.45-1.00)	.12 (.00-.50)	.19 (.13-.27)
	AE	**.81 (.67-.97)**	**--**	**.19 (.13-.26)**
Sleep efficiency (8 year)	ACE	.58 (.35-.88)	.20 (.03-.53)	.22 (.15-.30)
	AE	**.79 (.65-.95)**	**--**	**.21 (.15-.28)**
Sleep midpoint time variability (8 year)	ACE	.10 (.00-.34)	.71 (.53-.92)	.19 (.13-.27)
	CE	**--**	**.77 (.62-.94)**	**.23 (.19-.27)**
Effortful control composite (8 year)	ACE	.67 (.41-1.00)	.09 (.02-.52)	.26 (.17-.33)
	AE	**.76 (.61-.93)**	**--**	**.24 (.17-.31)**
Body mass index (BMI; 8 year)[a]	ACE	.92 (.57-1.11)	.00 (.00-.00)	.08 (.05-.10)
	AE	**.93 (.76-1.11)**	**--**	**.07 (.05-.10)**
Waist circumference (WC; 8 year)[a]	ACE	.91 (.53-1.05)	.00 (.00-.00)	.09 (.05-.11)
	AE	**.92 (.75-1.10)**	**--**	**.08 (.06-.11)**
Percent body fat (8 year)[a]	ACE	.92 (.65-1.23)	.00 (.00-.00)	.08 (.05-.10)
	AE	**.92 (.76-1.12)**	**--**	**.08 (.03-.10)**

Note. A = additive genetic components, C = shared environmental component, and E = nonshared environmental component. Bolded models denote the best fitting model. A, C and E are standardized variance components or estimates according to the total variance for that phenotype. Variance-based confidence intervals are presented in parentheses and are based on standardized path estimates. [a]BMI, WC, and percent body fat variance components were estimated for the sample by excluding opposite-sex DZ twin pairs.

Table 7

Univariate Scalar Sex-limitation Cholesky Decomposition Fit Statistics for Weight Indicators at Eight Years of Age

BMI Models	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
ACE – Full Scalar Model	2106.05	465	1176.05	--	--	--
ACE Non-scalar Model	2109.92	466	1177.92	1	3.87	<.01
AE Scalar Model	**2108.77**	**467**	**1174.77**	**2**	**2.72**	**.25**
E Scalar Model	2273.50	470	1333.50	5	167.45	<.001
AE Non-scalar Model	2128.46	470	1188.46	5	22.41	<.001
E Non-scalar Model	2277.52	471	1335.52	6	171.47	<.001
WC Models	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
ACE – Full Scalar Model	2217.20	462	1293.20	--	--	--
ACE Non-scalar Model	2220.29	463	1294.29	1	3.09	<.01
AE Scalar Model	**2217.91**	**464**	**1289.91**	**2**	**.71**	**.07**
E Scalar Model	2378.34	467	1444.34	5	161.15	<.001
AE Non-scalar Model	2234.03	467	1300.03	5	16.83	<.01
E Non-scalar Model	2379.80	468	1443.80	6	162.60	<.001
Percent Body Fat Models	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
ACE – Full Scalar Model	2797.80	441	1915.80	--	--	--
ACE Non-scalar Model	2801.77	442	1917.77	1	3.98	<.01
AE Scalar Model	**2799.83**	**443**	**1913.83**	**2**	**2.03**	**.36**
E Scalar Model	2937.46	446	2045.46	5	139.67	<.001
AE Non-scalar Model	2814.80	446	1922.80	5	17.00	<.01
E Non-scalar Model	2941.57	447	2047.57	6	143.77	<.001

Note. Bolded models denote the best fitting models for each weight indicator. Scalar models allow the proportion of variance accounted for by A, C, and E components to change based on a scalar (i.e., k) and the total variance to differ across males and females. Non-scalar sex-limitation models allow the proportion of variance accounted for by A, C, E, and the total variance to differ across males and females. 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 -2LL 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 8

Full and Best-fitting Scalar Sex-limitation Univariate ACE Model Estimates for Weight Indicators

Scale	Model	A	C	E
Body mass index (BMI) – Males	ACE	.95 (.04-3.25)	--	.05 (.02-.06)
	AE	**.95 (.91-.97)**	**--**	**.05 (.03-.09)**
Body mass index (BMI) – Females	ACE	.58 (.01-2.74)	.32 (.01-1.16)	.10 (.03-.24)
	AE	**.90 (.83-.94)**	**--**	**.10 (.06-.17)**
Waist circumference (WC) - Males	ACE	.89 (.07-2.96)	.06 (.02-.07)	.05 (.01-.06)
	AE	**.95 (.91-.97)**	**--**	**.05 (.03-.09)**
Waist circumference (WC) - Females	ACE	.72 (.06-2.81)	.16 (.01-.31)	.12 (.02-.21)
	AE	**.88 (.80-.93)**	**--**	**.12 (.07-.20)**
Percent body fat - Males	ACE	.95 (.45-1.76)	--	.05 (.01-.06)
	AE	**.95 (.91-.97)**	**--**	**.05 (.03-.09)**
Percent body fat - Females	ACE	.62 (.32-1.54)	.28 (.03-.40)	.10 (.01-.12)
	AE	**.90 (.83-.94)**	**--**	**.10 (.06-.17)**

Note. Best fitting full and reduced ACE models for males and females were scalar models which allow the proportion of variance accounted for by A, C, and E components to change based on a scalar (i.e., k) for males and females and the total variance to differ across males and females. A = additive genetic components, C = shared environmental component, and E = nonshared environmental component. Bolded models denote the best fitting model. A, C and E are standardized variance components or estimates according to the total variance for that phenotype. Variance-based confidence intervals are presented in parentheses and are based on standardized path estimates.

Table 9

Full and Best-fitting Bivariate Cholesky Decomposition Fit Statistics for Associations between Objective and Subjective Sleep Indicators

Scale	Model	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
Objective Sleep Duration and Sleep Efficiency (8 year)	ACE-ACE	3335.25	909	1517.25	--	--	--
	AE-ACE	**3331.78**	**911**	**1509.78**	**2**	**3.47**	**.99**
Objective Sleep Duration and Sleep Midpoint Time Variability (8 year)	ACE-ACE	839.09	909	-978.91	--	--	--
	ACE-ACE	**839.09**	**911**	**-980.91**	**2**	**.00**	**.99**
Objective Sleep Duration and Parent -reported Sleep Duration (8 year)	ACE-ACE	1973.29	1019	-64.71	--	--	--
	ACE-ACE	**1973.30**	**1020**	**-66.70**	**1**	**.01**	**.94**
Parent-reported Sleep Duration and Sleep Midpoint Time Variability (8 year)	ACE-ACE	1057.88	1019	-980.12	--	--	--
	ACE-ACE	**1058.60**	**1021**	**-983.40**	**2**	**.72**	**.70**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. 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 -2LL 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

Full and Best-fitting Bivariate Cholesky Decomposition Estimates for Correlated Objective and Subjective Sleep Indicators

Scales	Model	A11	C11	E11			
Objective Sleep Duration and Objective Sleep Efficiency	ACE-ACE	.80 (.45-.93)	--	.20 (.13-.27)			
		A21 .34 (.21-.65)	C21 --	E21 .15 (.08-.22)	A22 .14 (.07-.25)	C22 .30 (.06-.52)	E22 .07 (.05-.11)
	AE-ACE[a]	**A11 .80 (.59-.97)**	**C11 --**	**E11 .20 (.16-.32)**			
		A21 .37 (.28-.57)	**C21 --**	**E21 .14 (.08-.21)**	**A22 .14 (.07-.25)**	**C22 .26 (.12-.34)**	**E22 .08 (.05-.11)**
		A11	C11	E11			
Objective Sleep Duration and Sleep Midpoint Variability	ACE-ACE	.72 (.47-1.01)	.08 (.01-.51)	.19 (.13-.26)			
		A21 --	C21 .26 (.09-1.94)	E21 .01 (.01-.22)	A22 .11 (.01-.41)	C22 .44 (.01-1.88)	E22 .18 (.15-.31)
	ACE-ACE[b]	**A11 .72 (.47-1.01)**	**C11 .09 (.01-.51)**	**E11 .19 (.13-.26)**			
		A21 --	**C21 .26 (.09-1.94)**	**E21 .01 (.01-.22)**	**A22 .11 (.01-.41)**	**C22 .44 (.01-1.88)**	**E22 .19 (.15-.31)**
		A11	C11	E11			
Objective Sleep Duration and Parent-reported Sleep Duration	ACE-ACE	.71 (.46-1.00)	.09 (.01-.50)	.20 (.13-.27)			
		A21 --	C21 .66 (.01-1.11)	E21 .01 (.01-.02)	A22 .21 (.14-.36)	C22 --	E22 .12 (.07-.13)
	ACE-ACE[c]	**A11 .70 (.46-1.00)**	**C11 .10 (.01-.50)**	**E11 .20 (.13-.27)**			

		A21 --	**C21 .65 (.01-1.11)**	**E21 .01 (.01-.02)**	**A22 .21 (.14-.36)**	**C22 .01 (.05-1.12)**	**E22 .12 (.07-.13)**
		A11	C11	E11			
Parent-reported Sleep Duration and	ACE-ACE	.22 (.16-.39)	.61 (.47-.83)	.17 (.07-.13)			
Sleep Midpoint Variability		A21 --	C21 .03 (.01-.16)	E21 --	A22 .18 (.01-.34)	C22 .59 (.47-.86)	E22 .20 (.13-.26)
	ACE-ACE[d]	**A11 .21 (.16-.39)**	**C11 .62 (.47-.83)**	**E11 .17 (.07-.13)**			
		A21 --	**C21 .05 (.01-.16)**	**E21 --**	**A22 .18 (.01-.34)**	**C22 .57 (.47-.86)**	**E22 .20 (.13-.26)**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates. CIs and estimates for A21, C21 and E21 in the full and reduced models correspond with the percent of the variance in the second phenotype accounted for by the first phenotype. [a]C11 and C21 paths were dropped it the best fitting model. [b]A21 path was dropped in the best fitting model. [c]A21 path was dropped it the best fitting model. [d]A21 and E21 paths were dropped in the best fitting model.

Table 11

Full and Best-fitting Bivariate Cholesky Decomposition Fit Statistics for Associations between Objective Sleep and Weight Indicators

Scale	Model	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
Objective Sleep Duration and BMI (8 year)	ACE-ACE	1999.69	629	741.69	--	--	--
	ACE-AE	**2001.17**	**631**	**739.16**	**2**	**1.47**	**.48**
Objective Sleep Duration and Waist Circumference (8 year)	ACE-ACE	2077.01	626	825.01	--	--	--
	AE-AE	**2077.40**	**630**	**817.40**	**4**	**.39**	**.98**
Objective Sleep Duration and Percent Body Fat (8 year)	ACE-ACE	2492.14	615	1262.14	--	--	--
	ACE-AE	**2492.38**	**618**	**1256.38**	**3**	**.24**	**.97**
Objective Sleep Efficiency and BMI (8 year)	ACE-ACE	3314.73	629	2056.73	--	--	--
	ACE-AE	**3316.88**	**632**	**2052.88**	**3**	**2.15**	**.54**
Objective Sleep Efficiency and Waist Circumference (8 year)	ACE-ACE	3389.97	626	2137.97	--	--	--
	ACE-AE	**3392.36**	**629**	**2134.36**	**3**	**2.39**	**.50**
Objective Sleep Efficiency and Percent Body Fat (8 year)	ACE-ACE	3809.74	615	2579.74	--	--	--
	ACE-AE	**3810.36**	**618**	**2574.36**	**3**	**.62**	**.89**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. 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 12

Full and Best-fitting Bivariate Cholesky Decomposition Estimates for Correlated Objective Sleep Duration and Weight Indicators

Scales	Model	A11	C11	E11			
Objective Sleep Duration and BMI	ACE-ACE	.58 (.27-.95)	.22 (.02-.70)	.20 (.13-.27)			
		A21	C21	E21	A22	C22	E22
		.06 (.03-2.16)	--	--	.70 (.54-1.20)	.17 (.09-.18)	.07 (.05-.12)
	ACE-AE[a]	**A11**	**C11**	**E11**			
		.58 (.27-.95)	**.22 (.02-.70)**	**.20 (.13-.27)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.06 (.03-.18)	**--**	**--**	**.87 (.71-1.04)**	**--**	**.07 (.05-.10)**
		A11	C11	E11			
Objective Sleep Duration and Waist Circumference	ACE-ACE	.80 (.61-1.02)	--	.20 (.13-.29)			
		A21	C21	E21	A22	C22	E22
		.03 (.01-.15)	.18 (.01-.88)	--	.71 (.25-1.42)	--	.08 (.05-.11)
	AE-AE[b]	**A11**	**C11**	**E11**			
		.80 (.45-.81)	**--**	**.20 (.13-.28)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.03 (.01-.11)	**--**	**--**	**.89 (.71-1.06)**	**--**	**.08 (.06-.11)**
		A11	C11	E11			
Objective Sleep Duration and Percent Body Fat	ACE-ACE	.58 (.27-.95)	.22 (.02-.70)	.20 (.13-.27)			
		A21	C21	E21	A22	C22	E22
		.08 (.01-.39)	--	--	.86 (.49-1.10)	.07 (.07-.64)	.07 (.05-.09)
	ACE-AE[a]	**A11**	**C11**	**E11**			
		.58 (.27-.95)	**.22 (.02-.70)**	**.20 (.13-.27)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.10 (.02-.21)	**--**	**--**	**.83 (.68-1.02)**	**--**	**.07 (.05-.10)**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. CIs and estimates for A21, C21 and E21 in the full and reduced models correspond with the percent of the variance in the second phenotype accounted for by the first phenotype. [a]C21, C22, and E21 paths were dropped it the best fitting model. [b]C211, C21, C22, and E21 paths were dropped it the best fitting model.

Table 13

Full and Best-fitting Bivariate Cholesky Decomposition Estimates for Correlated Objective Sleep Efficiency and Weight Indicators

Scales	Model	A11	C11	E11			
Objective Sleep Efficiency and BMI	ACE-ACE	.32 (.12-.63)	.47 (.23-.80)	.21 (.14-.29)			
		A21 .15 (.11-.52)	C21 --	E21 --	A22 .57 (.30-.95)	C22 .21 (.03-.55)	E22 .07 (.04-.10)
	ACE-AE[a]	**A11 .32 (.12-.63)**	**C11 .47 (.23-.80)**	**E11 .21 (.14-.29)**			
		A21 .14 (.01-.41)	**C21 --**	**E21 --**	**A22 .78 (.56-1.03)**	**C22 --**	**E22 .08 (.05-.09)**
		A11	C11	E11			
Objective Sleep Efficiency and Waist Circumference	ACE-ACE	.36 (.14-.68)	.43 (.19-.78)	.21 (.15-.78)			
		A21 .10 (.01-.41)	C21 --	E21 --	A22 .70 (.35-.93)	C22 .22 (.03-.56)	E22 .08 (.05-.11)
	ACE-AE[a]	**A11 .36 (.32-.82)**	**C11 .43 (.07-.55)**	**E11 .21 (.15-.29)**			
		A21 .09 (.01-.16)	**C21 --**	**E21 --**	**A22 .83 (.71-1.00)**	**C22 --**	**E22 .08 (.06-.12)**
		A11	C11	E11			
Objective Sleep Efficiency and Percent Body Fat	ACE-ACE	.33 (.12-.63)	.46 (.22-.77)	.21 (.06-.29)			
		A21 .17 (.01-.58)	C21 --	E21 --	A22 .65 (.34-1.05)	C22 .11 (.01-.56)	E22 .07 (.05-.09)
	ACE-AE[a]	**A11 .33 (.12-.63)**	**C11 .46 (.22-.77)**	**E11 .21 (.06-.29)**			
		A21 .17 (.02-.46)	**C21 --**	**E21 --**	**A22 .76 (.53-1.03)**	**C22 --**	**E22 .07 (.05-.10)**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. CIs and estimates for A21, C21 and E21 in the full and reduced models correspond with the percent of the variance in the second phenotype accounted for by the first phenotype. [a] C21, C22, and E21 paths were dropped it the best fitting model.

Table 14

Full and Best-fitting Bivariate Cholesky Decomposition Fit Statistics for Associations between Effortful Control, Sleep, and Weight Indicators

Scale	Model	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
EC and Objective Sleep Duration (8 year)	ACE-ACE	1606.08	967	-327.92	--	--	--
	AE-ACE	**1606.61**	**970**	**-333.39**	**3**	**.53**	**.91**
EC and Objective Sleep Efficiency (8 year)	ACE-ACE	3539.89	967	1605.89	--	--	--
	AE-ACE	**3543.68**	**970**	**1603.68**	**3**	**3.79**	**.29**
EC and Parent-reported Sleep Duration (8 year)	ACE-ACE	1840.64	1077	-313.36	--	--	--
	ACE-ACE	**1840.64**	**1078**	**-315.36**	**3**	**2.00**	**.99**
EC and BMI (8 year)[a]	ACE-ACE	1901.15	663	575.15	--	--	--
	ACE-AE	**1901.64**	**666**	**569.64**	**3**	**.49**	**.92**

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 -2LL is the change in chi-squared values when dropping model parameters. *p* denotes the p-value level of significance for the chi-squared test. [a]EC and BMI model excludes DZ opposite-sex twins from models to account for sex differences in BMI. Sex and age were regressed out of variables prior to conducting the model.

Table 15

Full and Best-fitting Bivariate Cholesky Decomposition Estimates for Correlated Effortful Control, Sleep and Weight Indicators

Scales	Model	A11	C11	E11			
EC and Objective Sleep Duration	ACE-ACE	.66 (.41-1.00)	.08 (.02-.52)	.26 (.17-.33)			
		A21	C21	E21	A22	C22	E22
		.02 (.01-.15)	--	--	.70 (.43-.97)	.10 (.01-.50)	.20 (.13-.27)
	AE-ACE[a]	**A11**	**C11**	**E11**			
		.75 (.61-.93)	**--**	**.25 (.17-.32)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.03 (.01-.08)	**--**	**--**	**.67 (.43-.96)**	**.10 (.01-.49)**	**.20 (.13-.27)**
		A11	C11	E11			
EC and Objective Sleep Efficiency	ACE-ACE	.75 (.63-.96)	--	.25 (.17-.32)			
		A21	C21	E21	A22	C22	E22
		.01 (.01-.16)	--	--	.57 (.34-.85)	.20 (.02-.54)	.22 (.15-.29)
	AE-ACE[a]	**A11**	**C11**	**E11**			
		.75 (.61-.93)	**--**	**.25 (.17-.32)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.01 (.001-.05)	**--**	**--**	**.57 (.34-.87)**	**.20 (.03-.53)**	**.22 (.03-.30)**
		A11	C11	E11			
EC and Parent-reported Sleep Duration	ACE-ACE	.16 (.01-.81)	.36 (.09-.82)	.48 (.39-.60)			
		A21	C21	E21	A22	C22	E22
		--	.05 (.01-.31)	.01 (.01-.02)	.17 (.08-.44)	.61 (.33-.84)	.16 (.13-.21)
	ACE-ACE[a]	**A11**	**C11**	**E11**			
		.16 (.01-.81)	**.36 (.09-.82)**	**.48 (.39-.60)**			

		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		--	**.05 (.01-.31)**	**.01 (.01-.02)**	**.22 (.08-.44)**	**.63 (.33-.84)**	**.16 (.13-.21)**
		A11	C11	E11			
EC and Objective BMI	ACE-ACE	.73 (.40-1.16)	.02 (.02-.52)	.25 (.17-.33)			
		A21	C21	E21	A22	C22	E22
		.01 (.01-.09)	.02 (.01-.09)	--	.82 (.56-1.09)	.11 (.05-.12)	.07 (.05-.09)
		A11	**C11**	**E11**			
	AE-AE[b]	**.74 (.40-1.16)**	**--**	**.26 (.17-.33)**			
		A21	**C21**	**E21**	**A22**	**C22**	**E22**
		.02 (.01-.12)	**--**	**--**	**.91 (.74-1.09)**	**--**	**.07 (.05-.10)**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. CIs and estimates for A21, C21 and E21 in the full and reduced models correspond with the percent of the variance in the second phenotype accounted for by the first phenotype. [a]C11, C21, and E21 paths were dropped it the best fitting model. [b]C11, C21, C22, and E21 paths were dropped it the best fitting model.

Table 16

Full and Best-fitting Bivariate Cholesky Decomposition Fit Statistics for Associations between Objective Weight Indicators

Scale	Model	-2LL	*df*	AIC	Δ *df*	Δ -2LL	*p*
BMI and Percent Body Fat (8 year)	ACE-ACE	2794.70	627	1540.70	--	--	--
	AE-AE	**2796.23**	**630**	**1536.23**	**3**	**1.54**	**.67**
BMI and Waist Circumference (8 year)	ACE-ACE	2531.08	638	1255.08	--	--	--
	AE-AE	**2533.30**	**641**	**1251.30**	**3**	**2.23**	**.53**
Waist Circumference and Percent Body Fat (8 year)	ACE-ACE	3090.55	624	1842.55	--	--	--
	ACE-AE	**3090.58**	**626**	**1838.58**	**2**	**.04**	**.98**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models. 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 -2LL 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 17

Full and Best-fitting Bivariate Cholesky Decomposition Estimates for Correlated Objective Weight Indicators

Scales	Model	A11	C11	E11			
Objective BMI and Percent Body Fat	ACE-ACE	.80 (.57-1.07)	.13 (.01-.50)	.07 (.05-.09)			
		A21 .76 (.54-1.01)	C21 .05 (.03-.40)	E21 .05 (.03-.07)	A22 .12 (.08-.16)	C22 --	E22 .02 (.01-.03)
	AE-AE[a]	**A11 .93 (.77-1.12)**	**C11 --**	**E11 .07 (.05-.09)**			
		A21 .80 (.63-.93)	**C21 --**	**E21 .05 (.03-.07)**	**A22 .13 (.10-.16)**	**C22 --**	**E22 .02 (.01-.03)**
		A11	C11	E11			
Objective BMI and Waist Circumference	ACE-ACE	.93 (.76-1.10)	--	.07 (.05-.09)			
		A21 .69 (.47-.95)	C21 .08 (.02-.48)	E21 .04 (.02-.07)	A22 .15 (.12-.23)	C22 --	E22 .04 (.03-.06)
	AE-AE[b]	**A11 .92 (.76-1.10)**	**C11 --**	**E11 .08 (.05-.19)**			
		A21 .70 (.55-.88)	**C21 --**	**E21 .05 (.02-.07)**	**A22 .21 (.17-.27)**	**C22 --**	**E22 .04 (.03-.05)**
		A11	C11	E11			
Objective Waist Circumference and Percent Body Fat	ACE-ACE[c]	.76 (.53-1.05)	.16 (.01-.55)	.08 (.05-.11)			
		A21 .78 (.51-1.10)	C21 --	E21 .02 (.01-.05)	A22 .13 (.06-.23)	C22 .02 (.00-.00)	E22 .05 (.03-.06)
	ACE-AE[c]	**A11 .76 (.60-.94)**	**C11 .16 (.09-.25)**	**E11 .08 (.05-.11)**			
		A21 .79 (.62-.99)	**C21 --**	**E21 .03 (.01-.05)**	**A22 .14 (.07-.23)**	**C22 --**	**E22 .04 (.03-.06)**

Note. All models exclude DZ opposite-sex twins from models to account for sex differences in weight indicators. Sex and age were regressed out of variables prior to conducting models.

Bolded models denote the best fitting models. Variance-based confidence intervals (CIs) are presented in parentheses and are based on standardized path estimates. A11 = additive genetic components for first phenotype, C11 = shared environment component for first phenotype, E11 = nonshared environment component for first phenotype. A21 = additive genetic component shared between first and second phenotypes, C21 = shared environment component shared between first and second phenotypes, E21 = nonshared environment component shared between first and second phenotypes. A22 = additive genetic components for second phenotype, C22 = shared environment component for second phenotype, E22 = nonshared environment component for second phenotype. CIs and estimates for A21, C21 and E21 in the full and reduced models correspond with the percent of the variance in the second phenotype accounted for by the first phenotype. [a]C11, C21, and C22 paths were dropped it the best fitting model. [b]C11, C21, and C22 paths were dropped it the best fitting model. [c]C21 and C22 paths were dropped it the best fitting model.

APPENDIX B

FIGURES

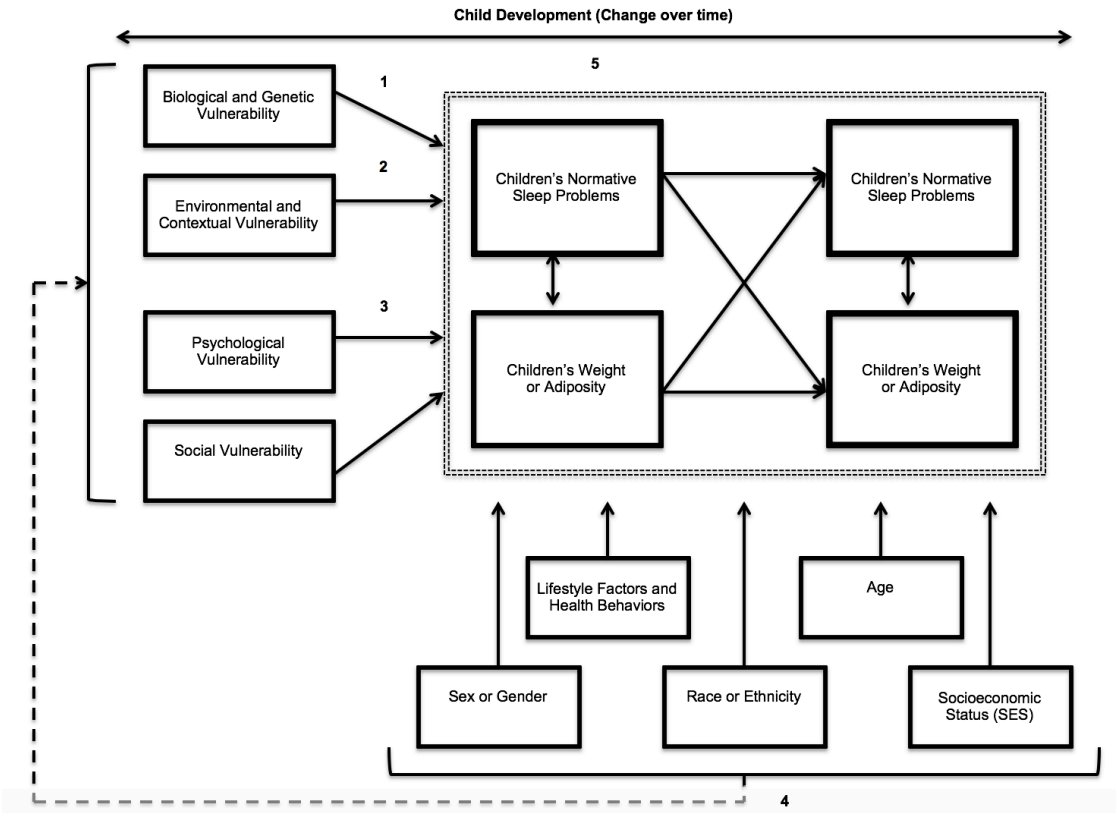

Figure 1. Proposed Conceptual Model. The proposed conceptual model highlights biopsychosocial and contextual influences on associations between child sleep problems and weight across child development, accounting for direct and indirect influences of lifestyle and demographic factors on links between child sleep and weight. Of interest to the current dissertation, Path 1 delineates genetic influences, Path 2 demonstrates environmental and contextual influences, Path 3 outlines possible influences of effortful control, Path(s) 4 describe the influence of various demographic, lifestyle and health factors, and Path 5 shows change within and across individuals over time in associations between child sleep problems and weight indicators.

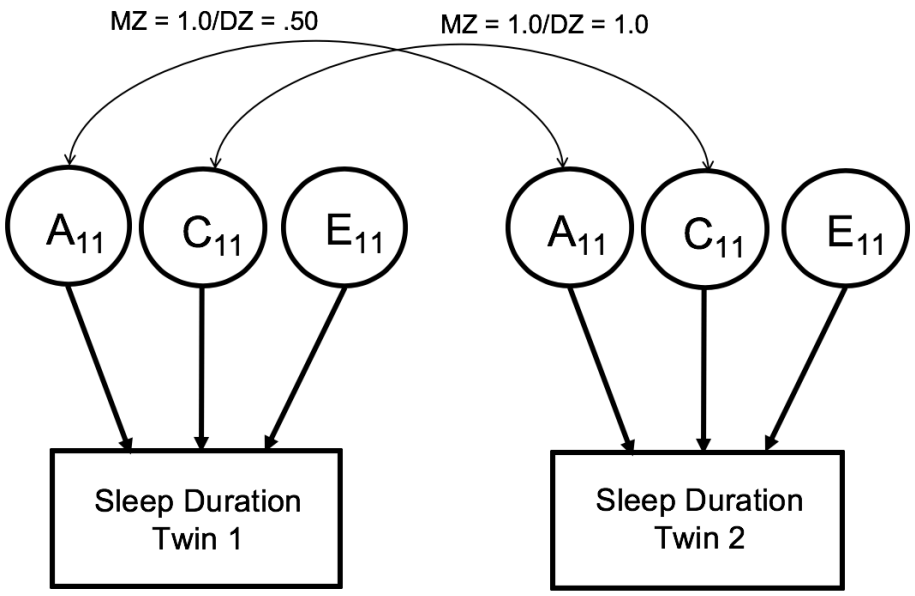

Figure 2. Example Univariate ACE Model. The model demonstrates genetic and environmental contributions on sleep duration for cotwins (Twin A on left, Twin B on right). A represents additive genetic contributions (path between MZ twins constrained to 1.0, path between DZ twins constrained to .5), C represents shared or common environmental contributions (path constrained to 1.0 for MZ and DZ twins), and E represents nonshared or unique environmental influences on a particular trait or behavior (sleep duration in this example).

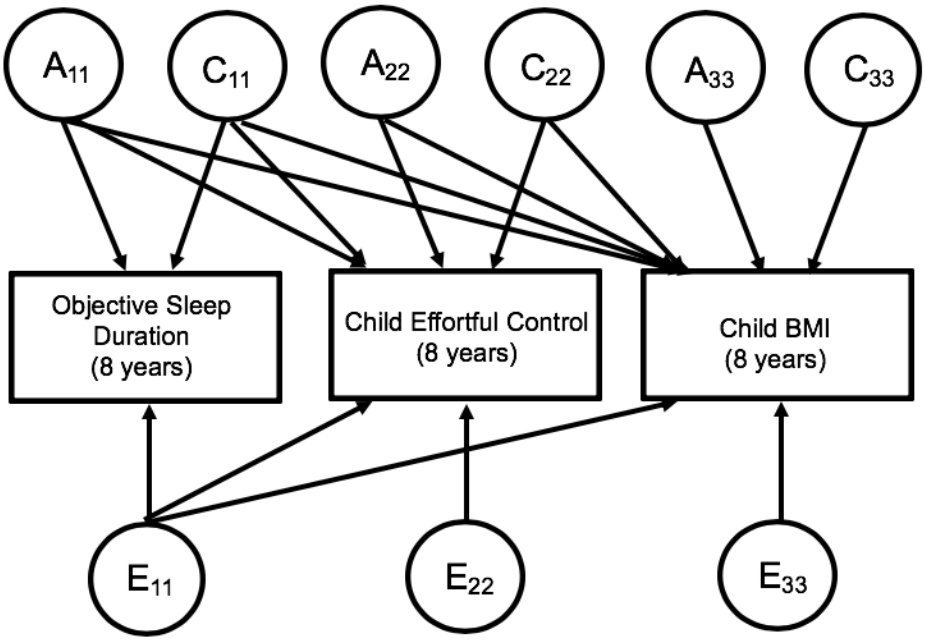

Figure 3. Example Multivariate Cholesky Decomposition Model. The model demonstrates genetic and environmental contributions on sleep duration for a single twin. A1, A2, and A3 represent possible shared additive genetic contributions between traits (after accounting for additive genetic influence in other traits), C1, C2, and C3 represent possible shared environmental contributions among traits (after accounting for common environmental influences in other traits), and E1, E2, and E3 represent possible unique environmental influences shared between traits or behaviors (after accounting for unique environmental influences in other traits).

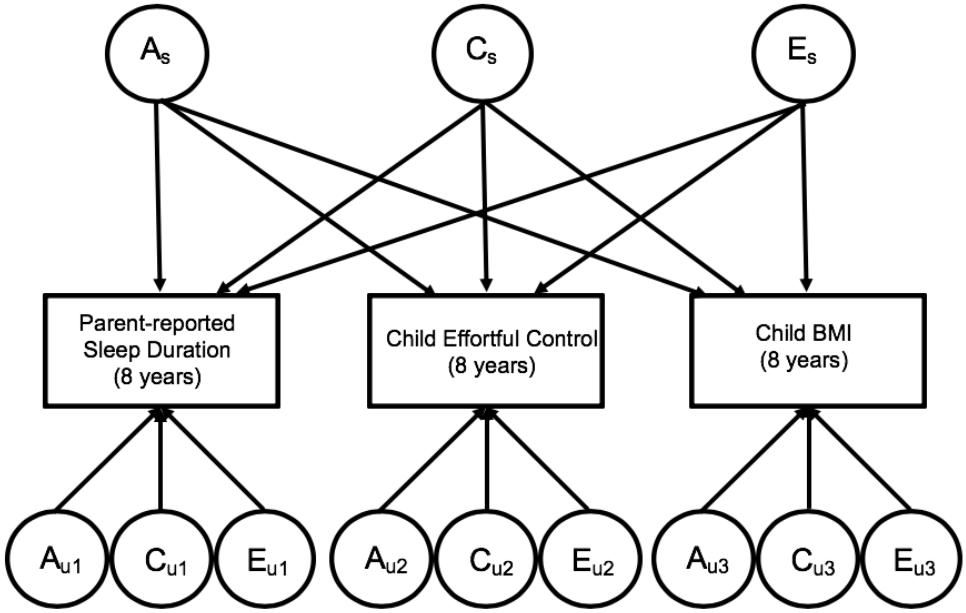

Figure 4. Example Independent Pathway Model. The model demonstrates genetic and environmental contributions on sleep duration for a single twin. As delineates shared additive genetic influences that may account for associations among traits, whereas A2 and A3 represent unique additive genetic contributions for specific traits. Cs delineates shared environmental factors common among traits or behaviors, whereas C2 and C3 represent unique shared environmental contributions to specific traits. Es delineates nonshared environmental contributions (including measurement error) that may account for associations among traits, whereas E2 and E3 represent unique nonshared environmental contributions for specific traits.

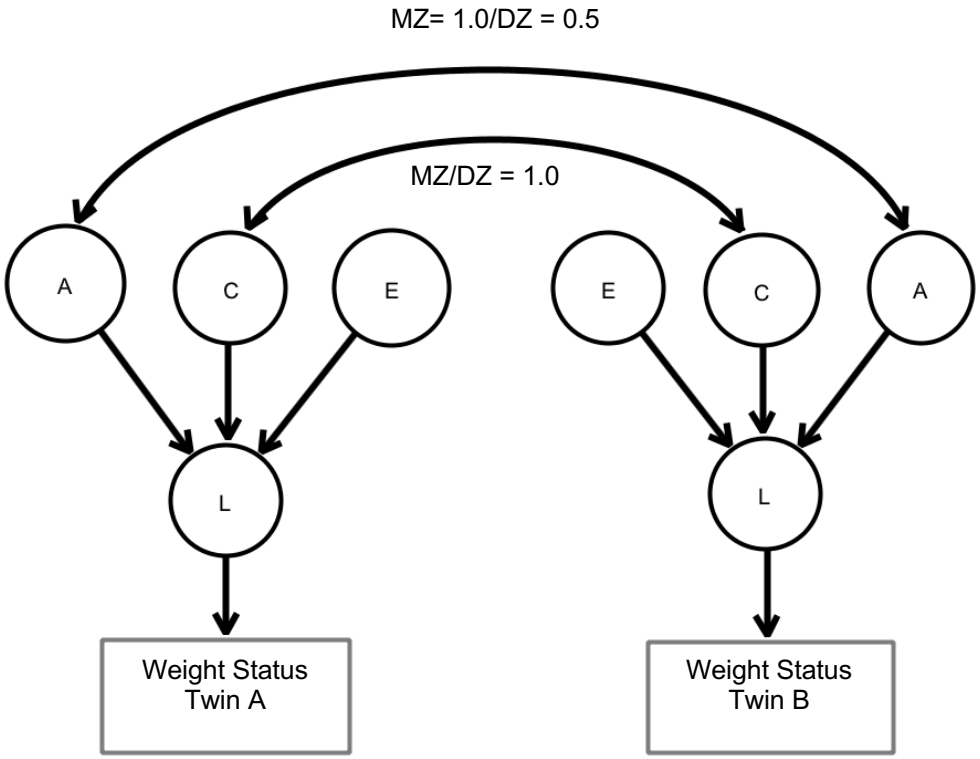

Figure 5. Example Liability Threshold Model. The model demonstrates genetic and environmental contributions on weight status for cotwins. A represents additive genetic contributions (path between MZ twins constrained to 1.0, path between DZ twins constrained to .5), C represents shared or common environmental contributions (path constrained to 1.0 for MZ and DZ twins), and E represents nonshared or unique environmental influences on a particular trait or behavior (sleep duration in this example). L represents a latent variable represents the liability or susceptibility of being classified as overweight or obese (i.e., usually represents being affected or having a disorder or diagnosis).

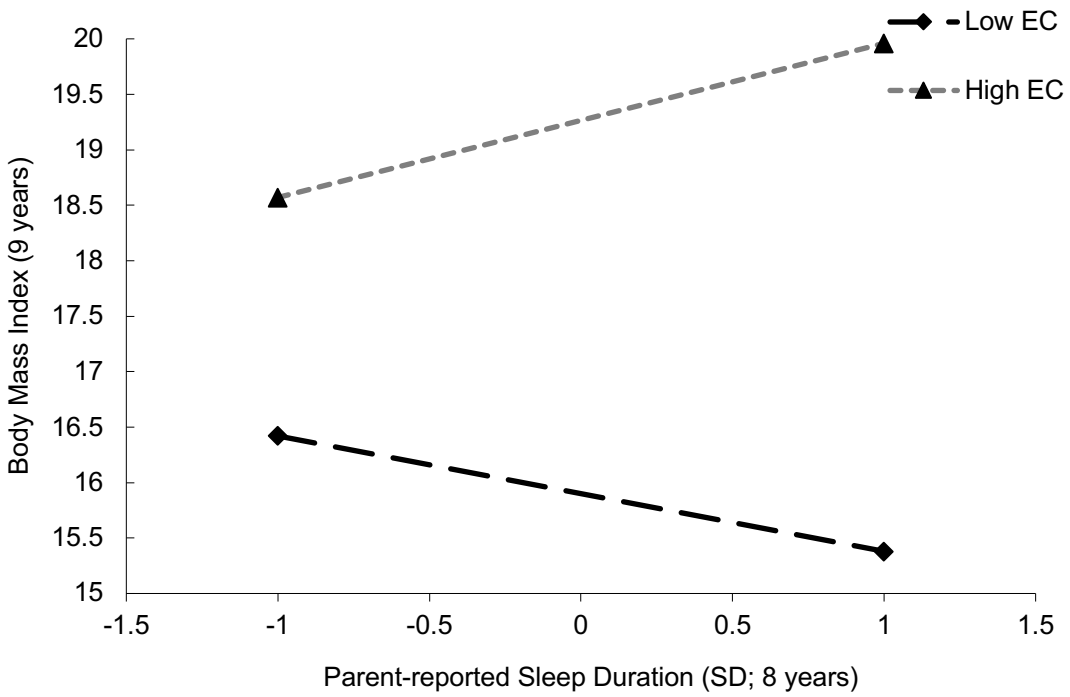

Figure 6. Simple Slopes Plot for Interaction Between Parent-reported Sleep Duration and EC at Eight Years Predicting BMI at Nine Years. The plot shows simple slope associations between parent-reported sleep duration at eight years and BMI scores at high and low levels of child EC. No simple slopes were significant (all $p > .05$.).

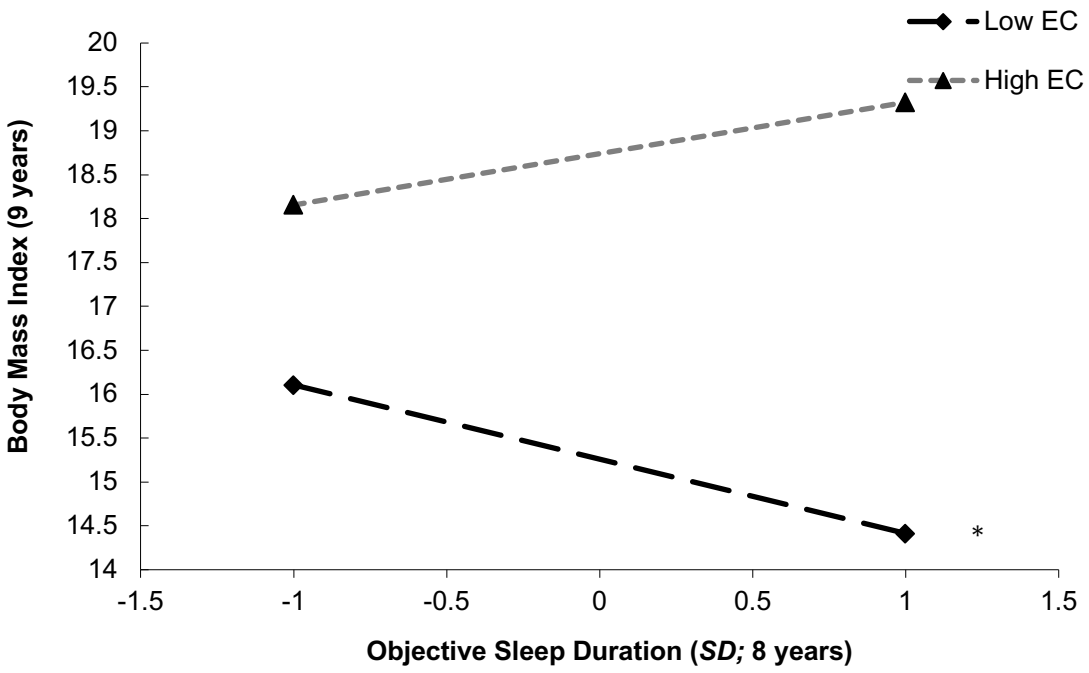

Figure 7. Simple Slopes plot for Interaction Between Objective Sleep Duration and EC at Eight Years Predicting BMI at Nine Years. The plot shows simple slope associations between sleep duration and BMI scores were significant for children with low EC (b = -.29, p < .05). Region of significance analyses indicate that simple slopes were significant for about 16.3% of children with low EC. Asterisk indicates significant simple slope at p < .05.

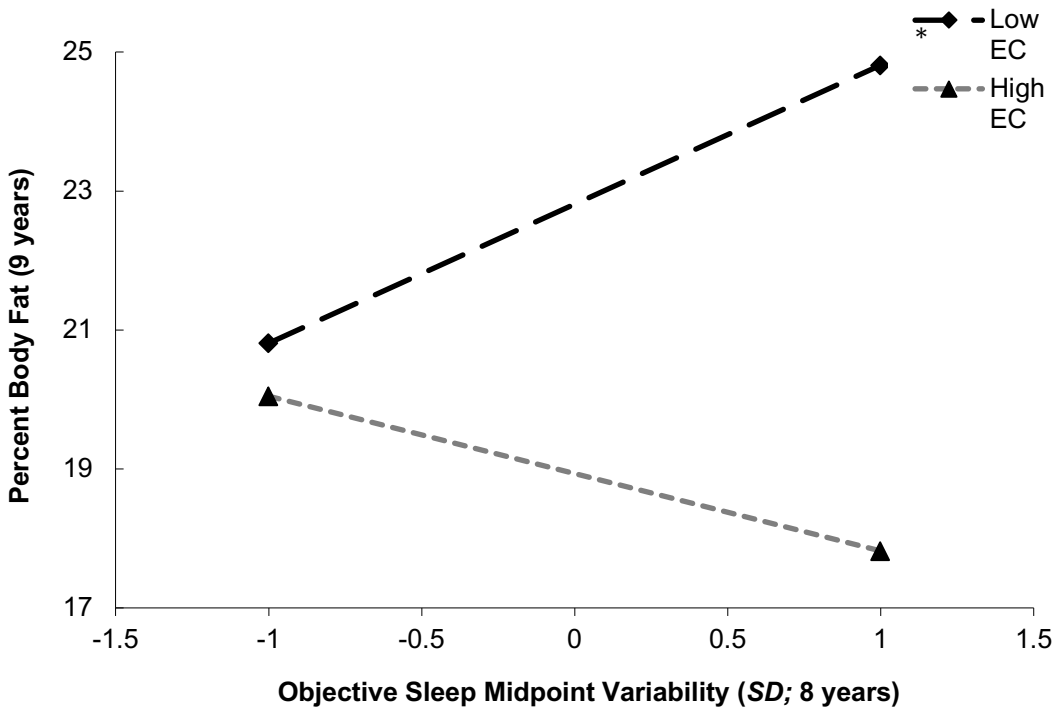

Figure 8. Simple Slopes Plot for Interaction Between Objective Sleep Midpoint Variability and EC at Eight Years Predicting Percent Body Fat at Nine Years. The plot shows simple slope associations between sleep midpoint variability and percent body fat were significant for children with low EC (b = 2.26, p = .05). Region of significance analyses indicate that simple slopes were significant for about 19% of children with low EC. Asterisk indicates significant simple slope at p = .05.