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A Life Course Approach to Racial/ethnic and Sex/Gender Disparities in Sleep Duration and Implications on Cardiometabolic Risk in Adulthood

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ABSTRACT

A Life Course Approach to Racial/ethnic and Sex/Gender Disparities in Sleep Duration and Implications on Cardiometabolic Risk in Adulthood

By Ryan Saelee

Cardiometabolic diseases remain the leading causes of morbidity and mortality in the U.S. with sleep as a significant risk factor. Race and sex disparities in sleep duration have been documented, with limited studies examining these disparities across developmental periods. Additionally, few studies have explored the impact of sleep duration across the life span on adult cardiometabolic risk factors (CRFs). Furthermore, racial disparities in adolescent sleep are well established but contributors of these disparities are largely unknown. This dissertation sought to address these gaps using data from the National Longitudinal Study of Adolescent to Adult Health.

Aim 1 examined whether neighborhood (socioeconomic disadvantage, social cohesion, and safety) and household (socioeconomic status (HSES) and single parent household) environments mediated racial differences in adolescent sleep duration. Results suggested, although HSES partially explained racial disparities, higher HSES was positively associated with short sleep duration. Future work should explore mechanisms linking varying levels of HSES to short sleep duration within each racial/ethnic group and identify buffers to reduce the detrimental impacts of HSES on sleep.

Aim 2 investigated racial/ethnic and sex/gender differences in sleep duration trajectories from adolescence to adulthood. Three trajectory types by race and sex were found: 1) Consistent Increasing Short Sleepers (i.e., increasing probability of short sleep into adulthood); 2) Late Onset Short Sleepers (i.e., no probability of short sleep in adolescence, but in adulthood); and 3) Early Onset Short Sleepers (i.e., declining probability of short sleep from adolescence into adulthood). The prevalence of Consistent Increasing Short Sleepers was highest among African American males, Late Onset Short Sleepers was highest among White females, and Early Onset Short Sleepers was the greatest among Latinx males.

Aim 3 assessed racial/ethnic and sex/gender differences in the association between sleep duration trajectories and adult CRFs. Females, regardless of race, and White males with a Consistent Increasing Short Sleeper trajectory generally had a lower prevalence of CRFs than Early or Late Onset Short Sleepers. There was a higher prevalence of dyslipidemia for African American males and elevated C-reactive protein for Latinx males among Early and Late Onset Short Sleepers.

Altogether, findings highlight the need for a life course approach and intervention during adolescence to improve adult cardiometabolic health.

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Chapter 1: Introduction

Background and Literature Review

Cardiometabolic Disease Burden and Health Risk Behaviors

Cardiometabolic diseases (e.g., cardiovascular conditions and metabolic disorders) are the leading cause of morbidity and mortality among U.S. adults and poses an enormous economic burden (1). In 2017, an estimated 868,662 deaths were due to cardiovascular disease (CVD) alone costing the U.S. an estimated $363.4 billion (2). Cardiometabolic risk factors (e.g., interrelated factors such as elevated fasting glucose, dyslipidemia, obesity, and hypertension that increases the risk for cardiometabolic diseases) are highly prevalent among U.S. adults. For instance, among U.S. adults, 39.6% are obese, 46.0% have hypertension, and 38.3% have elevated total cholesterol (2). Racial/ethnic and sex disparities in cardiometabolic health have been well established for the adult population (1). For example, diabetes is most prevalent among non-Hispanic Black and Hispanic males while the prevalence of obesity is the highest among non-Hispanic Black and Hispanic females (1). A large body of literature has explored physical activity, diet, and tobacco smoking as behavioral risk factors for cardiometabolic diseases. In particular, studies have found that physical inactivity and unhealthy diets are associated with an increased risk for obesity (3, 4), diabetes (5, 6), and hypertension (7, 8). In addition, studies indicate a higher risk of dyslipidemia (9-11) and inflammation (12-14) among those with less physical activity, unhealthy diets, and smoking. Thus, the primary focus of health behavior interventions to reduce cardiometabolic diseases and disparities have been on decreasing tobacco use, increasing physical activity, and improving diet (15, 16). An increasingly recognized health behavior that is a risk factor in the development of cardiometabolic risk factors is sleep duration (17, 18).

Mechanisms Linking Sleep Duration to Cardiometabolic Health

Systematic reviews and meta-analyses have shown that shorter sleep duration is associated with cardiometabolic risk factors such as obesity (19-21), diabetes (22, 23), elevated blood pressure (24, 25), high cholesterol (26), and inflammation (27, 28) among adults and adolescents. There are several proposed biological mechanisms in which sleep duration can influence cardiometabolic risk through metabolic dysregulation (29). Both experimental (30-32) and observation studies (33, 34) have found that shorter sleep duration is associated with decreases in leptin and increases in ghrelin release. These alterations can potentially increase hunger and cravings for high fat, sweet, starchy and salty foods subsequently increasing the risk for obesity, type-2 diabetes, hypertension, and dyslipidemia. Studies also indicate that shorter sleep duration can reduce glucose tolerance and insulin sensitivity, which are directly related with increased risk for type-2 diabetes along with lower HDL cholesterol and higher triglyceride levels (35-37). Based on experimental and observational studies, shorter sleep duration can elevate sympathetic and reduce para-sympathetic nervous system activities resulting in higher blood pressure (38). Furthermore, shorter sleep duration may cause inflammation through increases in the production of inflammatory markers such as C-reactive protein and interleukin-6 which are associated with metabolic syndrome (39). Short sleep duration may also impact cardiometabolic risk through influencing health behaviors such as physical activity and smoking. There is evidence to suggest that there may be a bidirectional relationship of sleep duration with physical inactivity and tobacco use in which shorter sleep duration can result in greater physical inactivity and smoking that can lead to shorter sleep duration. This cycle of short sleep duration, physical inactivity, and more smoking may be detrimental for cardiometabolic health.

Racial/ethnic and Sex/Gender Disparities in Sleep Duration among Adolescents and Adults

Short sleep duration is highly pervasive in the U.S. with estimates as high as 70% of adolescents (40) and 35% of adults (41) reporting short sleep duration. Significant racial

disparities have been well documented among both adolescents (42) and adults (43) with African Americans, Asians, and Hispanics generally having shorter sleep duration than non-Hispanic Whites. These disparities may be attributed to the historical and contemporary racially discriminatory practices and policies that place racial/ethnic minorities in greater social disadvantage and exclude them from accessing health-promoting resources (44). For example, racial/ethnic minorities are more likely to experience factors that negatively affect sleep such as psychosocial stressors, lower socioeconomic status, shift work, acculturation, and living in adverse physical (e.g. lower walkability, air and noise pollution) and social neighborhood environments (e.g. higher social fragmentation, lower social cohesion and safety) (45-52) .

Sex (i.e., biological characteristics such as chromosomes, hormone levels, and reproductive/sexual anatomy), and gender (i.e., a social construct to describe norms, roles, and behaviors that society associates with being male and female) differences in sleep duration have been observed in both adolescents and adults (53). Although sex and gender are distinct terms, research examining sex and gender differences in sleep duration have used these terms interchangeably as they often interact to influence health. Studies among adolescents have mixed findings with some studies suggesting shorter sleep duration among females (40, 54, 55), while others show longer sleep duration (56-58) than males. Biological pathways explaining shorter sleep duration among adolescent females could be attributed to earlier pubertal onset than males (59, 60). Although the need for sleep increases during puberty due to growth spurts, changes in biological sleep regulation (e.g. more wakefulness in the evenings and later bedtimes) combined with social factors (e.g. early school start times and increased academic pressure) may result in shorter sleep duration (61). Among females, this developmental period is also the beginning of the menstrual cycle where fluctuations of hormones (e.g. estrogen and progesterone) may result in shorter sleep duration. Due to gendered expectations, adolescent females are also more likely to have household responsibilities such as chores and caretaking (62) that may decrease the amount of time for sleep. For adolescent males, shorter sleep

duration may be influenced by poorer sleep hygiene practices (e.g. greater caffeine intake and more screen time) than adolescent females (63, 64).

Among adults, the majority of studies indicate that women have longer sleep (55, 65-68) duration while a few studies suggest shorter sleep than men (69, 70). Shorter sleep duration among adult females may also be attributed to the hormonal changes (e.g. estrogens and progesterone) that occur during pregnancy and menopause (71). In addition, gendered expectations pressure women into performing more labor including childcare and housework likely interfere with time for sleep (68). Although women tend to experience greater psychosocial stressors (e.g. gender-based discrimination, occupational stress, and financial strain) (46, 72-75) that are detrimental to sleep, they may be more likely to seek and receive social support than males (76-78) which could help buffer the negative impact of these stressors on sleep (79, 80). Research, although sparse, suggest that shorter sleep duration among males may be due to the poorer sleep hygiene and less positive attitude towards healthy sleep behaviors when compared to females (81).

The majority of studies have examined sleep disparities in relation to one social identity (e.g. race or gender), with limited studies investigating the simultaneous impact of multiple social identities (e.g. race/ethnicity and gender) on sleep. Intersectionality is a theoretical framework that explores how the convergence of multiple social identities (e.g. race, gender, social class, sexual orientation, and disability) grants individuals advantages/disadvantages that will impact their health and produce health inequities (82, 83). There is growing recognition for the need of an intersectional approach in addressing health disparities and in particular sleep disparities (43). Applying an intersectionality framework can advance sleep disparities research by more accurately documenting and identifying populations most at risk for poor sleep. This is particularly important for informing effective interventions to reduce disparities. The few studies that have examined sleep disparities through an intersectionality lens have found that sleep duration differs jointly by race/ethnicity and gender in adolescent and adult populations (57, 84-

87). For example, a study among high school students in Pennsylvania and another study among adults from the Multiethnic Study of Atherosclerosis (MESA) found that Black men and women reported the shortest sleep duration, while White women reported the longest sleep (57, 84, 85). However, these studies have primarily examined race and gender differences in sleep duration in one developmental period.

Sleep Duration across the Life Course

Research suggests that sleep duration declines as adolescents transition into adulthood that may be attributed to a combination of both biological and socio-contextual changes. Potential biological mechanisms include a circadian phase delay during pubertal development that favors an evening chronotype and the development of a sleep-wake homeostatic system that allows for more wakefulness during the daytime. Both processes could lead to progressively later bedtime from early adolescence into young adulthood (88). In conjunction with a later bedtime, adolescents encounter various socio-contextual pressures such as early school start times during adolescence, academic and work demands, growing responsibilities as they become increasingly independent from their parents, and start a family, that present additional barriers to sleep (89, 90).

Given that sleep duration can vary across the life course and sleep duration from earlier developmental periods can influence health later in life, assessing sleep duration across multiple developmental periods is critical. For example, studies have found that short sleep duration during adolescence is associated with obesity (91) and high cholesterol (92) in young adulthood. Instead of focusing on the impact of sleep at one point in time, examining the influence of sleep duration across multiple developmental periods may be more informative in predicting health outcomes. For instance, studies conducted in U.S. nationally representative samples found that consistent short sleep duration (<6 hours) from adolescence to young

adulthood was associated with a greater likelihood for obesity (93) and asthma (94) in young adulthood.

A valuable approach to exploring sleep duration across developmental periods is through characterizing sleep duration trajectories. Sleep duration trajectories can guide researchers in determining the critical periods of inadequate sleep duration, which may be useful for informing sleep interventions. However, research describing sleep duration trajectories across the life course remain limited. The few studies that have investigated sleep duration trajectories from adolescence to adulthood have found several distinct trajectories (94-96). A study conducted with a nationally representative U.S. cohort identified four trajectories types from adolescence to young adulthood (12-32 years old): increasing, decreasing, and consistently adequate (>7 hours/night), and consistently short sleep duration (< 7 hours/night). Another study of a Brazilian birth cohort found three sleep duration trajectory types from adolescence to young adulthood (11-22 years old) with variations by sex (97). In this study, both males and females had a trajectory of constant reduction in sleep duration from adolescence to young adulthood and another trajectory of decreasing sleep from age 11 to 18 that remained constant from age 18 to 22. However, for the third trajectory type, both males and females had an increase in sleep duration from age 11 to 18, but for males, the sleep remained constant from 18 to 22 years while it decreased for females during that age range. Additionally, a study that assessed self-reported sleep duration among Norwegian students at ages 13, 15, 23, and 30 years estimated various trajectory types with the most common trajectory being those with short sleep duration in early adolescence (defined as ≤8.5 hours at age 13 years, ≤8 hours at age 15 years, and ≤7 hours at age 23 and 30 years) that stabilizes into normal sleep in mid adolescence to adulthood (defined as >8.5 hours at age 13 years, >8 hours at age 15 years, and >7 hours at age 23 and 30 years) (96). These categories were found to be similar between males and females.

One of the limitations of previous research on sleep trajectories across the life course is that they did not apply an intersectionality framework to examine sleep duration trajectories from adolescence to adulthood by race and gender. It is plausible that sleep duration trajectories would vary by race and sex given the unique socio-contextual experiences that racial/ethnic minorities and women (e.g., racism and sexism) (46, 98, 99) encounter throughout life that can impact their sleep. Exploring race/ethnicity and sex/gender differences in sleep duration trajectories within and between each of these groups across the life course can help identify critical developmental periods where groups may be the most at risk for short sleep and susceptible to changes in sleep duration. This knowledge could be used to develop interventions targeting specific developmental periods to reduce sleep disparities. Furthermore, it is crucial to explore sleep duration trajectories from adolescence to adulthood in relation to adult cardiometabolic health. This will allow researchers to examine the cumulative impact of short sleep and provide greater insight on the developmental periods when sleep influences adult cardiometabolic health.

Neighborhood and Household Environment as Contributors to Racial Disparities in Adolescents

Given that adolescent sleep may predict sleep in adulthood, reducing the persistent racial disparities (42) in sleep among adolescents could alleviate sleep disparities in adulthood. Understanding the mechanisms driving these disparities can help to inform interventions to reduce these disparities in sleep duration and consequently health outcomes among adolescents. However, studies exploring these mechanisms remain sparse.

The neighborhood environment may be one of the pathways explaining racial differences in sleep duration among adolescents. Research indicates that residing in neighborhoods of higher socioeconomic disadvantage is associated with shorter sleep duration among children and adolescents (100-103). Socioeconomically disadvantaged neighborhoods often have fewer health promoting resources (e.g. recreational facilities, sidewalks, and healthy

food stores), and greater noise and air pollution that are less conducive to sleep (104-106). In addition, neighborhood socioeconomic disadvantage is correlated with worse social environments such as greater neighborhood disorder, lower safety, and less social cohesion (107). Studies have found that lack of safety and low social cohesion were associated with shorter sleep duration among adolescents (108-111). Mechanisms linking less safety and social cohesion to shorter sleep duration may be that residing in these detrimental neighborhood social environments may result in hypervigilance and psychological distress that lead to hormonal responses that interfere with sleep (112, 113). Furthermore, neighborhood environments are patterned by race/ethnicity with minorities having less access to healthier neighborhoods, consequently being more likely to live in neighborhoods of lower SES and worse social environments (114). The few studies examining the neighborhood environment as contributors to racial disparities in adolescent sleep outcomes have found that neighborhood disadvantage explained Black-White differences in sleep apnea (115) and later bedtimes (116) while one study found that neighborhood safety during childhood explained Black-White differences in sleep duration, daytime sleepiness, and time in bed during adulthood (117). Studies exploring the neighborhood environment as drivers of racial disparities in sleep duration during adolescence remain sparse.

The household environment characterized by household SES and family structure may be other important pathways underlying racial disparities in sleep among adolescents. Adolescents living in households of lower SES may have shorter sleep duration (118). Those with lower SES are more likely to have shift work, need for multiple jobs, and live in more noisy and crowded spaces interfering with the ability to establish healthy sleep habits for their children such as consistent bedtimes. Additionally, research suggests that lower household SES adolescents have greater access to media in their bedrooms and sedentary behavior which all can inhibit getting adequate sleep (119, 120). Another household environmental factor that has been linked to shorter sleep duration among adolescents is living in a single parent household.

One element of living in a single parent household that may explain shorter sleep duration is that it may reflect household SES as those living in a single parent household may have lower income than a two-parent household. Consequently, single parents may need to work multiple jobs that present barriers to monitoring and enforcing routine behaviors of their children such as earlier and consistent bedtimes that are beneficial for sleep (121-123). Furthermore, living in a single parent household may reflect instability of the home environment that could inhibit sleep (124). For instance, previous literature have found greater parent-child conflict among single parent families and an association between parent-child conflict and sleep problems among adolescents (125-127). One explanation for this association is that optimal sleep is best facilitated in an environment perceived as safe and free of threat, thus repeated exposure to feelings of instability in the home environment may increase vigilance and interfere with sleep (128). The experiences of these household level factors have also been found to vary by race/ethnicity with prior studies suggesting racial/ethnic minorities being more likely to have lower household SES and live in single parent households in comparison to non-Hispanic White adolescents (129, 130). Few studies have investigated whether racial differences in the household environment may explain racial disparities in adolescent sleep duration.

To date, studies have not examined whether household (e.g., household SES and living in a single parent household) and neighborhood (e.g. neighborhood socioeconomic disadvantage, social cohesion, and perceived safety) environments are mediators in the pathway of the association between race/ethnicity and self-reported short sleep duration.

Study Aims

Overall goal: To improve our understanding of sleep across the life course and help identify points of intervention to reduce racial/ethnic and sex disparities in sleep and improve cardiometabolic health during adulthood.

- AIM 1. Evaluate whether family (e.g. family SES and living in a single parent household) and neighborhood environments (neighborhood SES, perceived safety, and social cohesion) explain racial disparities in short sleep duration among U.S. adolescents.
  Hypothesis: Independently, adverse neighborhood and family environments will partially explain the association between race/ethnicity and short sleep duration.
- AIM 2. Describe race/ethnicity and sex/gender differences in sleep duration trajectories from adolescence to adulthood
- AIM 3. Estimate race/ethnicity and sex/gender differences in the association between sleep duration trajectories from adolescence to adulthood and adult cardiometabolic risk factors
  Hypotheses: Persistent short sleep duration from adolescence to adulthood would be associated with a greater likelihood for cardiometabolic risk factors.

Study Population

The data for this research came from the National Longitudinal Study of Adolescent to Adult Health (Add Health)(131), a nationally representative, school-based study of adolescent health behaviors and their outcomes during adulthood. Add Health uses a school-based study design where 80 high schools and 52 middle schools from the US were sampled stratified by region of country, urbanicity, school size, school type, and ethnicity. In 1994-1995, an in-school questionnaire on topics such as school extracurricular activities, friendships, and health status was administered to 90,118 students between grades 7-12. A subset of those that completed the in-school questionnaire and those that did not complete the questionnaire but were on the school roster were interviewed in their homes from 1994-1995 (Wave I; n=20,745; mean age=15.7 years). Four follow up interviews have been conducted using a subset of the original participants from the Wave I in-home interviews: Wave II in 1996 (n=14,738; mean age=16.2 years), Wave III from 2001-2002 (n=15,197; mean age=22.0 years), Wave IV in 2008

(n=15,701; mean age=28.5 years), and Wave V from 2016-2018 (n=12,300; mean age=37.0 years). For Wave V, a random subsample (n=5,381) of those from Wave I were selected and agreed to participate in collection of anthropometric and blood pressure measures. In addition, these participants provided venous blood samples that were assayed for glucose, hemoglobin A1c, lipid, and high sensitivity C-reactive protein concentrations. Aim 1 used data from Waves I and II, Aim 2 was from Waves I-IV, and Aim 3 came from Waves I-V.

Contributions to the Literature/Public Health Importance

- Prior studies have examined these neighborhood and family contextual factors as covariates and adjusted for them in their analyses assessing racial differences in sleep duration among adolescents (42). Adjusting for these factors does not allow for disentangling of whether these factors are confounders or mediators. A formal mediation analysis using structural equation modeling was conducted to statistically test whether these factors are mediators and determine whether they explain for racial differences in sleep duration among adolescents (132). Identifying these potential mediators are important for informing policies and programs to better household and neighborhood conditions which could improve sleep.
- Common methods to examine growth trajectories over time involve a single trajectory developed based on averages in the population with the assumption that this trajectory is representative of the entire population (133). However, this may mask the different trajectories that exist for those that deviate from the average trajectory, which is important, as there may be long-term impacts of short sleep duration from adolescence to adulthood on adult cardiometabolic health. Growth mixture modeling, an emerging statistical technique, allowed us to identify various sleep duration trajectories within an intersectionality framework of race and sex.
- Most studies have examined the association between sleep duration and cardiometabolic health outcomes in individual developmental periods (i.e. among children, adolescents, or

adults only), but not across the life course (134). A few studies have shown that sleep duration in early life may influence cardiometabolic health during adulthood, but these studies have not examined whether changes or stability in sleep duration across the life course have health implications. Understanding when changes in the sleep duration across the life course influences cardiometabolic risk will be useful in developing more targeted interventions at each of these developmental periods.

- The majority of prior studies have examined racial disparities in sleep duration and cardiometabolic health among African American and Whites and have limited geographic representation often including participants from a specific region in the United States (135). Aim 1 included a nationally representative sample with African American, American Indian, Asian, Latinx, and non-Hispanic White adolescents. Aims 2 and 3 included African American, Latinx, and non-Hispanic White participants. The racial/ethnic and geographic diversity will allow these study results to be more generalizable.

Chapter 2: Neighborhood and Household Environment as Contributors to Racial Disparities in Sleep Duration among U.S. Adolescents

Abstract

Racial disparities in adolescent sleep duration is well established. Investigating mechanisms underlying these disparities is important for informing interventions to reduce disparities. Racial/ethnic minority adolescents have reduced access to sleep promoting neighborhood and household environments that may contribute to the documented sleep disparities. This study examined whether neighborhood and household environments explained racial disparities in adolescent sleep duration. Participants came from Waves I and II of Add Health (n=13,019). Self-reported short sleep duration was defined as less than the recommended amount for age (<9 hours for 6-12 years, <8 hours for 13-18 years, and <7 hours for 18-64 years). Neighborhood factors included neighborhood socioeconomic disadvantage, perceived safety and social cohesion. Household factors included living in a single parent household and household socioeconomic status (HSES). Structural equation modeling was used to assess mediation of the neighborhood and household environment in the association between race/ethnicity and short sleep duration. Only HSES mediated racial disparities, explaining African American-Non Hispanic White (NHW) (11.6%), American Indian-NHW (9.9%), and Latinx-NHW (42.4%) differences. Unexpectedly, higher HSES was positively associated with short sleep duration. Future studies must examine mechanisms linking household SES to sleep and identify buffers for racial/ethnic minority adolescents against the detrimental impacts that living in a higher household SES may have on sleep.

Introduction

A large body of literature has linked short sleep duration (ranging from < 6 hours to < 8 hours) to obesity, type-2 diabetes, poorer mental health, and engagement in injury related behaviors such as tobacco use and drunk driving among adolescents (21, 23, 136, 137). Short sleep duration is highly prevalent among U.S. adolescents with as high as 60% of middle school and 70% of high school students reporting sleeping less than the recommended amount by age based on guidelines set by the American Academy of Sleep Medicine (<9 hours for age 6-12 years and <8 hours for age 13-18 years) (40). Furthermore, racial disparities in short sleep duration among adolescents has been well documented with racial/ethnic minority adolescents generally reporting shorter sleep duration than non-Hispanic Whites (42, 56, 138). The reasons for this disparity are unclear. There are limited studies that explore factors contributing to racial disparities in sleep duration among adolescents. Understanding the mechanisms driving these disparities among adolescents can help to inform interventions to reduce these disparities in sleep duration and consequently health outcomes among adolescents.

The neighborhood context may play an important role in explaining racial differences in sleep duration among adolescents. Previous studies suggests that living in neighborhoods of higher socioeconomic disadvantage is associated with shorter sleep duration among children and adolescents (100-103). Socioeconomically disadvantaged neighborhoods often have fewer health promoting resources (e.g. recreational facilities, sidewalks, and healthy food stores) which may encourage unhealthy behaviors (e.g., sedentary behaviors and unhealthy diets), and in turn, result in shorter sleep duration (45). In addition, disadvantaged neighborhoods have greater noise (e.g., traffic, police sirens, and construction work) and air pollution (e.g., $PM_{2.5}$ and nitrogen oxides) that may contribute to less sleep through arousals, inflammatory pathways, and stress responses (104-106). Moreover, neighborhood socioeconomic disadvantage is correlated with poorer social environments such as greater neighborhood disorder, less safety and lower social cohesion (107). Prior studies among adolescents have found that lack of safety and low

social cohesion were associated with shorter sleep duration (108-111). Living in these stressful neighborhood social conditions may result in hypervigilance and psychological distress that lead to physiologic hormones that interfere with sleep (112, 113). Furthermore, these neighborhood environments are patterned by race/ethnicity with racial/ethnic minorities being more likely to live in neighborhoods of lower SES and poorer social environments (114). However, studies examining whether racial differences in neighborhood environments exacerbate or mitigate racial disparities in sleep duration among adolescents are lacking.

In addition to the neighborhood SES, household-level factors such as household SES may be an important contributor to racial disparities in sleep among adolescents. Living in households of lower SES is associated with shorter sleep duration among adolescents (118). Those with lower SES are more likely to be shift workers, work multiple jobs, and living in more noisy and crowded spaces interfering with the ability to establish healthy sleep habits for their children such as consistent bedtimes. Additionally, adolescents of lower SES have less access to health care to prevent and/or treat medical conditions (e.g., asthma and obesity) that can negatively influence sleep. Household SES varies by race/ethnicity with racial/ethnic minority adolescents being more likely to have lower household SES than their non-Hispanic White peers (130). Thus, household SES may be a contributor to the documented adolescent sleep disparities. However, research examining whether household SES explains racial disparities in adolescent sleep duration remain sparse.

Another household factor that may contribute to racial disparities in adolescent sleep is living in a single parent household. There is evidence to suggest that living in a single parent household is associated with shorter sleep duration (124, 139). One component of living in a single parent household that may explain shorter sleep duration is that it could reflect household SES as those living in a single parent household may have lower income than a two-parent household. As a result, single parents may need to work multiple jobs, which could be a barrier to implementing routine behaviors of their children, such as earlier and consistent bedtimes, that

are beneficial for sleep (121-123). Furthermore, living in a single parent household may reflect instability of the home environment that could inhibit sleep (124). For instance, previous literature have found greater parent-child conflict among single parent families and an association between parent-child conflict and sleep problems among adolescents (125-127). One explanation for this association is that optimal sleep is best facilitated in an environment perceived as safe and free of threat, thus repeated exposure to feelings of instability in the home environment may increase vigilance and interfere with sleep (128). Research suggests that racial/ethnic minority adolescents may be more likely to live in single parent households in comparison to non-Hispanic White adolescents (129). Studies have not investigated whether living in a single parent household contributes to racial differences in adolescent sleep duration.

The primary purpose of this study is to simultaneously examine whether neighborhood (e.g. neighborhood socioeconomic disadvantage, safety, and social cohesion) and household contexts (e.g. household SES and single parent household) explain racial disparities in sleep duration among U.S. adolescents. Using structural equation modeling, the hypotheses being tested are: 1) racial/ethnic minorities are more likely to have shorter sleep duration than Whites, 2) living in adverse neighborhood and household environments will be positively associated with short sleep duration, and 3) racial/ethnic minorities will be more likely to live in these stressful neighborhood and household environments which will lead to greater reports of short sleep duration compared to Whites.

Methods

Study Population

The data for this study are from Waves I and II of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is an ongoing longitudinal, nationally representative, school-based study of adolescent health behaviors and their outcomes during adulthood. Details about the study design and procedures have been published elsewhere

(140). In brief, a school-based study design was used in which 80 high schools and 52 middle schools from the US were sampled and stratified by region of country, urbanicity, school size, school type, and ethnicity. In 1994-1995, an in-school questionnaire on topics such as school extracurricular activities, friendships, and health status were administered to 90,118 students between grades 7-12 with parental and student consent. A subset of those that completed the in-school questionnaire and those that did not complete the questionnaire but were on the school roster (n=20,745) were interviewed in their homes from 1994-1995 which formed Wave I (mean age: 16 years) of the study. Wave II (1996; mean age: 16 years) was conducted a year later and consists of a subset of Wave I participants (n=14,738). Sampling weights were constructed by the Add Health study team to be applied in analyses allowing for nationally representative estimates. Weights were unable to be constructed for participants selected out of the sampling frame for Add Health sub studies (141). This study included only those who participated in both Waves of data collection and had complete sample weights (n=13,568). Those who did not identify as African American, American Indian, Asian, Hispanic, or White (n=124) and were long sleepers defined as sleep duration greater than the American Academy of Sleep Medicine's (AASM) recommended amount of sleep by age (>12 hours for 6-12 years old, >0 hours for 13-18 year olds, and >9 hours for 18-64 years old) (n=405) were excluded due to their small sample sizes providing a final analytic sample of 13,019. Add Health was approved by the institutional review board of the University of North Carolina, Chapel Hill and written consent forms were obtained from adolescents and their parents.

Neighborhood Environment

Data on neighborhood contextual factors were obtained from Wave I. Participant home addresses at Wave I were geocoded using the following sources in order of priority: 1) street-segment matches from commercial geographic information system (GIS) databases; 2) global positioning system (GPS) units, when street segments were not available; 3) a ZIP+4/ZIP+2 or

a 5 digit zip centroid match when neither GIS or GPS data were available; and 4) respondent's geocoded school location (142). The home addresses were then linked to block, group, tract, and county information from the 1990 US Census and the National Archive of Criminal Justice Data for crime information (143). Those with missing geocoded data, missing source data, and unstable estimates due to small sample sizes were coded as missing (n=306). In addition, in-home interviews with adolescent participants were conducted in Wave I to collect information on neighborhood characteristics such as perceived safety and social cohesion and are further described below. Neighborhood socioeconomic disadvantage was assessed with a summary score constructed by standardizing and summing the following five census tract measures that have been used in prior Add Health research: proportions of female-headed households, individuals living below the poverty threshold, individuals receiving public assistance, adults with less than a high school education, and adults who were unemployed (144, 145). A higher summary score represented greater disadvantage and was modeled continuously. Perceived neighborhood safety was assessed based on adolescent report of yes or no to the question “Do you usually feel safe in your neighborhood?” and modeled as a binary variable. Neighborhood social cohesion was evaluated using a summary score from a prior Add Health studies based on adolescent report of yes or no to the following three items: 1)” You know most of the people in your neighborhood in the past month”; 2) “You have stopped on the street to talk with someone who lives in your neighborhood”; and 3) “People in this neighborhood look out for each other” (146, 147). Responses (yes=1, no=0) were summed to generate a score ranging from 0 to 3. The summary score was modeled as a continuous variable with a higher score representing greater social cohesion.

Household Environment

Information on family contextual measures were obtained from Wave I. During Wave I, an in-home interview was conducted with the adolescent and their parent to ascertain

information on household socio-demographics. For this study, family contextual measures included living in a single parent household and household SES. Living in a single parent household was determined by adolescent report of family structure and was modeled as a dichotomous variable (148). Household SES was assessed by a summary measure including parental education, parental occupation, and household income. The highest parental education level was used and categorized in three groups: 1) less than high school, or high school graduate or vocational school, 2) some college, or 3) college graduate or graduate education. Similarly, the highest parental occupational level was selected and classified as: 1) service industry, transportation, construction or military; 2) technical, office worker, sales; or 3) professional or manager. Household income was categorized into tertiles as low, medium, and high. A summary score was created using a weighted average of the sum of the parental education, parental occupation, and household income resulting in a score ranging from 0 to 2 with a higher score representing higher household SES.

Sleep Duration

Sleep duration data was obtained from Wave II and ascertained from adolescent participants by asking “How many hours of sleep do you usually get?” in which they were only allowed to respond in whole hours per day. Responses were categorized based on the recommended amount of sleep for age by the American Academy of Sleep Medicine (AASM): 9-12 hours for 6-12 years old, 8-10 hours for 13-18 year olds, and 7-9 hours for 18-64 years old (149, 150). Sleep was dichotomized as either short sleep duration (less than the amount recommended by age) or recommended sleep duration (within the recommended range by age). Very short sleep duration, defined as <6 hours of sleep was also modeled in sensitivity analysis.

Race/ethnicity

Participants were asked to provide a yes or no response to the question “Are you of Hispanic or Latino origin?” Following that question, participants were asked “What is your race?” with White, Black or African American, American Indian or Native American, Asian or Pacific Islander, or Other as possible choices and the ability to indicate more than one race. Based on recommendations by Add Health researchers (151), those that responded with yes to being of Hispanic or Latino origin were designated as Hispanic for their race/ethnicity and excluded from any race category that was selected. If participants selected "black or African American" and any other race, they were designated as African American for their race/ethnicity, and eliminated from the other selected race categories. The process was repeated for the remaining race categories in the following order: Asian, American Indian, and White. The race/ethnicity variable consisted of non-Hispanic African American, non-Hispanic American Indian, non-Hispanic Asian, Hispanic, and non-Hispanic White. Henceforth, the race/ethnicity groups will be referred to as African American, American Indian, Asian, Latinx, and White.

Statistical Analysis

Study sample characteristics were examined by race/ethnicity using PROC SURVEY procedures in SAS 9.4 to account for complex sampling weights (152). Structural equation modeling (SEM) was used to examine neighborhood and family contextual factors as mediators in the association between race/ethnicity and short sleep duration adjusting for self-reported sex (male vs. female) and age. An attempt was made to model the neighborhood and household environment as latent constructs mediating the association between race/ethnicity and short sleep, but did not meet the guidelines for identifiability (153). Therefore, each individual neighborhood and household measure was modeled as a mediator in the SEM. Non-Hispanic White adolescents were the reference group in all pathways involving comparisons between racial/ethnic groups. The SEMs accounted for complex sampling weights and were conducted with MPlus v8.4 using the weighted least squares means and variance estimator (WLSMV) to

obtain the standardized probit coefficients and standard errors (SE) for the total effect (TE), direct effect (DE), and indirect effect (IDE) of each pathway (154). Missing data was addressed using the default missing data procedure implemented for wslmv in MPlus (154). Covariance of error terms between each neighborhood measure and separately each family variable were included to account for potential collinearity. Modification indices were examined and covariance of error terms between the neighborhood and family characteristics with indices greater than 10 were included to improve model fit. Model fit was assessed with root mean square error of approximation (RMSEA), comparative fit index (CFI) and standardized root mean square residual (SRMR). Acceptable model fit is indicated by RMSEA<0.06, CFI>0.95, and SRMR<0.08 (155). Because the prevalence of sleeping less than 6 hours has been found to differ by race and has been associated with poorer health outcomes among adolescents such as obesity and binge drinking (91, 109, 156), sensitivity analysis was conducted in which very short sleep duration (<6 hours of sleep) was modeled as the outcome.

Results

Descriptive Analyses

Study sample characteristics by race/ethnicity are shown in Table 2-1. Overall, the mean age was 15.0 years (SE=0.11), 49.8% were female, 41.6% reported short sleep duration, and 15.6%, 2.1%, 4.0%, 12.3%, and 66.1% were African American, American Indian, Asian, Latinx, and White respectively. All characteristics significantly differed by race/ethnicity (p<.001) except sex and age. African American, American Indian and Latinx adolescents were more likely to live in neighborhoods with greater socioeconomic disadvantage compared to Asians and Whites. A smaller percentage of African American and Latinx adolescents reported living in a safe neighborhood compared to their American Indian, Asian, and White peers. On average, African Americans and White adolescents reported greater neighborhood social cohesion compared to all other racial/ethnic groups. In terms of household factors, a higher proportion of African

American, American Indian, and Latinx adolescents reported lower household SES than Asians and Whites. A higher proportion of African American and Hispanic adolescents reported living in a single parent household compared with their peers of other racial/ethnic groups. Furthermore, African American adolescents had the highest prevalence of short sleep duration (47.1%), followed by Asian (46.5%), American Indian (45.1%), Latinx (40.7%), and White (40.1%) adolescents. A similar trend was found for very short sleep duration in which the highest proportion were among African American (7.9%), followed by Asian (7.2%), American Indian (5.9%), Latinx (4.8%), and White (4.6%) adolescents.

Structural Equation Models

The simplified version of the results for each pathway without the SE and exact p-values are shown in Figure 1-1. The estimates, SE, and p-values for the total, direct, and indirect effects of the SEM comparing short sleep duration for African American, Asian, American Indian, and Latinx to White adolescents are shown in Table 1-2. Model fit statistics suggested acceptable model fit (RMSEA= .014, CFI= .996, and SRMR=.025).

Racial/Ethnic Differences in Short Sleep Duration

African American (TE: β=.055, SE=.014, p<.001) and Asian (TE: β=.026, SE=.013, p=.047) adolescents were more likely to have short sleep duration than White adolescents. There were no statistically significant Latinx-White and American Indian-White differences.

Racial/Ethnic Differences in Neighborhood and Family Contexts

African American adolescents were more likely to live in neighborhoods with greater socioeconomic disadvantage (DE: β=.455, SE=.028, p<.001), less safety (DE: β=-.186, SE=.023, p<.001), and more social cohesion (DE: β=.031, SE=.015, p=.042) than White adolescents. American Indian adolescents were more likely to live in greater neighborhood socioeconomic disadvantage (DE: β=.066, SE=.021, p=.002) and less safety (DE: β=-.039,

SE=.020, p=.049) compared with White adolescents, but there were no statistically significant differences in social cohesion. Asians were more likely to live in neighborhoods with less social cohesion than Whites (DE: β=-.084, SE=.012, p<.001), but there were no other statistically significant neighborhood differences. Compared to White adolescents, Latinx individuals reported living in neighborhoods of greater socioeconomic disadvantage (DE: β=.193, SE=.035, p<.001), lower safety (DE: β=-.167, SE=.021, p<.001), and less social cohesion (DE: β=-.067, SE=.012, p<.001).

African American adolescents were more likely to live in lower household SES (DE: β=-.180, SE=.028, p<.001) and to live in a single parent household (DE: β=.287, SE=.020, p<.001) than their White peers. American Indian adolescents were more likely to have lower household SES compared to White adolescents (DE: β=-.054, SE=.014, p<.001), but no significant differences of living in a single parent household between these two groups. There were no significant differences in household characteristics between Asian and White adolescents. Latinx adolescents lived in lower household SES (DE: β=-.222, SE=.021, p<.001) and were more likely to live in a single parent household (DE: β=.071, SE=.021, p=.001) than White adolescents.

Neighborhood and Household Characteristics on Short Sleep Duration

Higher household SES (DE: β=.069, SE=.020, p=.001) was significantly associated with greater likelihood of short sleep. There was no statistically significant association between living in a single parent household and short sleep duration or between any of the neighborhood characteristics and short sleep duration.

Mediation of Neighborhood and Family Environment

There were no significant mediation by the neighborhood environment in the association between race/ethnicity and short sleep duration (IDE: p's>.05). Household SES mediated 11.6%

of African American-White (IDE: β=-.011, SE=.004, p=.007), 9.9% of American Indian-White (IDE: β=-.003, SE=.001, p=.017), and 42.4% of Latinx-White short sleep duration differences (IDE: β=-.014, SE=.005, p=.007). That is, the difference in short sleep duration between each racial/ethnic group compared to Whites is increased by having higher household SES. However, there were no indirect effects for living in a single parent household. Furthermore, differences in short sleep duration between Asians and Whites were not significantly mediated by any of the family characteristics.

Sensitivity Analyses

In sensitivity analyses (Supplemental Table 2-1), only African American adolescents (TE: β=.088, SE=.015, p<.001) were more likely to have very short sleep duration compared to their White peers. Results for the associations between race/ethnicity with neighborhood and household characteristics were similar to that of the primary analyses. In addition, only higher household SES was associated with a lower likelihood for very short sleep duration (DE: β=-.071, SE=.035, p=.042). Also, household SES significantly mediated 30.1% of the Latinx-White differences (IDE: β=.016, SE=.008, p=.049) in very short sleep duration. No other significant indirect effects were found.

Discussion

To our knowledge, this is one of first studies to examine contributors to racial disparities in sleep duration among adolescents. This study comprised of racial groups beyond African American, Latinx, and White adolescents and included Asian and American Indian adolescents. In this study, African American, American Indian, and Asian adolescents were more likely to have short sleep duration than White their peers. Racial/ethnic minorities were more likely to live in worse neighborhood environments than White adolescents. Contrary to our hypotheses, these neighborhood characteristics were not related to short sleep duration and did not explain

the racial differences in sleep duration between racial/ethnic minority groups and White adolescents. Furthermore, results from this study show that African American, Latinx, and American Indian adolescents were more likely to live in adverse household environments than Asians and Whites. Living in a single parent household was not related to sleep duration and unexpectedly living in lower SES households was related to a lower likelihood of short sleep duration. Results further suggest that household SES partially contributed to African American-White, American Indian-White, and Latinx-White differences. That is, living in a lower household SES for these racial/ethnic minority groups decreased differences in sleep duration compared to White adolescents

Consistent with prior studies, our study found a higher proportion of short sleep duration among African American, American Indian, and Asian compared to White adolescents (40, 42). However, our results did not indicate sleep duration differences between Latinx and White adolescents. Findings from prior studies comparing sleep duration between Latinx and White adolescents have been mixed with Latinx reporting shorter or longer sleep duration than Whites (54, 157-161). These contradictory results could be due to differences in the study population. Latinx ethnicity in some studies, including the current study, were comprised of various Hispanic subgroups (e.g., Puerto Ricans, Mexican Americans, and Cuban Americans) whereas other studies assessed individual ethnic groups (e.g. Mexican Americans) (56, 157, 159, 162). The diverse sociocultural context of Hispanic subgroups (e.g., migration histories and levels of acculturation) may differentially influence their sleep and may explain the various Latinx-White disparity findings (163-165). Furthermore, the differences in the definition of short sleep duration may explain the contrasting results across studies. For instance, a study from the Youth Risk Behavioral Surveillance Survey that uses a one-item question to assess sleep found no significant Hispanic-White differences in short sleep duration defined similar to this study using the AASM guidelines. Whereas another study using data from a nationally representative sample of 8th, 10th and 12th graders with a one-item question assessment of sleep found that

Hispanics were more likely than White adolescents to have inadequate sleep which was defined as less than 7 hours. Additionally, there are variations in the measurement of sleep across studies in which some studies used objective measures of sleep duration (e.g. actigraphy and polysomnography), while others use self-reported subjective measures such as parent report or time diaries. Studies conducted with actigraphy and polysomnography generally have found no significant Hispanic-White differences in sleep duration among adolescents (56, 158, 166, 167). Inconsistent results were more common among studies with self-reported sleep measures. For instance, a study of children in Tucson, Arizona (158) using parent report found that Hispanic adolescents reported shorter sleep duration; while a study from the Child Development Supplement of the Panel Study of Income Dynamics using a time diary survey found greater sleep duration among Hispanic compared to White adolescents (161).

Similar to our findings, some studies among adolescents found null associations between neighborhood factors and sleep duration (108, 168). However, other studies among adolescents have found associations between neighborhood socioeconomic deprivation, lack of perceived safety and social fragmentation with shorter sleep duration (101-103, 108-111). The discrepancies between study results could be due to the differences in measurement of the neighborhood characteristics such as the use of different census measures for assessment of neighborhood socioeconomic disadvantage and varying scales to measure safety and social cohesion. Furthermore, the differences in results across studies could be the measurement of sleep duration in which some studies assessed sleep duration with actigraphy and/or self-reported sleep duration based on the sleep and wake time whereas our study used a one-item question with responses in whole hours.

Contrary to our hypothesis, living in a single parent household was not significantly associated with short sleep duration. A prior study among Black and White high school students by Troxel et al., found that living in a single parent household was associated with shorter actigraphy-assessed weekend sleep duration (124). In the Add Health study, sleep duration was

self-reported and not reported separately for weekday and weekend, which precluded us from examining whether the results may have differed based on weekday and weekend sleep and comparing the results between studies.

A few studies conducted outside of the U.S. (e.g., China, India, and Turkey) (169-174) have found shorter sleep duration among higher household SES adolescents citing heavier homework demands, academic pressures, and greater access to participation in extracurricular activities (e.g., sports and private tutoring) as some of the factors explaining shorter sleep duration (175-177). This may be the case for this study since Add Health is a school-based sample where academic pressures and participation in extracurricular activities may be more salient factors for adolescents in school compared to those not enrolled. Furthermore, these results are in line with prior literature indicating that racial/ethnic minority adolescents benefit less from being of higher household SES when compared to White adolescents (178-180). Prior studies suggest that racial/ethnic minority adolescents of higher SES may be more likely to live in areas and attend schools that have predominantly White students where they experience heightened discrimination (181-184). These experiences of discrimination may result in shorter sleep duration among (99, 185, 186).

Other studies conducted among U.S. adolescents contradict our findings in which lower household SES was associated with shorter sleep duration (58, 187, 188). The differences in results could be due to the use of actigraphy assessed sleep in these studies (58, 187, 188) whereas our study was limited to self-reported sleep in whole hours. In addition, there were variations in the measurement of SES in which some of these studies included indicators of SES individually (58, 187) instead of a composite measure. Furthermore, the discrepancy in findings may be explained by the differences in the age of the study samples in which previous studies included mainly younger adolescents in middle school (58, 187, 188) whereas our study consisted of mostly high school students. This may have led to differences in results because of

the greater academic and social pressures among high school compared to middle school students.

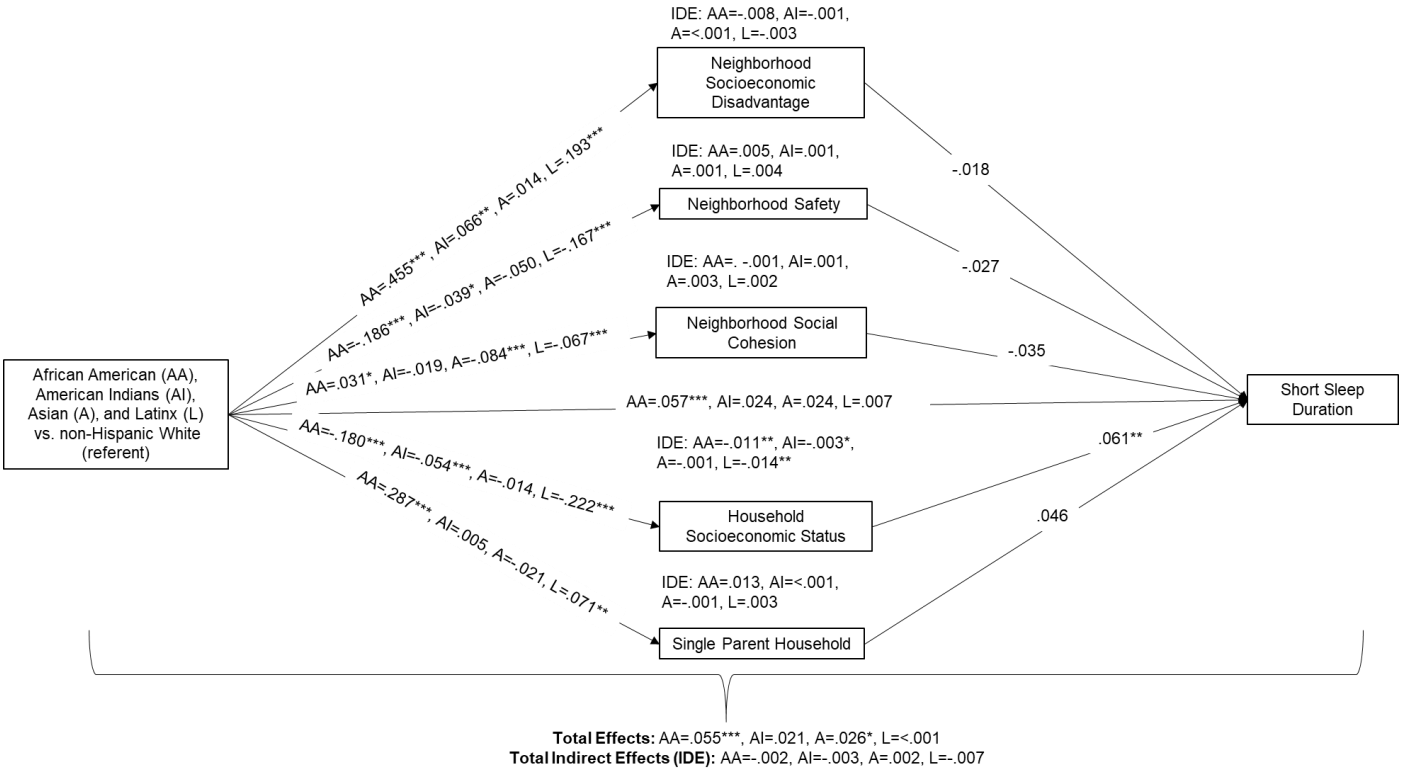
There are at least three strengths to this study. First, this is one of the first studies to formally test neighborhood and household characteristics as contributors to racial disparities in sleep duration comparing African American, Asian, Latinx, and American Indian to White adolescents. Second, the analyses simultaneously modeled both family and neighborhood characteristics, which allowed for the decomposition of specific direct and indirect effects and accounted for the potential correlation between these variables. Finally, the study cohort was large, racially/ethnically diverse, and nationally representative which allows for greater generalizability.

Despite the strengths of this study, the results should be considered in the context of the potential of three limitations. First, although the study attempted to include comprehensive measures of the neighborhood context, aggregate data may not represent individual level data and there may be other factors at the neighborhood level not captured in this study, such as measures of neighborhood disorder, crime data, and physical environment (e.g. noise, walkability, air pollution) that may contribute to racial disparities in sleep among adolescents (45). Similarly, there are other family level factors such as parental sleeping habits, traumatic life events (e.g. death in family), and caregiver stress that may explain racial differences in sleep (189-193). Second, studies comparing self-report to actigraphy assessed sleep duration among adolescents have found that they tend to overestimate their sleep and moderate correlations between measures, suggesting that there may be random misclassification of sleep duration that resulted in the null findings (57, 194). Although there were null results in the association between neighborhood contextual measures with short sleep duration and mediation by these variables, there were numerous significant pathways detected between race/ethnicity and family contextual factors with short sleep duration and mediation by these household SES. This indicates that the null findings in this study may not have been primarily due to the

measurement error of self-reported sleep duration. Finally, the adult literature has found that measurement error of self-reported sleep may vary by race/ethnicity (195, 196). Evidence of this bias in the adolescent literature is lacking, but may exist as studies have found racial differences in norms and attitudes about sleep that could influence the reporting of sleep duration (103, 197).

Given the large body of literature documenting significant racial disparities in adolescent sleep (42), understanding the contributors to these disparities is crucial for developing targeted interventions to mitigate these disparities. This study contributes to the early stages of trying to understand these pathways driving racial disparities in adolescent sleep duration and serve as a starting point for future research. Since household SES was found to be a potential pathway, it is paramount for future work to examine mechanisms linking varying levels of household SES to short sleep duration. In particular, future studies should be conducted within racial/ethnic groups to identify buffers that can reduce the harmful impacts that differing levels of household SES has on sleep. Knowledge on these mechanisms and buffers can be used to develop policies and intervention at the school level to improve sleep and reduce racial disparities in sleep.

Figure 2-1. Structural Equation Model of the Association between Race/ethnicity and Short Sleep Duration as Mediated by Neighborhood and Family Environmental Factors



Note: Paths emanating from race/ethnicity represent a comparison of each minority group (African American, Asian, American Indian and Latinx) to non-Hispanic White. All variables were modeled simultaneously and adjusted for sex and age. Total effects, indirect effects, and direct effects were estimated for all pathways. Standardized estimates are shown in each pathway. *p<.05, **p<.01, ***p<.001

Table 2-1. Sample Characteristics by Race/Ethnicity from Waves I and II of the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-96

Characteristics	Total (N=13,019)	African American (N=2,790; 15.6%)	American Indian (N=240; 2.1%)	Asian (N=928; 4.0%)	Latinx (N=2,210; 12.3%)	White (N=6,851; 66.1%)
Age, mean (SE)	15.04 (0.11)	15.19 (0.19)	14.89 (0.17)	15.23 (0.27)	15.14 (0.21)	14.98 (0.13)
Sex, Female	49.8%	51.3%	43.1%	47.6%	48.8%	50.0%
Parental Socioeconomic Status, mean (SE)	0.88 (0.03)	0.66 (0.04)	0.74 (.05)	0.94 (0.06)	0.55 (0.03)	0.99 (0.03)
Single parent household	23.6%	47.0%	19.4%	15.5%	24.9%	18.4%
Neighborhood Socioeconomic Disadvantage, mean (SE)	21.65 (0.92)	32.5 (0.96)	23.7 (2.26)	19.07 (1.34)	26.19 (1.29)	18.34 (0.98)
Neighborhood Perceived as Safe	89.8%	82.7%	88.3%	88.7%	82.8%	92.8%
Neighborhood Social Cohesion, mean (SE)	2.28 (0.02)	2.38 (0.03)	2.20 (0.07)	1.90 (0.07)	2.11 (0.03)	2.31 (0.03)
Short sleep duration	41.6%	47.1%	45.1%	46.5%	40.7%	40.0%
Very Short sleep duration (<6 hours of sleep)	5.2%	7.9%	5.9%	7.2%	4.8%	4.5%

Abbreviations: SE=Standard Error
Note: Results accounted for Add Health sampling weights; $p<0.001$ for comparison of all characteristics except for age and sex ($p's>0.05$)

Table 2-2. Results from Structural Equation Model of the Association between Race/ethnicity and Short Sleep Duration as Mediated by Neighborhood and Household Environmental Factors

Pathways (n=13,019)	β	SE	p-value
Total Effects:			
Race/ethnicity → Short Sleep Duration			
African American	.055	.014	<.001
American Indian/Alaska Native	.021	.013	.095
Asian	.026	.013	.047
Latinx	<.001	.015	.994
Direct Effects:			
Race/ethnicity → Short Sleep Duration			
African American	.057	.016	<.001
American Indian/Alaska Native	.024	.013	.058
Asian	.024	.013	.074
Latinx	.007	.015	.648
Race/ethnicity →Neighborhood Socioeconomic Disadvantage			
African American	.455	.028	<.001
American Indian/Alaska Native	.066	.021	.002
Asian	.014	.026	.583
Latinx	.193	.035	<.001
Race/ethnicity → Neighborhood Safety			
African American	-.186	.023	<.001
American Indian/Alaska Native	-.039	.020	.049
Asian	-.050	.026	.051
Latinx	-.167	.021	<.001
Race/ethnicity →Neighborhood Social Cohesion			
African American	.031	.015	.042
American Indian/Alaska Native	-.019	.010	.062
Asian	-.084	.012	<.001
Latinx	-.067	.012	<.001
Race/ethnicity → Parental Socioeconomic Status			
African American	-.180	.028	<.001
American Indian/Alaska Native	-.054	.014	<.001
Asian	-.014	.016	.397
Latinx	-.222	.021	<.001
Race/ethnicity → Single Parent Household			
African American	.287	.020	<.001
American Indian/Alaska Native	.005	.020	.819
Asian	-.021	.016	.195
Latinx	.071	.021	.001
Neighborhood Socioeconomic Disadvantage → Short Sleep Duration	-.018	.020	.373
Neighborhood Safety → Short Sleep Duration	-.027	.029	.359

Neighborhood Social Cohesion → Short Sleep Duration	-.035	.018	.058
Parental Socioeconomic Status→ Short Sleep Duration	.061	.021	.004
Single Parent Household→ Short Sleep Duration	.046	.026	.078
Indirect Effects:			
Total:			
Race/ethnicity → Short Sleep Duration			
African American	-.002	.011	.863
American Indian/Alaska Native	-.003	.002	.181
Asian	.002	.002	.337
Latinx	-.007	.006	.227
Specific:			
Race/ethnicity → Neighborhood Socioeconomic Disadvantage → Short Sleep Duration			
African American	-.008	.009	.375
American Indian/Alaska Native	-.001	.001	.397
Asian	<.001	<.001	.610
Latinx	-.003	.004	.378
Race/ethnicity → Neighborhood Safety → Short Sleep Duration			
African American	.005	.006	.371
American Indian/Alaska Native	.001	.001	.405
Asian	.001	.002	.423
Latinx	.004	.005	.366
Race/ethnicity → Neighborhood Social Cohesion → Short Sleep Duration			
African American	-.001	.001	.163
American Indian/Alaska Native	.001	.001	.236
Asian	.003	.002	.085
Latinx	.002	.001	.084
Race/ethnicity → Parental Socioeconomic Status → Short Sleep Duration			
African American	-.011	.004	.007
American Indian/Alaska Native	-.003	.001	.017
Asian	-.001	.001	.427
Latinx	-.014	.005	.007
Race/ethnicity → Single Parent Household → Short Sleep Duration			
African American	.013	.007	.071
American Indian/Alaska Native	<.001	.001	.818
Asian	-.001	.001	.327
Latinx	.003	.002	.081

Supplemental Table 2-1. Sensitivity Analysis examining the Association Between Race/ethnicity and Very Short Sleep Duration (< 6 hours) Mediated by Neighborhood and Household Environmental Factors

Pathways (n=13,019)	β	SE	p-value
Total Effects:			
Race/ethnicity → Very Short Sleep Duration			
African American	.088	.015	<.001
American Indian	.022	.021	.302
Asian	.037	.022	.085
Latinx	-.004	.023	.832
Direct Effects:			
Race/ethnicity → Very Short Sleep Duration			
African American	.064	.021	.003
American Indian	.017	.021	.408
Asian	.035	.022	.106
Latinx	-.025	.025	.313
Race/ethnicity →Neighborhood Socioeconomic Disadvantage			
African American	.397	.029	<.001
American Indian	.069	.021	.001
Asian	.008	.025	.752
Latinx	.237	.033	<.001
Race/ethnicity → Neighborhood Safety			
African American	-.186	.023	<.001
American Indian	-.039	.020	.053
Asian	-.050	.026	.051
Latinx	-.167	.021	<.001
Race/ethnicity →Neighborhood Social Cohesion			
African American	.031	.015	0.042
American Indian	-.019	.010	.063
Asian	-.084	.012	<.001
Hispanic	-.067	.012	<.001
Race/ethnicity → Parental Socioeconomic Status			
African American	-.180	.028	<.001
American Indian	-.054	.014	<.001
Asian	-.014	.016	.397
Latinx	-.222	.021	<.001
Race/ethnicity → Single Parent Household			
African American	.287	.020	<.001
American Indian	.005	.020	.786
Asian	-.021	.016	.191
Latinx	.071	.021	.001
Neighborhood Socioeconomic Disadvantage → Very Short Sleep Duration	-.016	.032	.630
Neighborhood Safety → Very Short Sleep Duration	-.020	.042	.640

Neighborhood Social Cohesion → Very Short Sleep Duration	-.019	.025	.455
Parental Socioeconomic Status→ Very Short Sleep Duration	-.071	.035	.042
Single Parent Household→ Very Short Sleep Duration	.051	.040	.205
Indirect Effects:			
Total:			
Race/ethnicity → Very Short Sleep Duration			
African American	.021	.015	.168
American Indian	.004	.003	.194
Asian	.002	.003	.380
Latinx	.020	.010	.039
Specific:			
Race/ethnicity → Neighborhood Socioeconomic Disadvantage → Very Short Sleep Duration			
African American	-.010	.013	.425
American Indian	-.001	.002	.625
Asian	<.001	<.001	.774
Latinx	-.004	.008	.627
Race/ethnicity → Neighborhood Safety → Very Short Sleep Duration			
African American	.004	.008	.625
American Indian	.001	.002	.650
Asian	.001	.002	.664
Latinx	.003	.007	.644
Race/ethnicity → Neighborhood Social Cohesion → Very Short Sleep Duration			
African American	-.001	.001	.511
American Indian	<.001	.001	.503
Asian	.002	.002	.461
Latinx	.001	.002	.454
Race/ethnicity → Parental Socioeconomic Status → Very Short Sleep Duration			
African American	.013	.007	.067
American Indian	.004	.002	.079
Asian	.001	.001	.469
Latinx	.016	.008	.049
Race/ethnicity → Single Parent Household → Very Short Sleep Duration			
African American	.015	.011	.181
American Indian	<.001	.001	.789
Asian	-.001	.001	.402
Latinx	.004	.003	.205

Abbreviations: β= Standardized probit coefficients, SE = Standard Error
Model fit: Root mean square error of approximation = 0.014, comparative fit index = 0.995, and standardized root mean square residual = 0.015
Note: Whites are the referent group in all pathways. Model adjusted for sex and age

Chapter 3: Racial/Ethnic and Sex/Gender Differences in Sleep Duration Trajectories from Adolescence to Adulthood in a U.S. National Sample

Abstract

Racial/ethnic and sex disparities in sleep duration have been documented in both adolescence and adulthood. Identifying race and sex differences in sleep duration trajectories from adolescence to adulthood can inform interventions on the developmental periods individuals are most at risk for short sleep duration. We examined racial/ethnic and sex/gender differences in self-reported sleep duration trajectories from adolescence to adulthood using data from Waves I, III, IV, and V of the National Longitudinal Study of Adolescent to Adult Health (1994-2018; n=12,593). Multi-group growth mixture modeling was used to enumerate sleep duration trajectories from adolescence to adulthood. There were three common trajectory types across race and sex groups: 1) Consistent Increasing Short Sleepers (i.e., increasing probability of short sleep into adulthood) (67.3%); 2) Late Onset Short Sleepers (i.e., no probability of short sleep duration in adolescence until adulthood) (20.2%); and 3) Early Onset Short Sleepers (i.e., declining probability of short sleep duration from adolescence into adulthood) (12.5%). The prevalence of the Consistent Increasing trajectory was highest among African American males, while Late Onset was highest among White females and Early Onset being the greatest among Latinx Males. Findings underscore the need to intervene in early adolescence to prevent short sleep duration in adulthood.

Introduction

Sleep plays a vital role in physical and cognitive development across the life span (198, 199). Short sleep duration (<8 hours 13-18 years of age and <7 hours for adults over the age of 18) (149, 150) has been implicated in a wide range of negative physical and mental health outcomes, such as obesity, diabetes, hypertension, and depression, among both adolescents and adults (24, 200-203). In the U.S., as high as 70% of adolescents and 35% of adults self-reported short sleep duration (40, 41). The transition from adolescence to adulthood is a critical period with a variety of biological and socio-contextual changes that converge leading to a decline in sleep duration into adulthood. Possible biological processes include a circadian phase delay during pubertal development that favors an evening chronotype and the development of a sleep-wake homeostatic system that allows for more wakefulness during the daytime; both processes result in a progressively later bedtime from early adolescence into young adulthood (88). In combination with a later bedtime, socio-contextual pressures such as early school start times during adolescence, academic and work demands, increasing responsibilities as they become independent from their parents, and family formation, present additional barriers to sleep (89, 90). In addition, racial/ethnic minorities and women have unique socio-contextual experiences from adolescence to adulthood that may impact their sleep duration patterns differentially across the life course that warrant further research.

Significant racial disparities in sleep duration have been well documented. African American, Asian, and Hispanic/Latino adolescents, generally report shorter sleep duration than non-Hispanic White adolescents with similar disparities found among adults (42, 43). These racial disparities in sleep duration may be attributed to the persistent racially discriminatory institutional and interpersonal policies and practices that subject racial/ethnic minorities to greater social disadvantage and restrict them from accessing opportunities and resources beneficial to health (44). For instance, research suggest that racial/ethnic minorities have greater exposure to factors detrimental to sleep such as psychosocial stressors, shift work,

lower socioeconomic status, acculturation, and adverse physical (e.g. air and noise pollution) and social neighborhood environments (e.g. lower social cohesion and safety) (45, 47-52). These socio-contextual factors are more salient among racial/ethnic minorities and could result in different sleep duration trajectories from adolescence to adulthood compared to their White peers.

Research examining sex differences in sleep duration among adolescents have mixed findings with some studies suggesting shorter sleep duration among females (40, 54, 55) while others show longer sleep duration (56-58) than males. Short sleep duration among adolescent females could be attributed to earlier pubertal onset than males. Although the need for sleep increases during puberty due to growth spurts, changes in biological sleep regulation (e.g. more wakefulness in the evenings and later bedtimes) coupled with social factors (e.g. early school start times and increased academic pressure) may result in reduced sleep duration (61). This is also the beginning of the menstrual cycle where fluctuations of hormones (e.g. estrogen and progesterone) may result in shorter sleep among females. Adolescent females are also more likely to have household responsibilities such as chores and caretaking (62) that may decrease the amount of time for sleep. On the other hand, shorter sleep duration among adolescent males may be due to their poorer sleep hygiene practices (e.g. greater caffeine intake and more screen time) than adolescent females (63, 64).

Among adults, the majority of studies indicate that women have longer sleep (55, 65-68) duration while a few studies suggest shorter sleep duration than men (69, 70). Although women tend to experience greater psychosocial stressors (e.g. gender-based discrimination, occupational stress, and financial strain) (46, 72-75) that are detrimental to sleep, they may be more likely to seek and receive social support than males (76-78) which could help buffer the negative impact of these stressors on sleep (79, 80). Research, although sparse, suggest that shorter sleep duration among males may be due to the poorer sleep hygiene and less positive attitude towards healthy sleep behaviors when compared to females (81) Shorter sleep

duration among adult females may be attributed to the hormonal changes (e.g. estrogens and progesterone) that occur during pregnancy and menopause (71). In addition, gendered expectations pressure women into performing more labor including childcare and housework interfering with time for sleep (68). The interaction of these various biological and sociocultural factors may play a role in shaping a sleep duration trajectory from adolescence to adulthood for women that may differ from men.

Studies have generally examined sleep disparities in relation to one social identity (e.g. race or gender), with few attempting to explore the impact of multiple identities (e.g. race/ethnicity and sex) on sleep. In the context of public health, intersectionality is a theoretical framework that seeks to understand how the interplay of multiple social identities (e.g. race, gender, social class, sexual orientation, and disability) provide individuals advantages/disadvantages that will influence their health and create health inequities (82). There is growing recognition for the need of an intersectional perspective in addressing health disparities and in particular sleep disparities (43). There is evidence that sleep duration differs jointly by race/ethnicity and sex in adolescent and adult populations (57, 84-87). For example, a study among high school students in Pennsylvania and another study among adults from the Multiethnic Study of Atherosclerosis (MESA) found that Black men and women reported the shortest sleep duration, while White women reported the longest (57, 84, 85). However, these studies have primarily focused on documenting race and sex differences in sleep duration in one developmental period.

Assessing sleep duration across multiple developmental periods is important given that there is evidence to suggest that sleep duration from earlier developmental periods can influence health later in life. For instance, studies have found that short sleep duration during adolescence is associated with obesity (91) and high cholesterol (92) in young adulthood. Rather than focusing on the impact of sleep at one point in time, examining the impact of sleep duration across multiple developmental periods may be more informative in predicting health

outcomes. For instance, a study among a national U.S. cohort, found that cumulative short sleep duration (< 6 hours/day) from adolescence to adulthood is associated with increased risk of obesity. (93)

An important approach to examining sleep duration across developmental periods is through characterizing sleep duration trajectories. Sleep duration trajectories could help to determine critical periods of inadequate sleep duration, which may be useful for informing sleep interventions. Yet, studies describing sleep duration trajectories across the life course remain limited. The few studies that have examined sleep duration trajectories from adolescence to adulthood have found various distinct trajectories (94-96). A study conducted using a nationally representative U.S. cohort found four trajectories that included increasing, decreasing, consistently adequate (>7 hours/night), and consistently short sleep duration (< 7 hours/night) from adolescence to young adulthood (12-32 years old). Another study of a Brazilian birth cohort found three sleep duration trajectories from adolescence to young adulthood (11-22 years old) with differences by sex (97). In this study, both males and females had a trajectory of constant reduction in sleep duration from adolescence to young adulthood and another trajectory of decreasing sleep from age 11 to 18 that remained constant from age 18 to 22. However, for the third trajectory, both males and females had an increase in sleep duration from age 11 to 18, but for males, the sleep remained constant from 18 to 22 years while it decreased for females during that age range. Furthermore, a study that assessed self-reported sleep duration among Norwegian students at ages 13, 15, 23, and 30 years found various trajectories with the most prevalent trajectory being those with short sleep duration (≤8.5 hours at age 13 years, ≤8 hours at age 15 years, and ≤7 hours at age 23 and 30 years) in early adolescence (age 13 years) that stabilizes into normal sleep (>8.5 hours at age 13 years, >8 hours at age 15 years, and >7 hours at age 23 and 30 years) in mid adolescence to adulthood (ages 15-30 years) (96). However, the studies did not apply an intersectionality framework to examine sleep duration trajectories from adolescence to adulthood by race/ethnicity and sex/gender.

Investigating race/ethnicity and sex/gender differences in sleep duration trajectories within and between each of these groups across the life course can help identify critical developmental periods where groups may be the most at risk for short sleep and susceptible to changes in sleep duration. This information could be used to develop interventions targeting specific developmental periods to reduce sleep disparities and ultimately health disparities.

To expand upon prior literature, the primary purpose of this study was to utilize an intersectionality approach to describe race/ethnicity and sex/gender differences in sleep duration trajectories from adolescence to adulthood.

Methods

Study Population and Data Source

Participants were from the National Longitudinal Study of Adolescent to Adult Health (Add Health) which is a nationally representative school-based sample with the purpose of investigating adolescent behaviors and their health in adulthood (140). There were 80 high schools and 52 middle schools selected with respect to region of country, urbanicity, school size, school type, and ethnicity to ensure representative of U.S. schools. Between 1994-1995, 90,118 participants from the participating schools were administered an in-school questionnaire related to school extracurricular activities, friendships, and health status. An in-home interview was conducted during those same years among a subset of these participants to form Wave I of the study (n=20,745). An additional four follow-up surveys were conducted on the subset of the Wave I participants: Wave II in 1996 (n=14,738; mean age=16.2 years), Wave III from 2001-2002 (n=15,197; mean age=22.0 years), Wave IV in 2008 (n=15,701; mean age=28.5 years), and Wave V from 2016-18 (n=12,300; mean age=37.0 years). A more in-depth description of the study design and procedures have been published elsewhere (140). In these analyses, those who identified as African American male, African American female, Hispanic male, Hispanic female, White male, White female from Wave I were included (n=18,649). Those with missing sleep duration data in Waves I, III, IV, and V were excluded (n=51). Long sleepers

(defined as >12 hour for 6-12 years old, >10 hours for 13-18 years old, >9 hours for over 18 years old) in any of the Waves were excluded (n=6,020) instead of being grouped with short sleepers or those with the recommended amount of sleep. The reason is that the mechanisms linking long sleep to health may differ from those linking short sleep to health (204) and the sample size for long sleepers in Wave I (n=556) and V (n=480) was small. The final analytic sample was 12,593 participants. Characteristics that included age, sex, race/ethnicity, parental education, participant education level, depressive symptoms, body mass index, and self-reported health were compared between those included and those excluded (See Supplemental Table 3-1). Those excluded were more likely to be female, have higher parental education and education level, report on average greater depressive symptoms, and more likely to report having fair or poor health across all Waves. Supplemental analyses including long sleepers were conducted and the results are shown in Supplemental Figure 3-1. The original Add Health study was approved by the institutional review board of the University of North Carolina, Chapel Hill and written consent forms were obtained from adolescents and their parents. This current analysis was approved by the institutional review board of Emory University.

Study Measures

Sleep Duration

Measurements of sleep duration varied by Waves. In Waves I and V, participants were asked to respond in whole hours to the question, “How many hours of sleep do you usually get?” In Waves III and IV, sleep duration was assessed by the following four items: 1) “On days when you go to work, school, or similar activities, what time do you usually wake up?”; 2) “What time do you usually go to sleep the night (or day) before?”; 3) “On days you don’t have to get up at a certain time, what time do you usually get up?”; 4) “On those days, what time do you usually go to sleep the night or day before?”. The first two questions are used to measure sleep hours during the weekday while questions 3 and 4 are for weekend sleep hours. Total sleep hours per day was calculated by a weighted average based on prior studies conducted on sleep

using the Add Health dataset, in which sleep hours during the weekday is multiplied by 5/7 and added to sleep hours during the weekend multiplied by 2/7 (93, 156). The recommended amount of sleep by age from the American Academy of Sleep Medicine (AASM) is 9-12 hours for 6-12 years old, 8-10 hours for 13-17 years old, and 7-9 hours for 18 or older (149, 150) and was categorized in analyses as short sleep duration (less than recommended by AASM) vs. healthy sleep duration (within recommended by AASM).

Race/ethnicity and Sex/Gender

Race/ethnicity categories were constructed based on recommendations by the Add Health research team (151). Respondents were first asked whether they were of Hispanic or Latino origin. A separate question asked respondents to indicate their race with White, Black or African American, American Indian or Native American, Asian or Pacific Islander, or Other as possible answers and the ability to select multiple racial groups. Those that indicated being Hispanic or Latino origin were categorized as “Hispanic” for their race/ethnicity. If participants did not indicate they were of Hispanic or Latino origin and selected "black or African American" as one of their racial identities, they were categorized as African American for their race/ethnicity, and their other selected racial categories were disregarded. This was repeated for the remaining racial groups in the following order: Asian, Native American, Other, and non-Hispanic White. Hereafter, Hispanic will be referred to as Latinx and non-Hispanic White will be referred to as White. Sex was assessed by asking whether respondents identified male or female. Add Health did not include a separate measure for gender, thus sex in this study will be used also be used to represent gender. The combined race/ethnicity and sex/gender categories for the analyses included the following six groups: African American male, African American female, Latinx male, Latinx female, White male, White female. Asian, Native American and Other race and sex combinations were excluded due to inadequate sample size for analysis.

Statistical Analysis

The distribution of participant characteristics were examined by race/ethnicity and sex using chi-square tests and ANOVA. There are several approaches to modeling longitudinal data with growth curve modeling being the most common. Unlike conventional growth curve modeling approaches that assume individuals come from a single population with common parameters (e.g., slopes, intercepts, and error variances) and that a single growth trajectory is representative of an entire population, growth mixture model (GMM) is a person-centered approach that relaxes these assumptions and allows for different groups of individual growth trajectories to vary around different parameters (133). Given our interest to apply an intersectionality framework and the poor model fit of single GMMs for this sample, multi-group GMMs were conducted to enumerate the sleep duration trajectory classes across the developmental periods for each race/ethnicity and sex/gender group. Models examined changes in probability of short sleep duration across developmental periods. Developmental periods were determined based on prior literature and included early adolescence (11-14 years), adolescence (15-19 years), emerging adulthood (20-24 years), young adulthood (25-34 years), and adulthood (35-44 years) (205-207). Since Waves I and II data were collected only a year apart, some participants had two sleep duration data points in the early adolescence or adolescence developmental period. Data in GMM need to be time-structured and to-date no options are available to account for clustering within a time frame (208). Thus, both data points were unable to be included in the analyses. To address this issue, GMM models including data from Wave I, III, IV, and V were conducted and compared to models including data from Waves II, III, IV, and V. Models including data from Wave I had better model fit and interpretability of classes than with data from Wave II. Therefore, results from analyses including Wave I are discussed below. Two to four class models were estimated and compared using the Akaike information criterion (AIC), Bayesian information criterion (BIC), and entropy. The best fitting model was determined based on lower AIC and BIC, higher entropy, and interpretability of classes (209). Growth mixture models were conducted using MPlus v8.4 with a maximum

likelihood estimator and expectation maximization algorithm (154). Missing data was addressed with full information maximum likelihood. Descriptive analyses were conducted in SAS v.9.4.

Results

Descriptive Analyses

Across developmental periods, the mean age was 13.4 years (SD=0.7) in early adolescence, 16.7 years (SD=1.3) in adolescence, 22.1 years (SD=1.3) in emerging adulthood, 28.7 years (SD=1.9) in young adulthood, and 38.2 years (SD=1.8) in adulthood (Table 3-1). The prevalence of short sleep duration varied by developmental periods with 29.7% in early adolescence, 46.8% in adolescence, 31.4% emerging adulthood, 30.5% in young adulthood, and 46.1% in adulthood. Short sleep duration was most prevalent among African American males across all developmental periods except early adolescence where African American females were more likely to have short sleep duration.

Multi-group Growth Mixture Models by Intersectionality Dimensions of Race/Ethnicity and Sex

The three-class model was the best performing model with the lowest AIC and BIC and the highest entropy (Supplemental Table 3-2). Thus, this resulted in 18 trajectories, i.e., three trajectories for each of the six race/ethnicity and sex intersectionality groups (Figure 3-1, Figure 3-2, and Supplemental Table 3-3). Through visual inspection, we found that the 18 trajectories could be grouped into three common sleep duration trajectory types as the three trajectories for each intersectionality group varied but were qualitatively comparable. The most prevalent trajectory (67.3%) type was labeled “Consistent Increasing Short Sleepers” as the sleep pattern began in early adolescence at a mid-level probability (ranging from 0.44-0.46) of short sleep duration and consistently increased into adulthood (probability ranging from 0.47-0.67). The second most prevalent trajectory type was labeled “Late Onset Short Sleepers” as this group represented those who had zero probability of short sleep duration from adolescence to young adulthood, but had a probability of short sleep duration in adulthood (20.2%). The least common

trajectory type was labeled as “Early Onset Short Sleepers” (12.5%) and included those with a high probability for short sleep duration during early adolescence only as short sleep declines to a low probability in the adult developmental periods.

Although these 18 trajectories could be grouped into three common trajectory types, within an intersectionality framework, there were notable differences between the race/ethnicity and sex groups across and within each of these trajectory types (Figure 3-1 and 3-2). First, the distribution of the trajectory types varied by race/ethnicity and sex groups (Table 3-2). Latinx males were the most likely to have an Early Onset Short Sleeper trajectory type. The Consistent Increasing Short Sleeper trajectory type was the most prevalent among African American males while the prevalence of Late Onset Short Sleeper trajectory type was the highest among White females. Second, the trajectories themselves varied across intersectionality groups. These differences are described below by race/ethnicity and sex/gender groups.

African American Females

Among African American females, the Consistent Increasing Short Sleeper trajectory, the probability of short sleep duration begins at 0.48 in early adolescence and ends at a probability of 0.60 with a consistent 0.03 probability increase across each of the developmental periods. The Early Onset Short Sleeper trajectory was characterized by a high probability (1.00) of short sleep duration in early adolescence and adolescence, but a sharp decline to zero probability in emerging adulthood that was maintained into adulthood. In the Late Onset Short Sleeper trajectory, there was zero probability of short sleep duration from early adolescence to young adulthood, but a mid-level probability (0.42) in adulthood.

African American Males

In the Consistent Increasing Short Sleeper trajectory, the probability of short sleep duration in early adolescence was 0.45 and grew to 0.67 in adulthood with an approximate average 0.05 increase in probability across each of the developmental periods. Similar to African American females, the Early Onset Short Sleeper trajectory for African American males

begin with a high probability of short sleep duration during early adolescence (1.00) and adolescence (0.93), but zero probability across all adult developmental periods. For the Late Onset Short Sleeper trajectory, there was zero probability of short sleep duration across all developmental periods except adulthood with a probability of 0.33.

Latinx Females

The Consistent Increasing Short Sleeper trajectory begins with a 0.46 probability of short sleep duration in early adolescence and consistently increases by 0.01 across developmental periods to 0.45 in adulthood. For the Early Onset Short Sleeper trajectory, there was a similarly high probability (1.00) of short sleep duration in early adolescence and adolescence, but declines to a low probability (0.06) in emerging adulthood that further decreases to zero in both young adulthood and adulthood. In the Late Onset Short Sleeper trajectory, the probability of short sleep duration is zero from early adolescence to young adulthood and increases to 0.29 in adulthood.

Latinx Males

The trajectories were the most unique among Latinx males. The Consistent Increasing Short Sleeper trajectory was similar to African American males with a 0.44 probability of short sleep duration in early adolescence that increase on average by 0.06 in probability across each subsequent developmental period resulting in a 0.67 probability in adulthood. However, for the Early Onset Short Sleeper trajectory, it begins with a mid-level probability (0.41) of short sleep duration in early adolescence and gradually declines to a low probability in adulthood (0.11). This is different from the other race/ethnicity and sex/gender groups where the probability sharply declines from a high probability of short sleep duration in adolescence to zero probability in adulthood. In addition, the Late Onset Short Sleeper trajectory had a zero probability of short sleep duration from early adolescence to young adulthood, but surges to 0.88 in adulthood. Among this trajectory type, Latinx males had by far the highest probability in adulthood compared to the other race/ethnicity and sex/gender groups.

White Females

White females had the lowest growth in probability for short sleep duration across the developmental periods for the Consistent Increasing trajectory. In this trajectory, the probability of short sleep duration (0.45) remains constant from early adolescence to adolescence and increases in emerging adulthood (0.46) which remains constant into adulthood. Similar to Latinx females and White males, the Early Onset Short Sleeper trajectory among White females begins with the same high probability (1.0) of short sleep duration in both early adolescence and adolescence with drastic decline to a low probability in emerging adulthood (0.06) and zero probability in young adulthood and adulthood. For the Late Onset Short Sleeper trajectory, there was a zero probability of short sleep duration starting in early adolescence to young adulthood with an increase to a 0.33 probability in adulthood.

White Males

For the Consistently Increasing Short Sleeper trajectory, the early adolescence probability of short sleep duration was 0.37 that increases to 0.52 in adulthood with an average 0.04 increase in the probability in each subsequent developmental periods. The Early Onset Short Sleeper trajectory among White males was comparable to Latinx and White females in which there is a high probability (1.0) of short sleep duration in both adolescent periods with a sharp decline to a low probability (0.06) in emerging adulthood that decreases to zero in the other adult developmental periods. In the Late Onset Short Sleeper trajectory, there was no probability of short sleep duration across developmental periods until adulthood with a 0.52 probability.

Discussion

Our study found three common sleep duration trajectory types from adolescence to adulthood in a nationally representative sample accounting for race and sex. These three sleep duration trajectory types included Early Onset Short Sleepers who had declining probability of

short sleep from adolescence to adulthood, Consistent Increasing Short Sleepers with increasing probability of short sleep duration from adolescence to adulthood, and Late Onset Short Sleepers with no probability of short sleep duration until adulthood. The distribution of these trajectory types differed across race/ethnicity and sex groups. Furthermore, there were observable differences across race and sex groups within each of these trajectory types.

The most prevalent sleep trajectory type in the overall sample were Consistent Increasing Short Sleepers. This suggests that for many individuals, the continual increase would eventually lead to consistent high risk for short sleep in older adulthood. Within each racial/ethnic group, the trajectory was more prevalent among males than females except for Latinx participants. The higher prevalence among males in general may be explained by the poorer sleep hygiene among males observed in adolescent and adult studies (63, 64, 81). Between racial/ethnic groups, this trajectory was the most prevalent among African American participants with the highest prevalence particularly among African American males. This is in line with studies that have shown African American males were the most likely to have the shortest average sleep duration in both adolescent and adult populations (57, 85, 87, 156). This may be due to stress related to the persistent discrimination African American males' experience (e.g. police brutality and criminal profiling) throughout the life course (210-212). In addition, various sociocultural factors (e.g. masculine socialization, stigma, and lack of access to healthcare and economic resources) may prevent African American males from seeking and receiving support to help mitigate the harmful psychological effects of discrimination (213-215). The high prevalence of this sleep trajectory among African American males may contribute to the significant health disparities (216) among this group across the life span.

The second most common trajectory was Late Onset Short Sleepers. A potential explanation for the healthy sleep duration from early adolescence to young adulthood is that these individuals may be in environments (e.g. higher household SES, greater neighborhood safety and social cohesion) that are more optimal for sleep (45, 118, 217). In adulthood, the

increase in probability of short sleep duration may be explained by the growing responsibilities and demands such as work and family formation that reduces the amount of time for sleep (90). Across all racial/ethnic groups, females were more likely to have a Late Onset Short Sleeper trajectory with the greatest prevalence among White females. This may be attributed to family formation and gendered expectations that pressure them into being the primary caretaker of the family allocating less time for sleep (68).

The Early Onset Short Sleepers were the least common trajectory overall. The existence of this trajectory could be due to the socio-contextual changes that occur as people transition from early adolescence into later adolescence. These changes may include earlier school start times, academic pressure, and greater social commitments due to an expanding social network, that all could prevent adolescents from obtaining adequate sleep duration (89, 218). As these individuals transition into adulthood and become free from the early school start times, they may enter the workforce or attend college where there may have greater flexibility in tailoring their sleep schedule (219-221). Across race/ethnicity, this trajectory was generally more prevalent among females with the exception of Latinx individuals. The higher occurrence of this trajectory among females may be due to being pressured into more responsibilities than males during early adolescence (e.g., caretaking and chores) that reduce the amount of time for sleep (62). However, as these individuals transition into adulthood and seek greater independence and autonomy, they often enter the workforce or attend postsecondary education allowing them to relinquish some of these responsibilities (90, 222).

Within the trajectory types, Latinx Males had the most distinct trajectories compared to the other race and sex groups. In the Late Onset trajectory, Latinx males had, by far, the highest probability of short sleep duration in adulthood. Also, the Early Onset trajectory for Latinx males differed from other groups with a lower probability of short sleep duration in early adolescence and a gradual decline in the probability of short sleep duration from adolescence into adulthood instead of a sharp decline. These findings for Latinx males could be due to the aggregating of

Latinx subgroups that included Mexican Americans, Puerto Ricans, Central/South Americans, and Cubans, into a single classification. By doing so, the diverse sociocultural context of Hispanic subgroups (e.g. levels of acculturation and nativity) were not accounted for that could potentially have influenced their sleep duration throughout adolescence to adulthood (50, 163, 164, 223, 224).

There are at least three strengths to this study. First, this is one of few studies to examine sleep duration trajectories from adolescence to adulthood including an older adult assessment time point compared to prior trajectory studies (94, 95). Second, to our knowledge, this is one of the first studies to use an intersectionality approach to examine race/ethnicity and sex differences of sleep trajectories. Finally, this study include a large diverse nationally representative cohort, which increases the generalizability of the results.

These findings should be interpreted within the context of the limitations in this study. One limitation of this study is the potential measurement error of sleep duration due to self-reporting. Prior studies have found that participants tend to overestimate their sleep duration when compared to objective measures such as actigraphy among both adolescents and adults (57, 194-196, 225-227). In the adult literature, there is evidence that measurement error of sleep duration may vary by race/ethnicity and sex/gender (195, 196, 227). For instance, in a study with MESA data comparing self-reported to actigraphy assessed weekend sleep duration ,Whites were more likely to overestimate their sleep duration than Blacks and Black women were more likely to overestimate their sleep than Black men (196). It is currently unclear whether these race and sex differences in measurement error exists among adolescents. Race and sex differences in the overestimation of sleep in both adolescence and adulthood may have led race and sex variations in the underestimation of the Consistent Increasing Short Sleeper trajectory type. Another limitation was that sleep duration was ascertained in whole hours in Waves I and V and not exact time. Furthermore, sleep duration was measured differently in Waves I and V by asking a single question of “How many hours of sleep do you usually get?”

whereas Waves III and IV weekday and weekend sleep duration. The variation in measurement prevented this study from examining whether the results would have differed by weekend and weekday sleep duration. This may be important given that adolescents and adults generally sleep less during the weekday and more during the weekend (88, 228, 229). Since weekday sleep duration is weighted higher than weekend sleep duration in calculating the average sleep duration per day, any changes in weekday sleep duration would more likely influence the trajectories than weekend sleep duration.

Future research should replicate this study with objective measures of sleep duration such as actigraphy and examine the trajectories of other sleep dimensions beyond duration. While beyond the scope of this study, future studies should examine the various factors that influence sleep are socially patterned by race/ethnicity and sex/gender (e.g. neighborhood and household environments, and depressive symptoms) (45, 230, 231), additional research is needed to examine the impact of these factors on race and gender differences in the development of sleep trajectories. Furthermore, future work should investigate these trajectories in relation to health outcomes, given that insufficient sleep duration across the life course is associated with poor health outcomes. These future studies will be important in developing more informed strategies to mitigate racial/ethnic and sex/gender health disparities.

The findings from this study have important implications for public health professionals and researchers. The results underscore the need to examine sleep within an intersectionality framework in order to advance sleep disparities research. The consequences of examining a single social identity may result in inaccurate documentation of the sleep disparities and subsequently ineffective strategies to reduce these disparities. Overall, this study highlights the need to intervene in both adolescence and adulthood. Intervening as early as adolescence to prevent short sleep in adulthood would be particularly important given that a high proportion of the sample had an increasing likelihood of short sleep duration from early adolescence to adulthood. It is also important to note that there was no trajectory of consistent healthy sleep

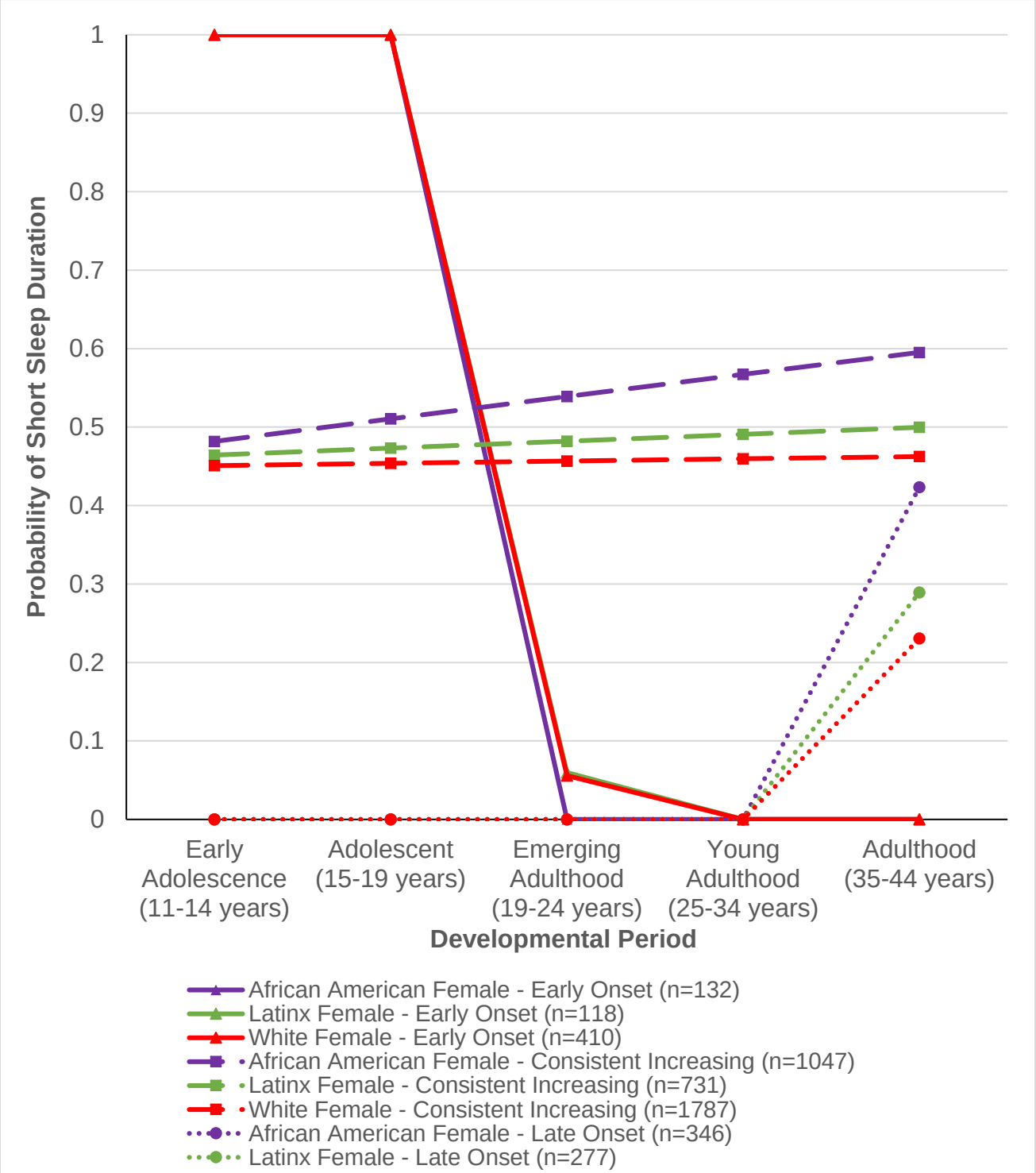
duration from adolescence to adulthood. This indicates that individuals would need some type of intervention at either adolescence or adulthood. Implementing targeted strategies for specific race/ethnicity and sex/gender groups at particular development periods can help improve sleep, and reduce racial/ethnic and sex/gender disparities in sleep.

Table 3-1. Participant Characteristics by Race/Ethnicity and Sex/Gender from Waves I, III, IV, and V of the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-2018

Characteristics	Total (N=12,593)		African American Female (N=1,525)		African American Male (N=1,573)		Latinx Female (N=1,126)		Latinx Male (N=1,302)		White Female (N=3,236)		White Male (N=3,831)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%
Developmental Periods														
Early Adolescence	3,283	26.1	443	29.0	432	27.5	236	21.0	253	19.4	955	29.5	964	25.1
Adolescence	10,111	80.3	1,201	78.8	1,221	77.6	934	82.9	1,112	85.4	2,561	79.1	3,073	80.2
Emerging Adulthood	7,018	55.7	852	55.7	780	49.6	578	51.3	670	51.5	1,951	60.3	2,187	57.1
Young Adulthood	8,911	70.8	1,132	74.2	934	59.4	762	67.7	806	61.9	2,476	76.5	2,741	71.5
Adulthood	6,938	55.1	880	57.7	613	39.0	614	54.5	533	40.9	2,142	66.2	2,146	56.0
Short Sleep Duration														
Early Adolescence*	975	29.7	174	39.3	158	36.6	72	30.5	76	30.0	278	29.1	217	22.5
Adolescence*	4,728	46.8	612	51.0	625	51.2	476	50.5	485	43.6	1,222	47.7	1,308	42.6
Emerging Adulthood*	2,202	31.4	270	31.7	309	39.6	159	27.5	231	34.5	501	25.7	732	33.5
Young Adulthood*	2,721	30.5	380	33.6	471	47.4	190	24.9	275	34.1	548	22.1	857	31.3
Adulthood*	3,197	46.1	522	59.3	387	63.1	286	46.6	256	48.0	844	39.2	902	42.0
Age in years[a]														
Early Adolescence	13.4 (0.7)		13.3 (0.7)		13.4 (0.7)		13.4 (0.7)		13.4 (0.7)		13.4 (0.7)		13.4 (0.7)	
Adolescence	16.7 (1.3)		16.7 (1.3)		16.7 (1.3)		16.7 (1.2)		16.8 (1.2)		16.7 (1.3)		16.6 (1.3)	
Emerging Adulthood	22.1 (1.3)		22.1 (1.3)		22.1 (1.3)		22.2 (1.3)		22.4 (1.3)		22.1 (1.4)		22.1 (1.3)	
Young Adulthood	28.7 (1.9)		28.6 (1.9)		28.7 (1.9)		29.0 (1.8)		29.1 (1.8)		28.6 (1.9)		28.7 (1.8)	
Adulthood	38.2 (1.8)		38.1 (1.8)		38.3 (1.8)		38.5 (1.7)		38.6 (1.8)		37.9 (1.8)		38.2 (1.8)	

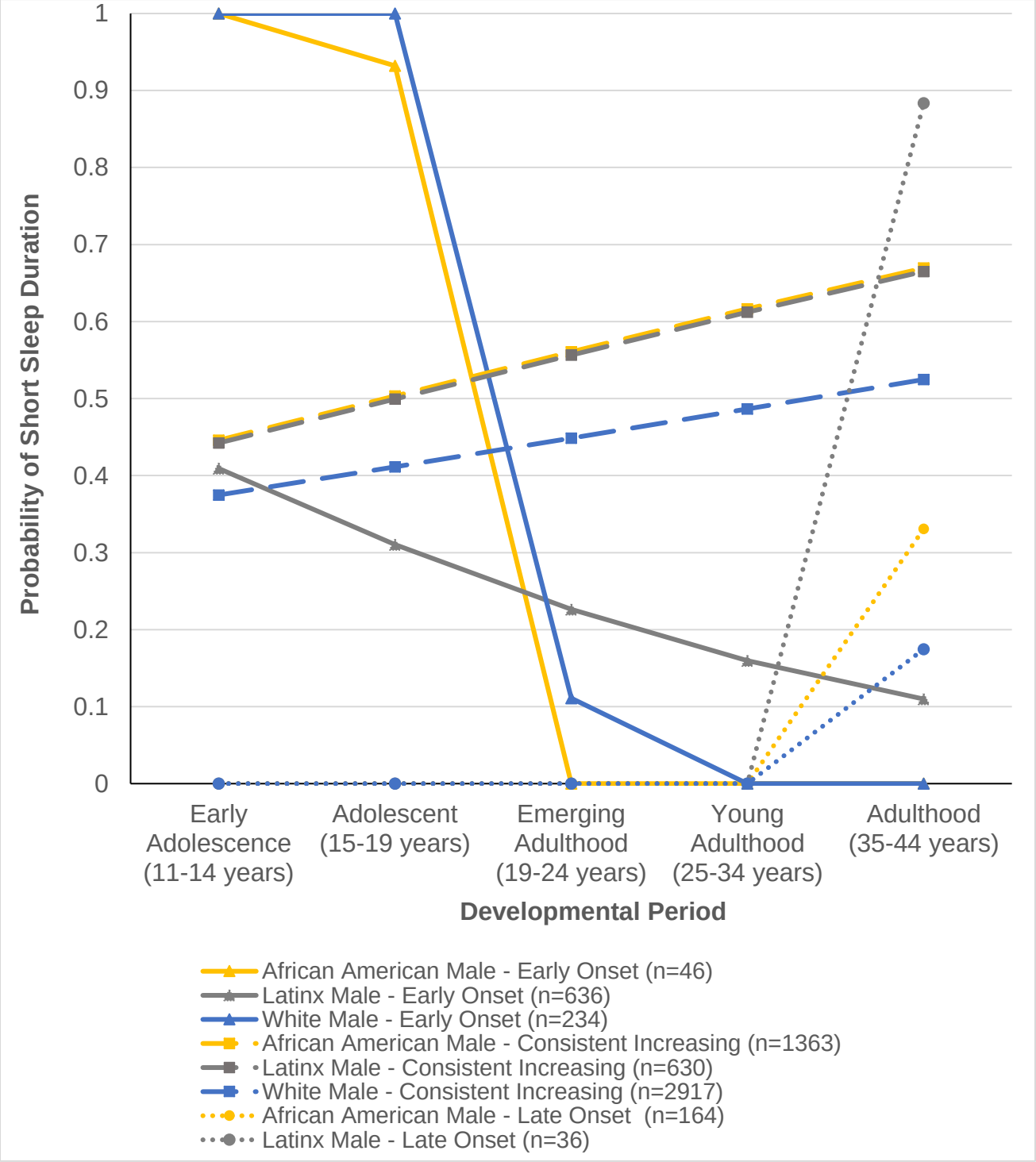
[a]Values expressed as mean (standard deviation)

Figure 3-1. Multi-Group Growth Mixture Model Short Sleep Duration Trajectories by Race/Ethnicity among Females, United States, 1994-2018



Note: The Early Onset trajectory for Latinx and White females are overlapping. The Late Onset trajectories are overlapping at zero probability from early adolescence to young adulthood for all race groups.

Figure 3-2. Multi-Group Growth Mixture Model Short Sleep Duration Trajectories by Race/Ethnicity among Males, United States, 1994-2018

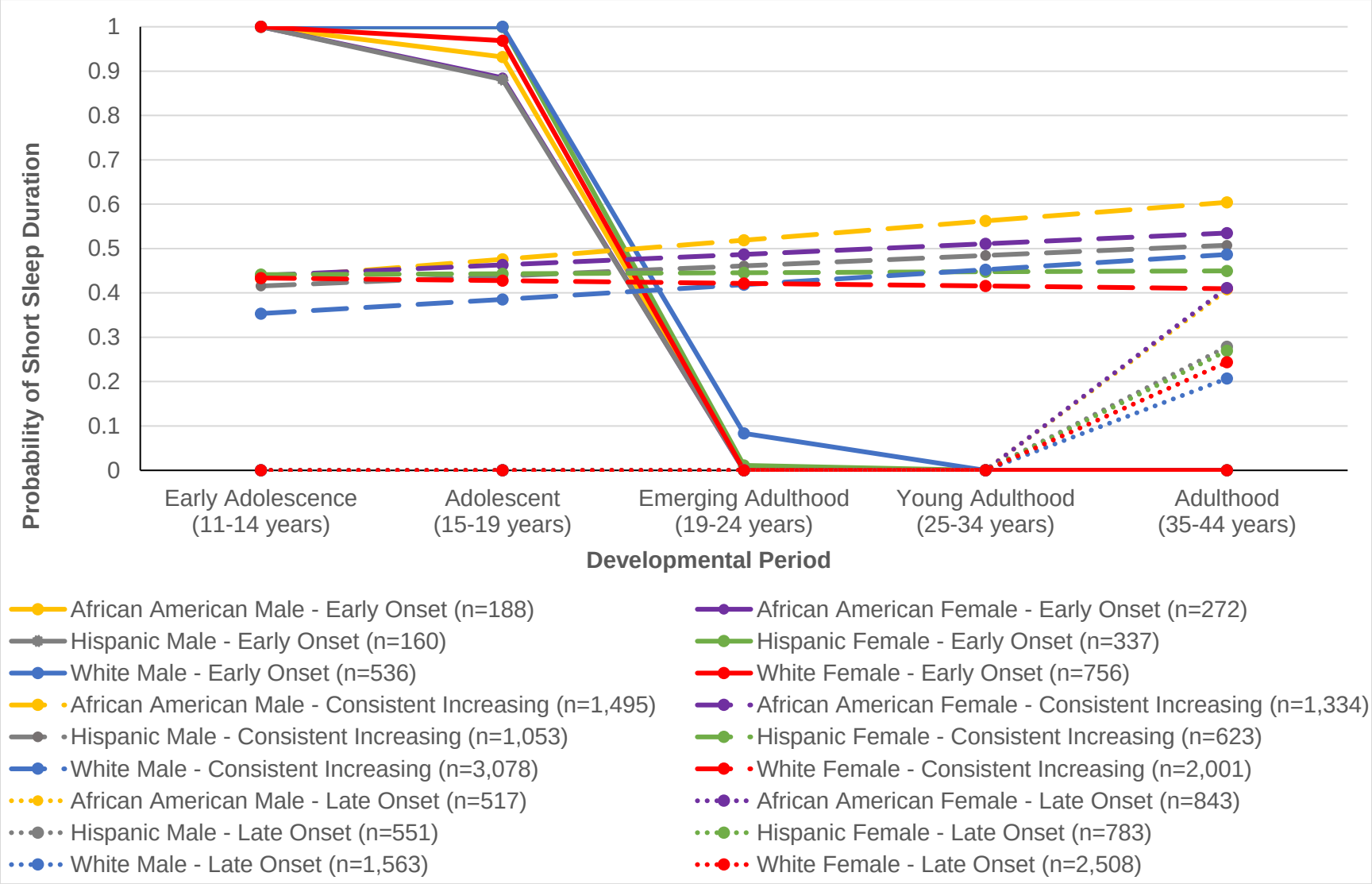


Note: The Early Onset trajectory for African American and White males overlap at zero probability from young adulthood to adulthood**.** The Late Onset trajectories are overlapping at zero probability from early adolescence to young adulthood for all race groups.

Table 3-2. Distribution of Growth Mixture Model Short Sleep Duration Trajectories by Race/Ethnicity and Sex/Gender, United States, 1994-2018

	Consistent Increasing (N=8,475; 67.3%)		**Late Onset (N=2,542; 20.2%)**		**Early Onset (N=1,576; 12.5%)**	
Race/Ethnicity and Sex	**N**	**%**	**N**	**%**	**N**	**%**
African American						
Female	1,047	68.7	346	22.7	132	8.7
Male	1,363	86.7	164	10.4	46	2.9
Latinx						
Female	731	64.9	277	24.6	118	10.5
Male	630	48.4	36	2.8	636	48.9
White						
Female	1,787	55.2	1,039	32.1	410	12.7
Male	2,917	76.1	680	17.8	234	6.1

Supplemental Figure 3-1. Growth Mixture Model Short Sleep Duration Trajectories by Race/Ethnicity and Sex/Gender including Long Sleepers, United States, 1994-2018



Supplemental Table 3-1. Comparison of Analytical Sample to Excluded Sample from Waves I, III, IV, and V of the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-2018

Characteristics	Analytic Sample (n=12,593)		Excluded (n=6,305)	
	N	%	N	%
Female, n (%)	5,887	46.8	3,594	53.4
Race/ethnicity, n (%)				
African American	3,098	24.6	1,571	25.9
Hispanic	2,428	19.3	1,097	18.1
White	7,067	56.1	3,388	55.9
Age in years[a]				
Wave I		15.7 (<0.1)		15.5 (<0.1)
Wave III		22.0 (1.8)		21.8 (1.8)
Wave IV		28.5 (1.8)		28.3 (1.8)
Wave V		38.1 (1.9)		37.8 (1.9)
Highest Parental Education, Wave 1				
< high school	3,646	30.3	1,791	31.4
High school graduate	1,585	13.2	903	15.8
Some college	2,635	21.9	1,202	21.1
College or graduate education	4,180	34.7	1,804	31.7
Highest Education, Wave 3, n (%)				
< high school	1,343	16.1	579	11.0
High school graduate	3,440	41.3	1,836	34.8
Some college	2,603	31.2	2,009	38.1
College or graduate education	952	11.4	851	16.1
Highest Education, Wave 4, n (%)				
< high school	2,962	33.2	1,411	26.3
High school graduate	3,732	41.8	2,119	39.5
Some college	1,622	18.2	1,275	23.8
College or graduate education	607	6.8	562	10.5
Highest Education, Wave 5, n (%)				
< high school	3,037	42.6	1,399	34.8
High school graduate	2,717	38.1	1,512	37.6
Some college	1,136	15.9	866	21.5
College or graduate education	242	3.4	247	6.1
Depressive Symptoms[a]				
Wave I		11.0 (7.5)		11.7 (7.7)
Wave III		6.2 (3.2)		6.6 (3.4)
Wave IV		7.6 (3.6)		8.1 (4.0)
Wave V		2.3 (2.5)		2.5 (2.7)
Body Mass Index in kg/m^{2}[a]				
Wave I		22.6 (4.4)		22.5 (4.6)

Wave III		26.5 (6.0)		26.3 (6.3)
Wave IV		29.3 (7.5)		29.2 (7.7)
Wave V		30.7 (7.6)		31.1 (8.2)
Self-reported Health, Wave 1				
Excellent	3,656	29.0	1,665	27.6
Very Good	5,069	40.3	2,261	37.4
Good	3,026	24.0	1,651	27.3
Fair	772	6.1	435	7.2
Poor	66	0.5	32	0.5
Self-reported Health, Wave 3				
Excellent	2,818	33.8	1,686	31.9
Very Good	3,453	41.4	2,070	39.2
Good	1,746	20.9	1,232	23.3
Fair	304	3.6	268	5.1
Poor	21	0.3	27	0.5
Self-reported Health, Wave 4				
Excellent	1,735	19.4	1,038	19.3
Very Good	3,509	39.3	1,917	35.7
Good	2,887	32.4	1,833	34.2
Fair	714	8.0	507	9.5
Poor	78	0.9	72	1.3
Self-reported Health, Wave 5				
Excellent	1,201	16.8	649	16.1
Very Good	2,666	37.4	1,379	34.3
Good	2,373	33.3	1,378	34.3
Fair	751	10.5	497	12.4
Poor	142	2.0)	117	2.9

[a]Values expressed as mean (standard deviation)

Supplemental Table 3-2. Growth Mixture Model Fit Indicators for Short Sleep Duration Trajectories by Race/Ethnicity and Sex/Gender, United States, 1994-2018

Number of Classes	AIC	BIC	Entropy
2	89591.49	89851.93	0.80
3	89184.43	89578.80	0.86
4	89184.66	89712.96	0.83

Note: Models were conducted with 5000 starts using an expectation maximization algorithm. The best log likelihood was not replicated for 2- and 4-class models.

Supplemental Table 3-3. Multi-Group Growth Mixture Model Results - Estimated Probabilities Across Developmental Periods for Each Trajectory Type by Race/Ethnicity and Sex/Gender, United States, 1994-2018 (n=12,593)

	Estimated Probabilities for Each Trajectory Type by Race/ethnicity and Sex																	
	African American Female			**African American Male**			**Latinx Female**			**Latinx Male**			**White Female**			**White Male**		
	Type:			Type:			Type:			Type:			Type:			Type:		
	C	**E**	**L**	**C**	**E**	**L**	**C**	**E**	**L**	**C**	**E**	**L**	**C**	**E**	**L**	**C**	**E**	**L**
Early Adolescence (11-14 years)	.48	1.00	0	.45	1.00	0	.46	1.00	0	.44	.41	0	.45	1.00	0	.37	1.00	0
Adolescent (15-19 years)	.51	1.00	0	.50	.93	0	.47	1.00	0	.50	.31	0	.45	1.00	0	.41	1.00	0
Emerging Adulthood (19-24 years)	.54	0	0	.56	.00	0	.48	.06	0	.56	.23	0	.46	.06	0	.45	.11	0
Young Adulthood (25-34 years)	.57	0	0	.62	.00	0	.49	0	0	.61	.16	0	.46	0	0	.49	0	0
Adulthood (35-44 years)	.60	0	.42	.67	.00	.33	.50	0	.29	.67	.11	.88	.46	0	.23	.52	0	.17

Abbreviations for trajectory type: C = Consistent Increasing Short Sleepers (i.e., increasing probability of short sleep into adulthood); E = Early Onset Short Sleepers (i.e., declining probability of short sleep from adolescence into adulthood); L = Late Onset Short Sleepers (i.e., no probability of short sleep in adolescence, but in adulthood)

Chapter 4: Racial/Ethnic and Sex/Gender Differences in the Association between Sleep Duration Trajectories and Adult Cardiometabolic Risk in a U.S. National Sample

Abstract

Research understanding the impact of sleep duration across developmental periods on adult cardiometabolic risk factors (CRFs) remains limited. Using data from Waves I, III, IV, and V of the National Longitudinal Study of Adolescent to Adult Health (1994-2018; n=2,302), growth mixture modeling and Poisson regression with robust error variance was used to estimate racial/ethnic and sex/gender differences in the association between self-reported sleep duration trajectories from adolescence to adulthood and adult CRFs (e.g., diabetes, dyslipidemia, elevated C-reactive protein (CRP), high waist circumference, hypertension, obesity, and metabolic syndrome). Females, irrespective of race, and White males with an Early Onset (i.e., declining probability of short sleep duration from adolescence into adulthood) or Late Onset (i.e., no short sleep duration until adulthood) compared to a Consistent Increasing (i.e., increasing probability of short sleep into adulthood) trajectory, generally had lower prevalence of CRFs. However, there was a higher prevalence of dyslipidemia for African American males and elevated CRP for Latinx males in Early and Late Onset trajectories. These findings highlight the cumulative and long-term influence of sleep duration on adult cardiometabolic health and the need to consider both adolescent and adult sleep duration when investigating the association between sleep duration and adult cardiometabolic risk.

Introduction

Cardiovascular disease (CVD) remains one of the leading causes of death in the U.S. accounting for 1 in every 4 deaths among adults (1). Racial/ethnic and sex/gender disparities in the prevalence of cardiometabolic risk factors have been well documented among adults (1). The prevalence of obesity is the highest among non-Hispanic Black and Hispanic females, and diabetes is the most prevalent among non-Hispanic Black and Hispanic males (1). Interventions to address these disparities have primarily focused on physical activity, diet, and tobacco use (15, 232). An emerging area of interest for interventions to prevent CVD risk is sleep.

Previous systematic reviews and meta-analyses in adult populations have linked short sleep duration (<7 hours of sleep per night) to numerous cardiometabolic risk factors such as obesity (19, 20), diabetes (202), hypertension (24), high cholesterol (26), and inflammation (27). Biological pathways in which sleep duration may influence cardiometabolic risk may be through altering metabolic hormones. Studies examining the associations of short sleep duration with appetite-regulating hormones found that short sleep duration was associated an increase in ghrelin and reduction in leptin (33, 34). These changes can result in increased hunger and appetite for high fat, sweet, starchy and salty foods subsequently increasing the risk for obesity, type-2 diabetes, hypertension, and dyslipidemia. Additionally, shorter sleep duration has been linked to decreased insulin sensitivity, which is directly related to an increased risk for type-2 diabetes, lower HDL cholesterol and higher triglyceride levels (35-37). Research also suggests that shorter sleep duration is associated with sympathetic nervous system hyperactivity resulting in elevated blood pressure (233). Furthermore, short sleep may lead to immune dysregulation and inflammation, which is associated with increased cardiometabolic risk (234, 235). Studies on behavioral pathways have found that shorter sleep duration is associated with lower levels of physical activity, and greater sedentary behaviors, smoking, and alcohol consumption, which may subsequently increase the risk for obesity, hypertension, dyslipidemia, inflammation, and diabetes (10, 236-245).

Research suggests that there may be a long-term impact of sleep duration on cardiometabolic health Longitudinal studies among children with multiple measures of sleep duration have found that increases in sleep duration is associated with decreases in body mass index and insulin resistance in later childhood (246, 247). On the contrary, prospective studies among adults with multiple assessments of sleep duration suggest that increases in sleep duration increases weight gain and diabetes risk, but improves diet quality and physical activity in later adulthood (248, 249). Collectively these studies indicate that there may be a lasting impact of sleep duration on cardiometabolic health, but whether there is longer-term impact into later developmental periods is not well understood. Studies among a nationally representative U.S. sample have found that shorter sleep duration during adolescence was associated with obesity (91) and high cholesterol for women (92) in young adulthood. However, these studies only included single measurements of sleep in one point in time. Including multiple measurements of sleep duration across various developmental periods is important as it may provide further insight on the on the long-term and cumulative impact of short sleep duration. For instance, studies conducted in U.S. nationally representative samples found that consistent short sleep duration (<6 hours) from adolescence to young adulthood was associated with a greater likelihood for obesity (93) and asthma (94) in young adulthood. However, there are limited studies exploring sleep duration trajectories from adolescence to adulthood in relation to adult cardiometabolic health. These studies are important for developing prevention strategies as this knowledge can provide greater clarity on the developmental periods when sleep influences adult cardiometabolic health

Short sleep duration is prevalent among both adolescents and adults. Approximately 40% of adolescents (40) and 35% of adults (41) report sleep less than the recommended amount of sleep by age. Racial/ethnic and sex/gender disparities in sleep duration among both adolescents (42, 56) and adults (43) have been documented with studies indicating that on average, African American males and females have the shortest sleep duration while White

females have the longest sleep duration (42, 43, 56, 57). Despite research suggesting that various sleep duration trajectories may exist from adolescence to adulthood (94, 95), no large cohort study to our knowledge has explored race/ethnicity and sex/gender differences in these trajectories. Racial/ethnic and sex/gender differences in sleep duration trajectories from adolescence to adulthood could occur given that various race and sex groups have unique sociocultural experiences (e.g. racism and sexism) that could differentially shape their sleep duration across the life course (46). Given that sleep duration is associated with cardiometabolic risk in both adolescent and adult populations, disparities in sleep duration trajectories during adolescence to adulthood could also possibly contribute to the observed racial/ethnic and sex/gender disparities in adult cardiometabolic health.

Thus, the purpose of this study is to examine racial/ethnic and sex differences in the association between sleep duration trajectories and cardiometabolic risk factors. We hypothesize that persistent short sleep duration from adolescence to adulthood will be associated with a greater likelihood for cardiometabolic risk factors.

Methods

Study Population

Data were from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is nationally representative longitudinal study examining adolescent behaviors and various health outcomes in adulthood. From 1994-1995, 90,118 students were recruited from 80 high schools and 52 middle schools based on region of country, urbanicity, school size, school type, and race/ ethnicity to participate in an in-school questionnaire regarding their extracurricular activities, relationships, and health behaviors. A random subsample of these students (n=20,745) were selected in 1995 to participate in the Wave I in-home interviews for more comprehensive information. Four follow-ups were conducted with the sample from Wave I that comprised Wave II in 1996 (n=14,738; mean age=16.2 years), Wave

III from 2001-2002 (n=15,197; mean age=22.0 years), Wave IV in 2008 (n=15,701; mean age=28.5 years), and Wave V from 2016-2018 (n=12,300; mean age=37.0 years). In Wave V, a random subsample of participants from the Wave I in-home interviews was selected and agreed to anthropometric assessments and blood sample collection (n=5,381). The study design and sampling methods are described in greater detail elsewhere (140).

Those with missing data on sleep across all Waves of data and those who did not identify as African American, Hispanic, or White were excluded (n=2,109). Long sleepers (defined as >12 hour for 6-12 years old, >10 hours for 13-18 years old, >9 hours for over 18 years old) were removed for analyses instead of being grouped with short sleepers or those with the recommended amount of sleep. This decision was based on prior literature suggesting that pathways connecting long sleep to health may differ from those linking short sleep to health and there was a limited sample size of long sleepers in Wave I (n=556) and V (n=480) (204, 250-253). The final sample consisted of 12,593 participants for the sleep duration trajectory analyses. For analyses examining the association between sleep duration trajectories and each cardiometabolic risk factor, those without blood draws for biomarker measures (e.g., hemoglobin A1c, fasting glucose, blood pressure measurements, lipid levels, and high sensitivity C-reactive protein) and those who refused to provide or had invalid anthropometric measures (e.g., height, weight, and waist circumference) (n=610) and had high-sensitivity C-reactive protein (hs-CRP) >10 mg/L were excluded (n=216) resulting in 2,302 participants for the analytic sample. In comparing the analytic sample (n=2,302) to those excluded, (i.e., non-participants of Wave V or had missing data) (n=18,443), participants were more likely to be African American, Hispanic, female, and have diabetes, elevated CRP, and high waist circumference, but less likely to have parents that completed a college education or completed college themselves (Supplemental Table 4-1). The Add Health study was approved by the institutional review board of the University of North Carolina, Chapel Hill with written consent

forms obtained from adolescents and their parents. This current analysis was approved by the institutional review board of Emory University.

Race/Ethnicity and Sex

Categories for race/ethnicity were created based on recommendations from Add Health investigators (151). In Wave I, participants were asked whether they were of Hispanic or Latino origin. A separate question asked participants to indicate their race with White, Black or African American, American Indian or Native American, Asian or Pacific Islander, or Other as potential choices with the ability to select multiple racial groups. Participants that identified being Hispanic or Latino origin were racialized as “Hispanic” and were eliminated from any race category they selected. If participants did not indicate they were of Hispanic or Latino origin and selected "black or African American" as one of their racial identities, they were designated as African American for their race/ethnicity, and were omitted from the other selected racial categories. This was repeated for the remaining racial groups in the following order: Asian, Native American, Other, and non-Hispanic White. Hence forth, Hispanic will be referred to as Latinx and non-Hispanic White will be referred to as White. Participants were asked to identify their sex as male or female. The Add Health study did not assess gender as a separate construct. Therefore, sex will represent both the biological and social aspects (e.g., gender). For this analysis, race/ethnicity and sex/gender groups included African American male, African American female, Latinx male, Latinx female, White male, and White female. Other race/ethnicity and sex/gender groups were excluded due to their small sample size (n=443).

Sleep Duration Trajectories

Sleep duration was measured in Waves I and V by asking participants to provide a response in whole hours to the question “How many hours of sleep do you usually get?” In Waves III and IV participants were asked the following four questions: 1) “On days when you go

to work, school, or similar activities, what time do you usually wake up?"; 2) "What time do you usually go to sleep the night (or day) before?"; 3) "On days you don't have to get up at a certain time, what time do you usually get up?"; 4) "On those days, what time do you usually go to sleep the night or day before?". The first two items were subtracted to estimate the sleep hours during the weekday while the other two items were used for weekend sleep hours. Total sleep hours per day was calculated using a weighted average of the weekday and weekend sleep duration (2/7*weekends + 5/7*weekdays). The American Academy of Sleep Medicine (AASM) guidelines by age for healthy sleep is 9-12 hours for 6-12 years old, 8-10 hours for 13-18 years old, and 7-9 hours for 18-64 years old (149, 150). Categories of sleep duration included short sleep duration (less than recommended by AASM) vs recommended sleep duration (within the recommended by AASM) with long sleepers (more than recommended by AASM) being excluded for the sleep duration trajectories. Multiple group growth mixture models (GMMs) were then used to identify sleep duration trajectories from adolescence to adulthood for each race and sex group. The GMMs examined changes in the probability of short sleep duration by developmental periods, which included early adolescence (11-14 years), adolescence (15-19 years), emerging adulthood (20-24 years), young adulthood (25-34 years), and adulthood (35-44 years). The 3-class, compared to the 2 and 4-class models, was the best fitting model based on having the lowest Akaike and Bayesian information criterion, and highest entropy. The model resulted in 18 different trajectories (i.e., three trajectories for each race and sex group) with varying probabilities. Based on visual inspection, these trajectories were grouped into three common trajectory types: 1) Consistent Increasing Short Sleepers (i.e., increasing probability of short sleep into adulthood); 2) Late Onset Short Sleepers (i.e., no probability of short sleep duration in adolescence until adulthood); and 3) Early Onset Short Sleepers (i.e., declining probability of short sleep duration from adolescence into adulthood). The distribution of the sleep duration trajectory types by race/ethnicity and sex/gender are displayed in Figures 3-1

and 3-2. Methods for the enumeration of sleep duration trajectories by race and sex have been described in greater detail in our previous study (254).

Cardiometabolic Risk Factors (CRFs)

The CRFs were measured using the standardized protocol for the in-home interviews by trained staff at Wave V (255-259). Height was measured to the nearest 0.5 cm using a carpenter's square, steel tape measure, and adherent Post-it note. Weight was measured to the nearest 0.1 kg using a digital scale. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Waist circumference was assessed to the nearest 0.5 cm at the superior border of the iliac crest using a circumference tape measure. Systolic and diastolic blood pressure were measured three times at 30-second intervals, after the participant had rested five minutes in a seated position, using an oscillometric blood pressure monitor. The average of the second and third readings were used for the analysis. Venous blood samples were collected during the home visit and assayed in a laboratory for triglycerides, total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, glucose, hemoglobin A1c (HbA1c), and hs-CRP. CRFs included in analyses were dichotomized based on clinical cutoffs: obesity (BMI ≥ 30 kg/m^2) (260), high waist circumference (>102 cm for males/ >88 cm for females) (260), diabetes (HbA1c ≥6.5% or fasting glucose ≥126 mg/dl or non-fasting glucose ≥200 mg/dl or self-reported history of diabetes except during pregnancy or anti-diabetic medication-use in the past 4 weeks) (261), hypertension (systolic ≥130mm Hg and/ or diastolic ≥ 80 mm Hg blood pressure) (262), and dyslipidemia (fasting or non-fasting total cholesterol ≥ 200 mg/dl, fasting triglycerides ≥150 mg/dl or fasting or non-fasting HDL cholesterol <40 mg/dl or fasting LDL cholesterol > 100mg/dl) (263), and elevated CRP (hs-CRP> 3 mg/L) (264). The definition of diabetes also included self-reported information due to the small sample size of participants with diabetes based only on glucose and HbA1c measures (n=88).

Statistical Analysis

Distribution of participant characteristics were compared by race/ethnicity and sex using chi-square tests and ANOVAs. Trajectory classes from the GMMs from our prior study (254) were used to conduct stratified analyses by race/ethnicity and sex/gender groups to examine associations between sleep duration trajectories (Early Onset and Late Onset vs. Consistent Increasing) and each of the CRFs. This was conducted using Poisson regression models with robust error variance to estimate prevalence ratios (PR) and 95% confidence intervals (CI) because of the high prevalence of each CRF (>10%). However, diabetes prevalence was low (>10%) across race and sex groups, thus logistic regression was utilized to estimate the odds ratio (OR) and the 95% CIs. The regression models discussed below (see Table 4-2) did not account for sampling weights given the GMMs were not weighted and a smaller sample size due to missing sampling weights. Sensitivity analyses were conducted applying sampling weights to the regression models for comparison to the unweighted results (Supplemental Table 4-2). Descriptive analyses and both Poisson and logistic regression models were conducted with SAS v9.4 (152).

Results

Sample Characteristics

Racial/ethnic and sex differences in sleep duration trajectories and each of the CRFs are shown in Table 4-1 for the sample analyzed in the Poisson and logistic regression models. Overall, the mean age was 37.9 years and the most prevalent sleep duration trajectory was Consistent Increasing (57.7%), followed by Early Onset (14.6%) and then Late Onset (27.7%). The most prevalent CRF in the overall sample was dyslipidemia (62.5%) followed by hypertension (49.3%), high waist circumference (48.8%), obesity (44.6%), elevated CRP (37.9%), and then diabetes (6.9%).

The Early Onset short sleep duration trajectory was the most prevalent among Latinx males while the Consistent Increasing trajectory was the most prevalent among African American males and the Late Onset trajectory was the most common among White females. The prevalence of CRFs were the highest among African American and Latinx participants with significant sex differences.

Regression Results Stratified by Race/ethnicity and Sex/Gender (See Table 4.2)

African American Females

The prevalence of hypertension was lower among Early Onset compared to Consistent Increasing short sleepers (PR: 0.88; 95% CI: 0.27, 0.97). There were no differences in the prevalence of other CRFs comparing Early and Late Onset to Consistent Increasing trajectory.

African American Males

The prevalence of dyslipidemia was higher among Early (PR: 1.49; 95% CI: 1.21, 1.85) and Late Onset (PR: 1.29; 95% CI: 0.99, 1.68) compared to Consistent Increasing short sleepers. No other significant associations were found in comparing the prevalence of the CRFs between trajectories.

Latinx Females

Early Onset compared to Consistent Increasing short sleepers were more likely to have elevated CRP (PR: 0.43; 95% CI: 0.19, 0.98). There was no association for the other CRFs. Those with a Late Onset trajectory were less likely to have elevated CRP (PR: 0.62; 95% CI: 0.38, 1.03), high waist circumference (PR: 0.65; 95% CI: 0.45, 0.94) and obesity (PR: 0.58; 95% CI: 0.38, 0.88) than those with a Consistent Increasing trajectory, but no associations were detected for the other CRFs.

Latinx Males

There were no associations found comparing Early Onset to Consistent Increasing short sleepers. The prevalence of elevated CRP (PR: 2.15; 95% CI: 1.03, 4.51) were higher among

Late Onset compared to Consistent Increasing short sleepers, but there were no differences found for the other CRFs.

White Females

The prevalence of elevated CRP (PR: 0.61; 95% CI: 0.43, 0.86), high waist circumference (PR: 0.74; 95% CI: 0.59, 0.92), and obesity (PR: 0.76; 95% CI: 0.58, 1.00) was lower for Early Onset compared to Consistently Increasing short sleepers, and no differences were found for the other CRFs. Similar associations were found comparing Late Onset to Consistent Increasing short sleepers with a lower prevalence of elevated CRP (PR: 0.75; 95% CI: 0.60, 0.93), high waist circumference (PR: 0.88; 95% CI: 0.77, 1.01), obesity (PR: 0.80; 95% CI: 0.66, 0.96), and metabolic syndrome (PR: 0.71; 95% CI: 0.54, 0.93) among Late Onset short sleepers.

White Males

Early Onset compared to Consistent Increasing short sleepers were more likely to have dyslipidemia (PR: 1.10; 95% CI: 0.98, 1.24), but less likely to have obesity (PR: 0.71; 95% CI: 0.51, 0.99). No associations were observed for the other CRFs. For the Late Onset to Consistent Increasing trajectory comparison, Late Onset short sleepers were less likely to have high waist circumference (PR: 0.74; 95% CI: 0.58, 0.94), hypertension (PR: 0.88; 95% CI: 0.76, 1.01), and obesity (PR: 0.71; 95% CI: 0.57, 0.89), but no other significant associations with the other CRFs were found.

Sensitivity Analysis

Analyses accounting for Add Health weights yielded similar results to the unweighted analyses. The primary difference was that weighted results had less precise confidence intervals due to a smaller sample size from excluding participants with missing data on weights. (Supplemental Table 4-2).

Discussion

In a nationally representative U.S. sample, there were racial/ethnic and sex/gender differences in the association between sleep duration trajectories from adolescence to adulthood and adult cardiometabolic risk factors. The findings support our hypothesis that those with consistent short sleep duration from adolescence to adulthood would be more likely to have adverse cardiometabolic health. Females, irrespective of race, and White males with an Early Onset or Late Onset (i.e., no short sleep duration until adulthood) compared to a Consistent Increasing trajectory, generally had lower prevalence of CRFs. However, there was a higher prevalence of dyslipidemia for African American males and elevated CRP for Latinx males in the Early and Late Onset compared to the Consistent Increasing trajectory.

The results from this study are consistent with prior research examining the cumulative impact of sleep duration from adolescence to adulthood on health during adulthood. Two separate studies utilizing data from Add Health found that consistent short sleep duration from adolescence to young adulthood was associated with a higher likelihood of obesity (93) and asthma (94) when compared to those with consistent adequate sleep. Our study expanded on these prior studies by examining racial/ethnic and sex/gender differences in the cumulative impact of short sleep duration from adolescence to adulthood on adult cardiometabolic health. The null findings for some CRFs among the various race and sex groups may be attributed to the small sample in each strata that resulted in the study being underpowered to detect an association.

The contrasting findings of a higher prevalence of dyslipidemia for Early and Late Onset compared to Consistent Increasing short sleepers among African American males could also be explained by the small sample size of Early and Late Onset short sleepers that resulted in spurious associations. Another explanation for the higher prevalence of dyslipidemia for Early Onset compared to Consistent Increasing short sleepers is the high probability of short sleep duration during adolescence among Early Onset short sleepers whereas Consistent Increasing

short sleepers have approximately half the probability for short sleep duration in adolescence. This suggests that short sleep duration during adolescence may have a long-term adverse impact on adulthood cardiometabolic health absent of short sleep in adulthood. A previous Add Health study also found that short sleepers during adolescence were more likely to have high cholesterol in young adulthood (92). In addition, our findings for Latinx males having greater prevalence of elevated CRP among Late Onset short sleepers could be due to the high probability of short sleep duration in adulthood for Late Onset compared to the Consistently Increasing short sleepers who had consistent mid-level probability of short sleep duration from adolescence to adulthood. Collectively, the results from this and prior research highlight the long term impact of sleep and the need to include longitudinal measures of sleep duration across the life course when investigating the relationship between sleep duration and adult cardiometabolic risk for specific race/ethnicity and sex/gender groups.

There were at least three strengths of this study. First, the longitudinal design allows for the examination of the impact of sleep duration across developmental periods on adult cardiometabolic risk. Second, the study was conducted among a large racially diverse sample that allowed for comprehensive analysis of subgroup differences. Finally, this study was able to include objective measurements (e.g. venous blood samples and measured height, weight, and waist circumference) for each cardiometabolic risk factor, which is important for reducing measurement error based on self-report.

Despite these strengths, there are at least two limitations that need to be considered when interpreting the results. First, the assessment of sleep duration based on self-report in which past research has shown that both adolescents (57, 194, 225, 265) and adults (195, 196, 227) tend to over report their sleep duration compared to objective measures (e.g., actigraphy) with variations by race/ethnicity and sex/gender among adult populations (195, 196). For example, a study comparing actigraphy to self-reported sleep using data from the Multi-Ethnic Study of Atherosclerosis found that Black and Hispanic women had the highest self-reporting

bias, followed by White women and men, then Chinese women, and finally Black and Hispanic men (196). Data from the Jackson Heart Study found that self-reported averaged sleep duration was an underestimate while self-reported wake-bed differences were overestimates of actigraphy-assessed sleep duration (266). These potential measurement errors may have influenced the results for the growth mixture models in which the greater over reporting by some race/ethnicity and se/gender groups may have underestimated the prevalence of individuals in the consistent increasing probability of short sleep duration. Additionally, a large number of participants from the original Wave I in-home assessment who were excluded from analysis were more likely to be African American, Latinx, male, with lower education levels for their parents and themselves, and more likely to have cardiometabolic risk factors compared to those in the analyses. These differences in characteristics could potentially reduce the generalizability of these results.

To our knowledge, this is one of the first studies to investigate racial/ethnic and sex/gender differences in the impact of sleep duration trajectories from adolescence to adulthood on adult cardiometabolic health. The findings from this study underscore the need to consider adolescent sleep duration when investigating the relationship between sleep duration and adult cardiometabolic health. Furthermore, the implications of this research highlight the importance of intervening on sleep duration during adolescence in order to improve cardiometabolic health and reduce disparities in adulthood.

Future research should consider replicating this study with objective measures of sleep duration to reduce the amount of measurement error from self-reported sleep. Studies are also needed that include a larger sample size of diverse racial/ethnic minority participants with complete biomarker data to improve generalizability of results. Most importantly, future work must identify factors that buffer the impact of short sleep duration across the life course on adult cardiometabolic health for each race and sex group. This knowledge can be used to develop targeted interventions (e.g., sleep hygiene education, stress management, and increasing

physical activity) that help alleviate cardiometabolic risk and further reduce racial/ethnic and sex disparities in cardiometabolic health.

Table 4-1. Sample Characteristics by Race/Ethnicity and Sex/Gender for Analysis on the Association between Sleep Duration Trajectories and Cardiometabolic Risk Factors from the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-2018

Characteristics	Total (N=2,302)		African American Female (N=253)		African American Male (N=181)		Latinx Female (N=174)		Latinx Male (N=158)		White Female (N=811)		White Male (N=725)	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%
Sleep Duration Trajectories														
Early Onset Short Sleepers	337	14.6	25	9.9	12	6.6	27	15.5	75	47.5	123	15.2	75	10.3
Consistent Increasing Short Sleepers	1,328	57.7	159	62.9	150	82.9	95	54.6	70	44.3	388	47.8	466	64.3
Late Onset Short Sleepers	637	27.7	69	27.3	19	10.5	52	29.9	13	8.2	300	37.0	184	25.4
Cardiometabolic Risk Factors														
Diabetes	159	6.9	24	9.5	17	9.4	16	9.2	8	5.1	48	5.9	46	6.3
Dyslipidemia	1,437	62.4	129	51.0	125	65.8	96	55.2	119	75.3	439	54.1	536	73.9
Elevated C-Reactive Protein	643	27.9	87	34.4	52	28.7	60	34.5	44	27.9	266	32.8	134	18.5
High Waist Circumference	1,123	48.8	181	74.5	69	38.1	92	52.9	66	41.8	433	53.4	282	38.9
Hypertension	1,135	49.3	126	49.8	124	68.5	42	24.1	99	62.7	281	34.7	463	63.9
Obesity	1,027	44.6	139	54.9	93	51.4	82	47.1	83	52.5	323	39.8	307	42.3

Table 4-2. Results for the Association between Sleep Duration Trajectories and Cardiometabolic Risk Factors in Wave V of the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-2018

Cardiometabolic Risk Factors by Race/Ethnicity and Sex/Gender	Early vs. Consistent (Ref)		Late vs. Consistent (Ref)	
	PR	95% CI	PR	95% CI
African American Female (n=253)				
Diabetes	0.68	0.15, 3.13	0.48	0.16, 1.48
Dyslipidemia	0.97	0.65, 1.46	0.87	0.62, 1.13
Elevated CRP	0.68	0.33, 1.41	1.03	0.71, 1.50
High Waist Circumference	0.89	0.65, 1.22	1.03	0.87, 1.22
Hypertension	0.51	0.27, 0.97	0.85	0.63, 1.13
Obesity	0.88	0.57, 1.35	1.06	0.83, 1.35
Metabolic Syndrome	0.61	0.24, 1.54	0.88	0.53, 1.45
African American Male (n=181)				
Diabetes	1.15	0.14, 9.74	0.70	0.09, 5.76
Dyslipidemia	1.49	1.21, 1.85	1.29	0.99, 1.68
Elevated CRP	1.16	0.50, 2.69	0.92	0.42, 2.03
High Waist Circumference	1.08	0.54, 2.17	0.82	0.41, 1.63
Hypertension	0.93	0.62, 1.41	0.66	0.41, 1.08
Obesity	0.61	0.27, 1.38	0.67	0.37, 1.24
Metabolic Syndrome	1.14	0.49, 2.63	0.90	0.41, 1.98
Latinx Female (n=174)				
Diabetes	0.77	0.16, 3.77	1.02	0.32, 3.21
Dyslipidemia	1.02	0.72, 1.46	0.83	0.60, 1.16
Elevated CRP	0.43	0.19, 0.98	0.62	0.38, 1.03
High Waist Circumference	0.72	0.46, 1.12	0.65	0.45, 0.94
Hypertension	0.88	0.40, 1.93	0.91	0.50, 1.67
Obesity	0.72	0.44, 1.17	0.58	0.38, 0.88
Metabolic Syndrome	0.68	0.29, 1.59	0.70	0.37, 1.34
Latinx Male (n=158)				
Diabetes	0.36	0.07, 1.90	1.08	0.12, 10.11
Dyslipidemia	0.97	0.81, 1.16	0.90	0.61, 1.32
Elevated CRP	1.43	0.82, 2.51	2.15	1.03, 4.51
High Waist Circumference	1.07	0.72, 1.60	1.60	0.95, 2.69
Hypertension	0.87	0.67, 1.13	1.17	0.83, 1.65
Obesity	0.96	0.70, 1.31	1.16	0.72, 1.89
Metabolic Syndrome	1.07	0.66, 1.74	1.28	0.59, 2.78
White Female (n=811)				
Diabetes	0.81	0.34, 1.90	0.66	0.34, 1.27
Dyslipidemia	1.00	0.84, 1.20	0.91	0.79, 1.05
Elevated CRP	0.61	0.43, 0.86	0.75	0.60, 0.93
High Waist Circumference	0.74	0.59, 0.92	0.88	0.77, 1.01
Hypertension	1.09	0.84, 1.42	0.93	0.75, 1.15
Obesity	0.76	0.58, 1.00	0.80	0.66, 0.96

Metabolic Syndrome	0.83	0.58, 1.19	0.71	0.54, 0.93
White Male (n=725)				
Diabetes	0.90	0.17, 1.96	0.98	0.49, 1.95
Dyslipidemia	1.10	0.98, 1.24	0.96	0.87, 1.07
Elevated CRP	0.83	0.48, 1.44	0.90	0.62, 1.30
High Waist Circumference	0.82	0.59, 1.13	0.74	0.58, 0.94
Hypertension	0.95	0.78, 1.14	0.88	0.76, 1.01
Obesity	0.71	0.51, 0.99	0.71	0.57, 0.89
Metabolic Syndrome	0.75	0.52, 1.08	0.83	0.66, 1.05

Abbreviations: Ref = Referent Group, PR = Prevalence Ratio, CI = Confidence Interval, CRP = C-Reactive Protein
Note: The estimate for diabetes is an odds ratio.

Supplemental Table 4-1. Comparison of Characteristics between Analytic Sample in Regression Models to those Excluded †

Characteristics	Analytic Sample (n=2,302)	Excluded (n=18,443)
Race/Ethnicity and Sex/Gender Groups, % (n)*		
African American Female	11.0 (253)	13.5 (2,205)
African American Male	7.9 (181)	12.4 (2,030)
Hispanic Female	7.6 (174)	9.7 (1,577)
Hispanic Male	6.9 (158)	9.9 (1,616)
White Female	35.2 (811)	27.3 (4,461)
White Male	31.5 (725)	27.3 (4,458)
Age in years, Wave 1, mean (SD)	15.6 (1.7)	15.6 (1.8)
Age in years, Wave 5, mean (SD)*	37.9 (1.9)	38.0 (1.9)
Highest Parental Education, Wave 1, % (n)*		
< high school	25.7 (573)	30.5 (5,326)
High school graduate	10.0 (222)	14.0 (2,451)
Some college	22.7 (505)	21.1 (3,687)
College or graduate education	41.7 (930)	34.4 (6,011)
Highest Education, Wave 5, % (n)*		
< high school	2.7 (62)	4.5 (452)
High school graduate	12.3 (282)	18.8 (1,872)
Some college	36.9 (848)	37.7 (3,763)
College or graduate education	48.1 (1,104)	39.0 (3,895)
Cardiometabolic Risk Factors, Wave V, % (n)		
Diabetes*	6.9 (159)	10.0 (308)
Dyslipidemia	62.4 (1,437)	61.2 (1,489)
Elevated C-Reactive Protein*	27.9 (643)	44.2 (1,018)
High Waist Circumference*	48.8 (1,123)	53.3 (1,614)
Hypertension	49.3 (1,135)	48.2 (1,379)
Obesity	44.6 (1,027)	46.7 (1,389)

†Excluded due to non-participation in Wave V or missing data
*p-value <0.05, compared using chi-square tests for categorical variables or t-tests for continuous variables

Supplemental Table 4-2. Weighted Results for the Association between Sleep Duration Trajectories and Cardiometabolic Risk Factors in Wave V of the National Longitudinal Study of Adolescent to Adult Health, United States, 1994-2018

Cardiometabolic Risk Factors by Race/Ethnicity and Sex/Gender	Early vs. Consistent (Ref)		Late vs. Consistent (Ref)	
	PR	95% CI	PR	95% CI
African American Female (n=239)				
Diabetes	1.76	0.48, 6.51	0.69	0.28, 1.71
Dyslipidemia	0.81	0.41, 1.63	0.91	0.68, 1.21
Elevated CRP	0.52	0.78, 1.73	1.17	0.78, 1.73
High Waist Circumference	0.94	0.70, 1.25	1.08	0.90, 1.29
Hypertension	0.79	0.36, 1.74	0.90	0.66, 1.23
Obesity	1.00	0.63, 1.60	1.09	0.84, 1.41
Metabolic Syndrome	1.20	0.46, 3.15	1.01	0.54, 1.87
African American Male (n=179)				
Diabetes	0.50	0.05, 4.62	<.001	<.001, 0.04
Dyslipidemia	1.65	1.35, 2.00	1.27	0.95, 1.71
Elevated CRP	0.67	0.18, 2.56	0.44	0.13, 1.56
High Waist Circumference	1.51	0.74, 3.06	0.85	0.41, 1.75
Hypertension	0.95	0.53, 1.69	0.37	0.15, 0.89
Obesity	0.53	0.16, 1.75	0.69	0.34, 1.41
Metabolic Syndrome	1.01	0.28, 3.61	0.43	0.14, 1.34
Latinx Female (n=167)				
Diabetes	4.23	1.10, 16.32	0.57	0.12, 2.75
Dyslipidemia	1.72	1.21, 2.45	0.75	0.10, 1.40
Elevated CRP	0.61	0.14, 2.61	0.67	0.27, 1.65
High Waist Circumference	0.72	0.46, 1.12	0.65	0.45, 0.94
Hypertension	2.56	0.90, 7.28	1.32	0.48, 3.61
Obesity	0.66	0.23, 1.86	0.52	0.23, 1.17
Metabolic Syndrome	1.85	0.82, 4.19	0.55	0.17, 1.81
Latinx Male (n=154)				
Diabetes	0.18	0.01, 2.78	0.03	<0.01, 0.60
Dyslipidemia	0.92	0.71, 1.20	1.09	0.91, 1.30
Elevated CRP	1.11	0.40, 3.10	0.72	0.17, 3.06
High Waist Circumference	0.54	0.25, 1.16	0.71	0.19, 2.63
Hypertension	0.70	0.44, 1.13	1.27	0.83, 1.94
Obesity	0.56	0.31, 1.01	0.39	0.34, 2.10
Metabolic Syndrome	0.66	0.26, 1.66	1.16	0.26, 5.20
White Female (n=796)				
Diabetes	0.72	0.23, 2.22	0.86	0.34, 2.17
Dyslipidemia	0.97	0.78, 1.21	0.88	0.72, 1.07
Elevated CRP	0.60	0.35, 1.04	0.94	0.71, 1.23
High Waist Circumference	0.78	0.58, 1.04	0.95	0.77, 1.18
Hypertension	0.96	0.67, 1.37	0.92	0.68, 1.25
Obesity	0.75	0.53, 1.08	0.93	0.73, 1.17

Metabolic Syndrome	0.87	0.56, 1.33	0.81	0.56, 1.18
White Male (n=717)				
Diabetes	0.19	0.04, 0.90	0.99	0.43, 2.28
Dyslipidemia	1.14	1.00, 1.30	0.97	0.84, 1.11
Elevated CRP	0.56	0.26, 1.20	1.10	0.75, 1.61
High Waist Circumference	0.79	0.52, 1.21	0.78	0.58, 1.06
Hypertension	0.93	0.69, 1.24	0.88	0.72, 1.06
Obesity	0.69	0.45, 1.07	0.67	0.51, 0.89
Metabolic Syndrome	0.78	0.50, 1.21	0.82	0.62, 1.08

Abbreviations: Ref = Referent Group, PR = Prevalence Ratio, CI = Confidence Interval, CRP = C-Reactive Protein
Note: The estimate for diabetes is an odds ratio.

Chapter 5: Public Health Implications and Future Research

Public Health Implications

Cardiometabolic diseases remain pervasive in the U.S. with persistent race and sex disparities reported (1). Sleep may be an important modifiable risk factor that can help decrease cardiometabolic disease burden since various studies have demonstrated an association between short sleep duration and increased adverse cardiometabolic risk factors (20, 38, 202, 235). Short sleep duration is highly prevalent among U.S. adolescents and adults with racial/ethnic and sex disparities documented across adolescent and adult populations (42, 43, 56). The majority of studies have investigated sleep disparities in one developmental period and in relation to one social identity (e.g. race or sex) with few studies applying an intersectionality framework in documenting sleep disparities jointly by race and sex and across developmental periods. In addition, the underlying mechanisms driving racial disparities in sleep duration among adolescents are not well understood and warrant further research. This dissertation attempted to address these critical gaps in the literature and inform policies and interventions to reduce sleep disparities and improve cardiometabolic health.

Aim 1 sought to examine whether the neighborhood (e.g. neighborhood socioeconomic disadvantage, social cohesion, and perceived safety) and household environment (e.g. household SES and living in a single parent household) partially explain the racial disparities in short sleep duration among adolescents. Findings from Aim 1 of this dissertation provide further insight to whether neighborhood and household environments play a role in producing racial disparities in sleep duration among adolescents. Although the neighborhood factors explored in this study were not found to be significant mediators, household SES may be an important mechanism driving these racial disparities in sleep duration. Unexpectedly, living in a higher SES household was associated with a higher probability of short sleep duration among adolescents. It is important to note that these findings should not be interpreted as the need to lower household SES among racial/ethnic minorities in order to reduce sleep disparities. Rather,

the implication of these findings is that a nuanced approach, such as taking into account household SES, is needed to develop effective interventions to mitigate racial disparities in sleep among adolescents. This may include implementing strategies to protect adolescents from the negative influence that living in a higher household SES environment has on sleep. Some of the factors linking higher household SES to sleep among adolescents may be greater academic pressures and access to extracurricular activities (e.g., private tutoring, student clubs, and sports) that reduce the amount of time for sleep. Particularly for racial/ethnic minorities, there is evidence to suggest that higher SES may not confer the same health benefits as for non-Hispanic Whites. This may be attributed to higher SES racial/ethnic minorities being more likely to experience greater racial discrimination and social isolation which is associated with shorter sleep duration. Potential school-based interventions could involve policies for later school start times to allow more time for sleep and implementing requirements for students and staff to enroll in anti-racism courses which could reduce experiences of racial discrimination for racial/ethnic minority adolescents.

Aim 2 of this dissertation investigated race and sex differences in self-reported sleep duration trajectories from adolescence to adulthood. The results revealed that, although there were 18 trajectories for each of the six race and sex groups, they can be grouped into three trajectory types: 1) Consistent Increasing Short Sleepers (i.e., increasing probability of short sleep into adulthood); 2) Late Onset Short Sleepers (i.e., no probability of short sleep duration in adolescence until adulthood); and 3) Early Onset Short Sleepers (i.e., declining probability of short sleep duration from adolescence into adulthood). Race and sex differences in the distribution of these sleep trajectory types were observed with African American males being the most likely to have the Consistent Increasing trajectory, Latinx males being the most likely to have a Late Onset trajectory, and non-Hispanic White females having the highest prevalence of an Early Onset trajectory. These findings suggest that sleep interventions during early adolescence may be crucial in preventing short sleep duration in adulthood, given that the

Consistent Increasing short sleep trajectory was the most prevalent in the total sample combined. However, across race and sex groups, the prevalence of the Consistent Increasing short sleep trajectory was the highest among African American males indicating that interventions to improve sleep and alleviate disparities may have the greatest public health impact in communities with a high proportion of African American males. Studies examining sleep disparities exclusively by race or sex may miss these nuances and inaccurately suggest certain race or sex groups to be at higher risk for short sleep duration when it may depend on both race and sex. These findings further add to the mounting evidence of the importance in using an intersectionality approach when documenting sleep disparities across the life course.

Aim 3, to our knowledge, is one of the first studies to examine the impact of sleep duration trajectories from adolescence to adulthood on adult cardiometabolic health by race and sex. Among females, irrespective of race, and White males, having a consistently increasing probability of short sleep duration was generally associated with a higher prevalence of cardiometabolic risk factors. On the contrary, there was a higher prevalence of dyslipidemia for African American males and elevated C-reactive protein for Latinx males who had a probability of short sleep in either adolescence or adulthood compared to those with an increasing probability of short sleep from adolescence into adulthood. Findings from this study reveal a long-term and cumulative impact of short sleep duration across the life course on cardiometabolic health. Moreover, the implications of these results suggests that interventions and policies to improve sleep may need to target specific race and sex groups and developmental periods in order to improve cardiometabolic health.

Altogether, this dissertation contributes to the literature by strengthening the case for the need to apply a life course framework in examining sleep in relation to adult cardiometabolic health. Doing so can advance sleep disparities research and inform interventions targeting specific race and sex groups at certain developmental periods to reduce these disparities. Ultimately, this can decrease cardiometabolic disease disparities.

Future Research

The limitations of this dissertation provides insight for future studies to further advance sleep and cardiometabolic health disparities research. Across all of the aims, sleep duration was self-reported which can result in measurement error of sleep by race and sex. Although it may be resource intensive because of the long follow-up period and large sample size, future work should replicate these study aims using an objective measure of sleep such as actigraphy to reduce measurement error. Given the reduced sample size in Aim 3 due to missing cardiometabolic measures for a large number of participants, future studies examining the relationship between sleep duration trajectories and cardiometabolic health should recruit a larger and more racially diverse cohort with complete data on cardiometabolic risk factors. This can improve the power of the study and allow for the examination of other racial/ethnic and sex groups not included in our current study.

Beyond addressing the current limitations of this dissertation, future research must identify mechanisms underlying both race and sex sleep disparities and the relationship between sleep duration trajectories from adolescence to adulthood with adult cardiometabolic health. Given that race and sex were proxy measures for racism and sexism in this dissertation, future work must include direct assessments interpersonal and structural racism and sexism and assess its impact on sleep duration trajectories across the life course. Since Aim 1 findings suggest that adolescents living in a higher household SES were more likely to have short sleep duration, future studies should identify buffers that protect adolescents from the harmful effects of living in a higher household SES environment. Also, because Aims 2 and 3 found race and sex differences in sleep duration trajectories and associations with various adult cardiometabolic risk factors, future work should explore modifiable factors, such as depressive symptoms, physical activity, and diet, that may link sleep duration trajectories to adult cardiometabolic health. Understanding these mechanisms will be more informative in developing effective

interventions and strategies to help mitigate sleep and cardiometabolic health race and sex disparities.

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