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Association between Adolescent Sleep Duration and Cardiovascular Risk in Young Adulthood: A Longitudinal Analysis of Add Health Data

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Abstract

Background

Cardiovascular disease (CVD) remains the leading cause of death in the United States, and the pathways underlying cardiometabolic dysfunction begin in childhood. Sleep is a core cardiovascular health behavior, yet most U.S. adolescents fall short of the recommended daily duration. Few studies have longitudinally linked adolescent sleep duration to a standardized, validated long-term CVD risk metric in a nationally representative cohort. We therefore examined the association between sleep duration in adolescence and CVD risk in adulthood and assessed whether the association was modified by sex.

Methods

We used data from Waves I and IV of the National Longitudinal Study of Adolescent to Adult Health (Add Health). The exposure was adolescent sleep duration at Wave I. The primary outcome was the 30-year Framingham risk score at Wave IV. Survey-weighted logistic regression was used to estimate adjusted odds ratios across sequential models adjusting for an a priori confounder set (age, race/ethnicity, parental education, family poverty, and physical activity). Effect modification by sex was tested. All analyses accounted for the Add Health complex survey design.

Results

The final analytic sample comprised 4,071 participants. 38% slept fewer than the recommended number of daily hours. Short sleep duration was associated with increased odds of high 30-year CVD risk (adjusted OR 1.31, 95% CI 1.04–1.65; p = 0.024; strengthening to OR 1.68 with age excluded), with no significant differences by sex (p = 0.59).

Conclusions

Short sleep duration in adolescence is associated with long-term CVD risk. Thus, incorporating adolescent sleep into primordial cardiovascular prevention efforts may improve cardiovascular health in adulthood.

Background

Cardiovascular diseases (CVDs) remain the leading cause of death globally, with U.S. incident cases nearly doubling from 34.7 million in 1990 to 66.8 million in 2021, and deaths are projected to reach 35.6 million annually by 2050. Total CVD-related healthcare costs are projected to exceed $2.8 trillion annually by 2050,(1–3) underscoring the urgent need to identify early-life, modifiable determinants of cardiovascular health.

Although overt CVD manifests in adulthood, the biological and behavioral pathways underlying cardiometabolic dysfunction begin much earlier, with atherosclerotic changes detectable by

adolescence.(4) Works by Barker and Fordsdahl, who linked deprived childhood conditions and impaired fetal growth to later cardiometabolic disease laid the foundation for the development of Life course epidemiology, and encouraged further studies into how early life factors influence cardiometabolic risk later in life.(5, 6) Longitudinal cohorts consistently show that high blood pressure, adiposity, and lipids track from childhood into adulthood, with cumulative exposure promoting the adult cardiometabolic pathophysiologic process.(7, 8)

Sleep is increasingly recognized as a major cardiovascular health determinant. The American Heart Association's Life's Essential 8 now includes sleep,(9) yet most U.S. adolescents fall short of recommended targets.(10, 11) National data show that approximately 7 in 10 high school students sleep fewer than 8 hours on school nights, with the proportion reporting insufficient sleep increasing from 68.9% to 76.8% between 2007 and 2023.(10, 11) Physiologic circadian phase delay during puberty, academic demands, screen exposure, and other social factors collectively drive chronic sleep restriction during adolescence.(10, 12)

Short sleep duration is linked to sympathetic nervous system dysregulation, systemic inflammation, insulin resistance, and elevated cardiovascular risk in adults.(13) In adolescents, cross-sectional studies have linked shorter sleep duration to greater BMI and waist circumference, and poorer sleep quality to hypertension and elevated non-HDL cholesterol.(14) Such designs, however, cannot establish whether adolescent sleep precedes cardiovascular risk later or merely occurs with it. To address this, a sibling-comparison study using Add Health found that adolescents who slept six or fewer hours per night had significantly greater cardiometabolic risk 13 years later. The association persisted in sibling fixed-effects models that excluded shared family confounding, but was sex-specific, remaining robust among males while attenuating to null among females once controlling for family background.(15)

Most adolescent studies examining sleep and cardiovascular health are cross-sectional. Few studies have longitudinally evaluated adolescent sleep duration against standardized, validated cardiovascular risk metrics in a nationally representative U.S. cohort or formally examined whether sex modifies this association.(12, 16, 17) This study addresses both gaps using longitudinal data from the National Longitudinal Study of Adolescent to Adult Health (Add Health),(18) with findings that could support adolescent sleep interventions as components of primordial CVD prevention.

Methods

Data source and study sample

This study used secondary data from Add Health, the largest nationally representative longitudinal study of U.S. adolescent health, spanning six waves from 1994 to 2025.(18) Add Health enrolled adolescents in grades 7–12 from 1994-95 using a stratified, school-based cluster design with oversampling of racial/ethnic minorities. Data were collected through in-home interviews administered by trained field staff at Wave I (1994/1995), with follow up surveys and or biomedical examinations at Wave II (1996),

Wave III (2001–2002), Wave IV (2008), Wave V (2016–2018), and Wave VI (2022–2025).(18–24) For this study, publicly available Wave I and Wave IV in-home interview files, together with the Wave IV public-use Cardiovascular and Anthropometric Measures biomarker files, were downloaded from the Add Health public-use data repository at the Carolina Population Center and merged on the unique respondent identifier (AID). The public-use dataset (N = 6,504) represents a random one-third subsample of the full Add Health cohort (20,745).

The analytic sample included participants who completed the Wave I in-home interview during adolescence and the Wave IV biomedical examination in young adulthood (ages 24–32 years), with available data on sleep duration and cardiometabolic factors needed to calculate the CVD risk score. Participants who did not respond to Wave IV, who had missing parental data for estimating socioeconomic status, and who were pregnant during Wave IV were excluded.

Primary outcome (Wave IV)

30-year Framingham risk score (FRS)

The primary outcome is the 30-year Framingham CVD risk score (FRS), a validated Cox-proportional-hazards-based algorithm that predicts the risk of CVD events (myocardial infarction, fatal or nonfatal stroke, angina pectoris, congestive heart failure, and coronary death) occurring within 30 years, while accounting for other causes of death.(25, 26) Following prior application of this score in this same cohort, the FRS was calculated using an existing SAS macro based on the following self-reported and objectively measured CVD risk factors, all assessed at Wave IV: age (continuous), sex (male/female), body mass index (continuous, calculated from measured weight and height), smoking status (yes/no, from self-reported cigarette use in the past 30 days), diabetes status (yes/no, from self-reported physician-diagnosed diabetes), systolic blood pressure (continuous, mmHg), and use of antihypertensive medication (assumed based on participants who self-reported doctor-diagnosed high blood pressure at Wave IV since Add Health does not directly report antihypertensive use at Wave IV).(20, 26) The primary outcome was dichotomized as “low risk” (FRS < 20%) and “high risk” (FRS ≥ 20%), consistent with cut-offs used in prior research and existing American College of Cardiology and American Heart Association’s clinical guidelines.(26–28)

Exposure (Wave I)

Sleep duration

Usual sleep duration, the primary exposure, was derived from the Wave I question "How many hours of sleep do you usually get?" and recategorized per the American Academy of Sleep Medicine guidelines as short (< 8 hours/night), recommended (8–10 hours/night, reference), or long (> 10 hours/night). (19, 29)

Covariates

Following a review of the literature, the following Wave I variables, selected a priori were controlled for in the analyses: Age (continuous, years) self-reported at Wave I. Age is associated with both sleep and cumulative cardiovascular risk.(17) Sex was coded as “male” or “female”. Race/ethnicity was categorized as “White”, “Black or African American”, “Hispanic”, or “Other”. Prior studies have shown that race/ethnicity is patterned by structural factors that independently affect both sleep and cardiovascular risk.(17, 30) Adolescent socioeconomic status, operationalized as two separate Wave I parent-reported measures: (1) parental education, recategorized as “less than high school”, “high school/GED”, “less than college degree”, or “college degree or above”; and (2) family poverty, a binary indicator derived from an affirmative parental response to any of the questions about the receipt of social benefits, food stamps, supplemental security income, unemployment compensation, social security, or aid to families with dependent children.(17, 31) Physical activity was operationalized based on responses to the following questions which assess moderate-to-vigorous physical activity (MVPA) at Wave I: “During the past week, how many times did you (i) go roller-blading, roller-skating, skateboarding, or bicycling; (ii) play an active sport, such as baseball, softball, basketball, soccer, swimming, or football; and (iii) exercise, such as jogging, walking, karate, jumping rope, gymnastics or dancing?”. In accordance with prior studies and national guidelines, physical activity was categorized as highly active (≥ 5 MVPA sessions/week), active (1–4 MVPA sessions/week) or inactive (0 MVPA sessions/week).(32, 33)

Statistical analyses

Descriptive analyses reported weighted frequencies and percentages for categorical variables and weighted means for continuous variables, for the full analytic sample overall and stratified by sleep duration category. Differences across sleep categories were formally tested using survey-adjusted Rao-Scott chi-square tests (categorical variables) and survey-adjusted linear regression or ANOVA (continuous variables). Statistical significance was set at a two-sided α of 0.05, and the corresponding test statistics and p-values were reported. Survey-weighted logistic regression was used to estimate unadjusted odds ratios (OR) and adjusted odds ratios (AORs) for high CVD risk (FRS ≥ 20%) with sleep duration category as the primary exposure and recommended sleep (8–10 hours/night) as the reference group. Three sequential models were fit: Model 1 (unadjusted); Model 2 (partially adjusted for age and race/ethnicity); and Model 3 (fully adjusted for the complete a priori confounder set described above: age, race/ethnicity, parental education, family poverty, and physical activity). We additionally examined each covariate using a change-in-estimate criterion, considering a covariate an influential confounder if its removal changed the sleep-CVD odds ratio by 10% or more. Effect modification by sex was evaluated by adding a multiplicative interaction term (sleep category × sex) to the fully adjusted model from Aim 1 and assessed its statistical significance at a two-sided α of 0.05. Sex-stratified AORs were reported. We retained sex in the final fully adjusted model as a potential confounder if it did not meet the threshold for effect modification. Because age at Wave IV, an input to the Framingham Risk Score used to calculate the outcome, is strongly correlated with age at Wave I, which is included as a covariate, we conducted a sensitivity analysis re-estimating the fully adjusted model (Model 3) with age removed as a covariate

(Model 3^) to evaluate whether adjusting for age would distort the sleep-CVD association. Potential selection bias due to loss to follow-up was assessed using two attrition sensitivity analyses comparing (1) baseline characteristics between participants included in the final analytic sample and those excluded for any reason, and (2) Wave IV completers and noncompleters. Statistical significance for all odds ratios was assessed at a two-sided α of 0.05. All analyses accounted for the Add Health's stratified, clustered sampling design and used the Wave IV grand-sample cross-sectional weight, following Add Health analytic guidelines.(34) We used domain statements instead of deleting participants based on our exclusion criteria to maintain the survey design elements. All analyses were conducted using SAS 9.4 procedures appropriate for complex survey data.

Results

Analytic sample

Of the 6,504 adolescents in the Wave I public-use sample, 5,114 (78.6%) participated in the Wave IV study. After applying our exclusion criteria, the final analytic sample (Fig. 1) comprised 4,071 participants, representing a national sample of 17,590,432 following the application of appropriate survey weights.

Baseline characteristics

The baseline characteristics of participants are presented overall, and by sleep category in Table 1. In the survey-weighted sample, 37.7% reported short sleep (< 8 hours/night), 59.6% recommended sleep (8–10 hours/night), and 2.7% long sleep (> 10 hours/night). The mean age of participants at Wave I was 15.4 years (SE = 0.1). Most of the study sample were males (52.3%), White (71.8%), and were highly active (65.7%). Wave I characteristics differed significantly across sleep duration categories, with the exception of sex ($p = 0.43$). Short sleepers were, on average, older than recommended sleepers (mean age 16.0 vs 15.0 years; $p < 0.0001$) and differed by race/ethnicity ($p = 0.0002$), parental education ($p = 0.02$), family poverty ($p = 0.03$), and physical activity ($p = 0.02$). High 30-year CVD risk (FRS ≥ 20%) at Wave IV was observed in 15.6% of the overall weighted sample. The prevalence differed significantly across adolescent sleep duration categories ($p = 0.0005$), with the highest prevalence among short sleepers (19.2%), followed by long sleepers (16.0%), and the lowest prevalence among recommended sleepers (13.4%).

Table 1
Baseline (Wave I) Characteristics of Add Health Participants Overall and by Adolescent Sleep Duration Category

Characteristic	All	Short Sleep (< 8 h)	Recommended Sleep (8–10 h)	Long Sleep (> 10 h)	p-value
Unweighted, N	4071	1535	2426	110	
Total weighted, %	100	37.7	59.6	2.7	
Continuous variables, Mean (SE)					
Age, years	15.4 (0.1)	16.0 (0.1)	15.0 (0.1)	14.7 (0.2)	**< .0001**
Categorical variables, % (SE)					
Sex					0.4318
Male	52.3 (1.1)	50.7 (1.8)	53.2 (1.2)	54.6 (5.7)	
Female	47.7 (1.1)	49.3 (1.8)	46.8 (1.2)	45.4 (5.7)	
Race/ethnicity					**0.0002**
White	71.8 (2.8)	68.3 (3.2)	74.2 (2.7)	66.8 (6.7)	
Black or African American	14.5 (2.1)	17.8 (2.7)	12.2 (1.9)	18.0 (4.9)	
Hispanic	10.3 (1.6)	9.2 (1.7)	11.1 (1.7)	9.5 (2.6)	
Other	3.4 (0.6)	4.7 (0.9)	2.5 (0.5)	5.7 (3.9)	
Parental Education					**0.0206**
Less than High School	10.5 (1.2)	9.8 (1.3)	10.5 (1.2)	19.5 (6.5)	
High School/GED	26.3 (1.4)	25.3 (1.9)	26.7 (1.6)	32.2 (5.6)	
Some College	30.4 (1.1)	30.9 (1.6)	30.1 (1.2)	30.8 (5.2)	
College Degree or Above	32.7 (1.9)	34.0 (2.3)	32.7 (1.9)	17.5 (4.5)	

SE: Standard error. Differences across sleep duration categories tested using Rao-Scott chi-square tests (categorical variables) and survey-weighted ANOVA (continuous variable).

Characteristic	All	Short Sleep (< 8 h)	Recommended Sleep (8–10 h)	Long Sleep (> 10 h)	p-value
Family Poverty					**0.0306**
Yes	16.5 (1.4)	16.7 (1.6)	15.8 (1.4)	27.4 (6.6)	
No	83.5 (1.4)	83.3 (1.6)	84.2 (1.4)	72.6 (6.6)	
Physical Activity					**0.0173**
Highly Active	65.7 (1.1)	63.2 (1.4)	67.2 (1.4)	67.9 (5.4)	
Active	29.6 (1.0)	30.6 (1.5)	29.1 (1.3)	26.0 (4.8)	
Inactive	4.7 (0.4)	6.2 (0.7)	3.7 (0.4)	6.1 (2.3)	
30-year CVD risk					**0.0005**
High CVD risk (FRS ≥ 20), %	15.6 (0.9)	19.2 (1.2)	13.4 (1.0)	16.0 (4.6)	
Low CVD risk (FRS < 20), %	84.4 (0.9)	80.8 (1.2)	86.6 (1.0)	84.0 (4.6)	

SE: Standard error. Differences across sleep duration categories tested using Rao-Scott chi-square tests (categorical variables) and survey-weighted ANOVA (continuous variable).

Associations between adolescent sleep and 30-year CVD risk

In unadjusted survey-weighted logistic regression analysis, short sleep duration was associated with 54% greater odds of high CVD risk relative to recommended sleep duration and the effect was significant (OR 1.54, 95% CI 1.26–1.87, $p < 0.0001$), whereas long sleep was not associated with higher odds of high CVD risk (OR 1.24, 95% CI 0.64–2.40) (Table 2). Adjustment for age and race/ethnicity (Model 2) attenuated the short-sleep association to nonsignificance (adjusted OR 1.19, 95% CI 0.97–1.47), coinciding with short sleepers being on average older than recommended sleepers and age being strongly associated with the outcome (adjusted OR 1.33, 95% CI 1.25–1.42). Because age at Wave IV is also a direct input to the Framingham risk score, we examined this attenuation further in a prespecified sensitivity analysis (below).

Table 2
Odds Ratios (OR) for High versus Low CVD Risk (FRS ≥ 20%) at Wave IV

Predictor (referent)	**Model 1** **OR (95% CI)**	**Model 2** **OR (95% CI)**	**Model 3** **OR (95% CI)**
Sleep Duration			
Recommended (ref)	-	-	-
Short Sleep (< 8 h)	**1.54 (1.26–1.87)***	1.19 (0.97–1.47)	**1.31 (1.04–1.65)[a]**
Long Sleep (> 10 h)	1.24 (0.64–2.40)	1.38 (0.70–2.73)	1.31 (0.70–2.45)
Age	-	**1.33 (1.25–1.42)***	**1.33 (1.24–1.43)***
Race			
White (ref)	-	-	-
Black/African American	-	1.06 (0.78–1.43)	1.05 (0.75–1.46)
Hispanic	-	0.91 (0.66–1.25)	0.80 (0.54–1.19)
Other	-	0.87 (0.44–1.73)	0.83 (0.44–1.57)
Sex			
Male (ref)	-	-	-
Female	-	-	**0.15 (0.11–0.19)***
Parental Education			
College Degree or Above (ref)	-	-	-
Less than High School	-	-	**1.63 (1.01–2.62)[b]**
High School/GED	-	-	**1.32 (1.01–1.73)[b]**
Some College	-	-	1.10 (0.80–1.50)
Family Poverty			
No (ref)	-	-	-
Yes	-	-	1.11 (0.82–1.51)
Physical Activity			
Highly Active (ref)	-	-	-

OR: odds ratio; CI: confidence interval. Model 1: unadjusted. Model 2: adjusted for age and race/ethnicity. Model 3: fully adjusted for age, race/ethnicity, parental education, family poverty, physical activity, and sex.*P-value < 0.0001.[a]P-value = 0.0236. [b]P-value = 0.04. Bolded OR and CI denote statistical significance.

Predictor (referent)	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
Active	-	-	1.04 (0.82–1.33)
Inactive	-	-	0.79 (0.48–1.31)

OR: odds ratio; CI: confidence interval. Model 1: unadjusted. Model 2: adjusted for age and race/ethnicity. Model 3: fully adjusted for age, race/ethnicity, parental education, family poverty, physical activity, and sex.*P-value < 0.0001.[a]P-value = 0.0236. [b]P-value = 0.04. Bolded OR and CI denote statistical significance.

In the fully adjusted model (Model 3), which additionally controlled for parental education, family poverty, physical activity, and sex, short sleep duration was significantly associated with high 30-year CVD risk (adjusted OR 1.31, 95% CI 1.04–1.65; p = 0.024) but long sleep remained nonsignificant (adjusted OR 1.31, 95% CI 0.70–2.45). Sex was the strongest independent predictor of the outcome (female vs male OR 0.15, 95% CI 0.11–0.19). Compared with parents with a college degree or higher, parental with a high school/GED (adjusted OR 1.32, 95% CI 1.01–1.73) or less than high school education (adjusted OR 1.63, 95% CI 1.01–2.62) were significantly associated with high 30-year CVD risk.

Effect modification

The multiplicative interaction between sleep and sex added to the fully adjusted model was not statistically significant (Wald test F = 0.53, p = 0.59), providing no evidence that the sleep-CVD association differed by sex. In sex-stratified models (Table 3), the short-sleep association was significant among males (adjusted OR 1.38, 95% CI 1.07–1.78) but not among females (adjusted OR 1.09, 95% CI 0.67–1.78); with overlapping confidence intervals, long sleep was non-significant in both males and females. Because the interaction did not meet the prespecified significance threshold, the interaction term was dropped, and sex was retained as a confounder in the fully adjusted model.

Table 3
Association Between Adolescent Sleep Duration (Wave I) and CVD Risk (High vs Low at Wave IV) Stratified by Sex

Sleep Duration	Males* AOR (95% CI) (N = 1947)	Females* AOR (95% CI) (N = 2124)
Recommended Sleep (= Ref)	-	-
Short Sleep (< 8 h)	1.38 (1.07–1.78)	1.09 (0.67–1.78)
Long Sleep (> 10 h)	1.50 (0.73–3.08)	0.60 (0.12–2.98)

AOR: adjusted odds ratio; CI: confidence interval. Ref: reference*All models fully adjusted for age, race/ethnicity, parental education, family poverty, and physical activity.

Sensitivity analyses

Because age at Wave IV is a direct input to the Framingham risk score, we conducted a sensitivity analysis re-estimating the fully adjusted model with age removed as a covariate (Model 3^, Supplementary Table 1). Excluding age strengthened the short-sleep association considerably, from an OR of 1.31 (95% CI 1.04–1.65) in the age-adjusted model to an OR of 1.68 (95% CI 1.34–2.09, $p < 0.0001$) with age removed. The estimates for the remaining covariates (race/ethnicity, sex, parental education, family poverty, and physical activity) were similar between the two models. Two attrition comparisons were examined. First, compared with participants excluded from the analytic sample for any reason, included participants were more often White and had higher parental education and lower family poverty (all $p < 0.001$), and differed modestly in physical activity ($p = 0.02$). However, the included and excluded groups did not differ in the distribution of the exposure, adolescent sleep duration ($p = 0.53$), or sex ($p = 0.24$) (Supplementary Table 2). Second, comparing Wave IV completers to noncompleters specifically, completers differed from noncompleters on race/ethnicity, parental education, family poverty, sex, and sleep duration (all $p < 0.05$), but not in physical activity ($p = 0.63$) (Supplementary Table 3).

Discussion

We examined the association between adolescent sleep duration and CVD risk in young adulthood, and whether this association varies by sex. In this nationally representative cohort, about 38% of the adolescents reported sleeping less than the recommended number of hours, and short sleep duration was associated with significantly higher odds of elevated 30-year CVD risk in adulthood. This association strengthened further when age was excluded from the model as a sensitivity analysis, suggesting that the primary estimate may understate the true magnitude of the relationship. Long sleep duration was not significantly associated with CVD risk in any model, and the sleep-by-sex interaction was not statistically significant.

Our findings agree with results from prior studies linking short sleep to cardiometabolic dysfunction through pathways such as sustained sympathetic activation, systemic inflammation, insulin resistance, hypertension and elevated low density lipoprotein(LDL) cholesterol.(7, 8, 13, 14) Because more than one-third of adolescents in this sample reported fewer than the recommended hours of sleep, even a modest independent association between short sleep and CVD risk is relevant at the population level given how common the exposure is. Adjustment for age considerably attenuated the crude association: short sleepers were, on average, nearly a year older than recommended sleepers, and age was a strong independent predictor of the outcome. The association remained significant after adjusting for age, suggesting that age-related confounding does not fully explain the relationship and that other residual confounders (such as diet, family history, insomnia, and sleep apnea) or mechanisms may also be contributing.

Our formal test to examine sex as an effect modifier was not statistically significant, so we cannot conclude that the sleep-CVD association truly differed by sex in this analysis. Sex was therefore retained in the final models as a confounder rather than an effect modifier. That said, in stratified models the short-sleep association was significant among males but not females, a pattern consistent with prior

work: a sibling fixed-effects study found the sleep-cardiometabolic risk association remained significant among males but attenuated to null among females once shared family background was controlled for,(15) and separate work examining adolescent sleep health and cardiovascular risk in adulthood reported similar sex-specific patterns.(17) Our findings may reflect limited statistical power or a true absence of interaction. We speculate that hormonal differences, such as the relatively protective cardiovascular effect of estrogen in premenopausal women,(35) may contribute to a true sex difference, but this explanation was not directly tested in our data.

The finding that an association was detectable in a 30-year risk score anchored in young adulthood suggests that adolescent sleep may contribute to cardiometabolic trajectories that are already discernible by the third decade of life, reinforcing adolescence as a window for primordial cardiovascular prevention. Because sleep duration is a modifiable behavior, these findings support incorporating brief sleep screening into routine adolescent well-visits and considering sleep alongside diet and physical activity in pediatric cardiovascular risk counseling. Population-level strategies such as delayed school start times may also be relevant, although this study cannot directly evaluate their effectiveness.

The two attrition comparisons yielded different conclusions regarding the exposure. Compared with excluded participants, sleep duration and sex were not significantly associated with inclusion; however, when Wave IV completers were compared with noncompleters specifically, both sleep duration and sex were significantly associated with completion, although the absolute differences were small (38.4% vs 36.2% short sleep among completers vs non-completers). This discrepancy means that selective attrition related to the exposure cannot be fully ruled out, although the modest magnitude of the difference suggests that any resulting selection bias is likely small. Race/ethnicity, parental education, and family poverty were associated with attrition in both comparisons, so the analytic sample is not fully representative of the original Wave I cohort in terms of these characteristics. Confounding was addressed through sequential, a priori-specified adjustment; when the crude model was compared with the fully adjusted model, adjustment for age and race/ethnicity produced the largest change in the sleep-CVD odds ratio. The persistence of a significant association after full adjustment indicates that confounding by the measured covariates does not fully explain the observed relationship, although residual confounding by unmeasured factors, such as genetic predisposition or shared family environment, remains possible.

We considered the inclusion of substance use and depressive symptoms in our a priori confounding set. Substance use and depressive symptoms are reciprocally related to sleep duration and have been linked to cardiovascular risk, raising the question of whether they should be treated as confounders.(17, 36) Short sleep durations may plausibly precede and contribute to substance use and depressive symptoms, placing these factors on the causal pathway to later cardiovascular risk.(36) Because adjusting for mediators introduces overadjustment bias, substance use and depressive symptoms were excluded as covariates.

Outcome misclassification was partially, but not fully, minimized: systolic blood pressure and BMI were measured directly at Wave IV, but smoking status, diabetes diagnosis, and antihypertensive medication use, also inputs to the Framingham risk score, were based on self-reports. As such, some nondifferential misclassification of the outcome likely remains. Because sleep duration was also self-reported, exposure misclassification is also possible; as this misclassification is likely nondifferential with respect to an outcome measured years later, it would be expected to bias estimates toward the null rather than create a spurious association.

The sensitivity analysis removing age as a covariate provides additional insight into this issue. Removing age substantially strengthened the short-sleep association (OR 1.68 vs. 1.31 with age included), whereas the other covariate estimates largely unchanged. This pattern is consistent with our a priori concern that age at Wave I, being highly correlated with the Wave IV age used to calculate the outcome, may partly suppress the sleep-CVD association when included as a covariate, rather than purely correcting for confounding. We retained Model 3 (with age) as our primary, prespecified analysis, since Wave I age remains a substantively defensible confounder of the sleep-CVD relationship independent of its role in the outcome calculation. The true association may lie between these two estimates, and the age-adjusted OR of 1.31 should be interpreted as a conservative lower bound rather than a definitive effect size.

Strengths and limitations

This study uses a large, nationally representative cohort followed from adolescence into young adulthood, allowing temporal ordering between the exposure and outcome. The outcome was calculated using a validated, widely used long-term CVD risk prediction score rather than an ad hoc composite cardiovascular risk score. Because age at Wave IV is a direct input to the outcome, we conducted a sensitivity analysis re-estimating the fully adjusted model with age removed, allowing readers to see how sensitive the sleep-CVD association is to this methodological choice. All analyses accounted for Add Health's complex survey design using Domain statements rather than deleting excluded participants outright, preserving the stratification and clustering structure needed for valid variance estimation.

Some limitations of this study should also be acknowledged. First, sleep duration is self-reported and therefore subject to recall bias. However, this exposure misclassification is likely nondifferential with respect to the outcome, and it is expected to bias the estimates toward the null and attenuate the real effect sizes of the sleep-CVD association. Second, long sleep duration was reported by a very small proportion of the sample (2.7%, unweighted n = 110), and the corresponding wide confidence intervals for its association with the outcome (OR 1.31, 95% CI 0.70–2.45) in the fully adjusted model indicate that these estimates are imprecise. Third, this study reports only one dimension of sleep. Sleep is a multidimensional factor that includes sleep duration, sleep quality, and the presence of conditions that affect sleep such as insomnia, among other dimensions of sleep health. Using only sleep duration may not completely characterize sleep health. Fourth, only baseline sleep at Wave I was measured. Sleep patterns may change over time which could affect long-term CVD risk. Fifth, the risk algorithm used to

calculate the outcome was originally derived from the Framingham Offspring cohort, a population that was predominantly White and assessed in the 1970s. Given the greater racial and ethnic diversity of the Add Health sample, recalibration of this tool may be needed to better predict CVD risk in this population. However, this concern is partially mitigated by this study's focus on the relative association between sleep and risk rather than on absolute risk estimates. Finally, as with any observational secondary analysis, residual confounding by unmeasured factors (e.g., genetic predisposition, detailed dietary intake, family, and medical history) cannot be entirely ruled out.

Future studies could benefit from more granular measures of sleep health beyond duration alone, including sleep quality, regularity, and diagnosed sleep disorders such as insomnia. Designs with greater power to formally test for sex-specific effects, such as the full Add Health dataset (20,745 participants) rather than the one-third public use dataset would help clarify whether the sex pattern observed here indicates a true difference or reflects limited precision. Replication using directly measured lipid panels to calculate the Framingham risk score, rather than the BMI-substituted version used here, would also help determine whether the association is sensitive to this methodological choice.

Conclusions

Short sleep duration in adolescence is an important predictor of long-term CVD risk. Thus, incorporating adolescent sleep into primordial cardiovascular prevention efforts may improve cardiovascular health in adulthood.

Abbreviations

Add Health: National Longitudinal Study of Adolescent to Adult Health

AID: Add Health unique respondent identifier

ANOVA: Analysis of variance

AOR: Adjusted odds ratio

BMI: Body mass index

CI: Confidence interval

CVD: Cardiovascular disease

FRS: Framingham Risk Score

GED: General Educational Development

IRB: Institutional Review Board

LDL: Low-density lipoprotein

MVPA: Moderate-to-vigorous physical activity

OR: Odds ratio

SAS: Statistical Analysis System

SD: Standard deviation

SE: Standard error

Declarations

Ethics approval and consent to participate

The University of North Carolina, Chapel Hill institutional review board approved the original Add Health study, with formal written informed consent obtained from participating adolescents and parents. Since this current study used publicly available, de-identified data, it was considered exempt from IRB approval by The George Washington University.

Consent for publication

Not applicable.

Availability of data and materials

The datasets analyzed in the current study are available in the Add Health public-use data repository, Carolina Population Center, University of North Carolina at Chapel Hill (https://addhealth.cpc.unc.edu), and are available on agreeing to a data use agreement.

Competing interests

The authors declare that they have no competing interests.

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Author's contributions

MA conceived and designed the study, conducted the statistical analysis, interpreted the data, and drafted the manuscript. SQ supervised the study, contributed to data interpretation, and critically reviewed and suggested revisions of the manuscript. All authors read and approved the final manuscript.

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Figures

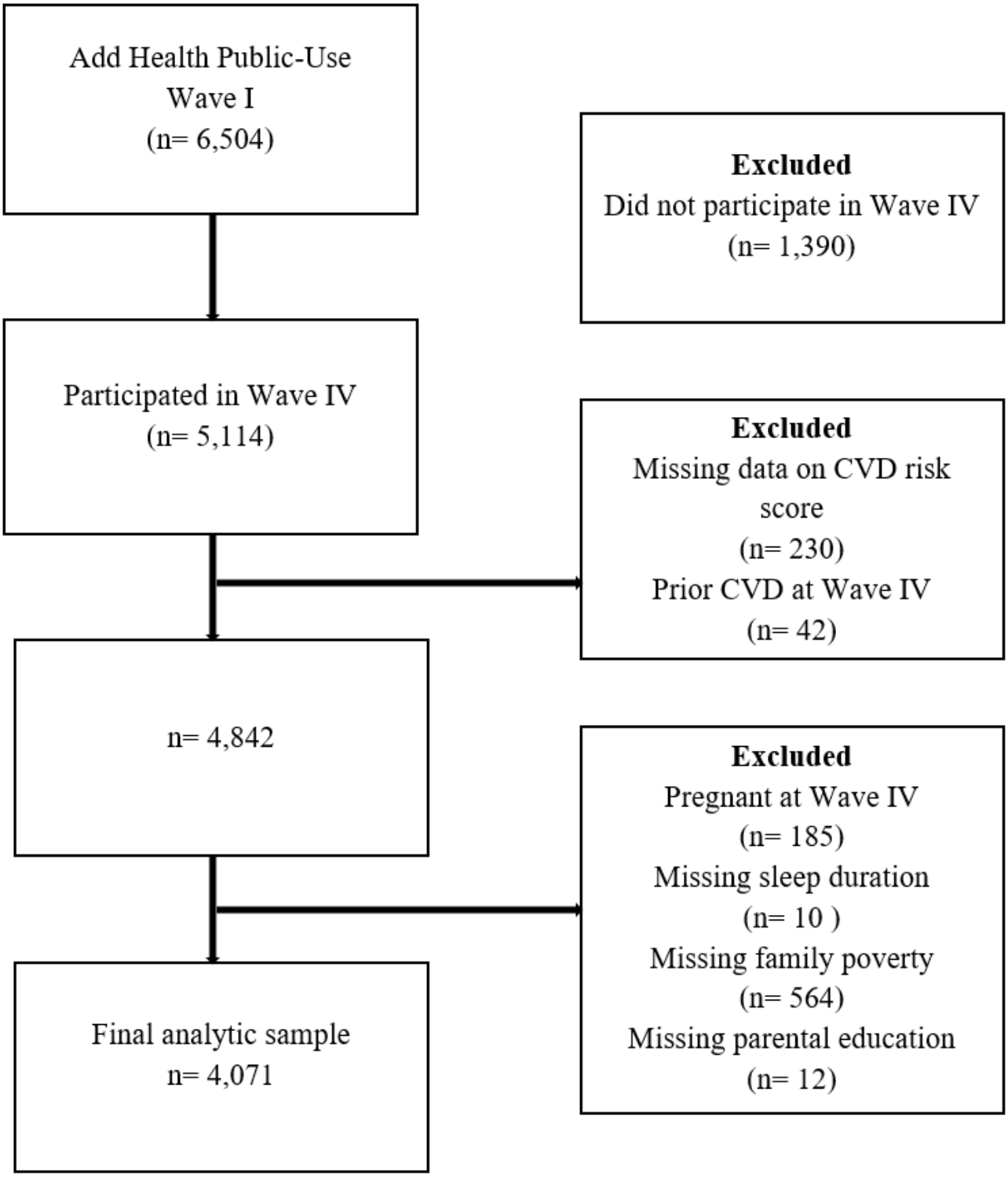


Figure 1

Flow chart showing the final analytic sample used in the study

CVD- cardiovascular disease

Supplementary Files

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