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Sleep duration and body mass: Direction of the associations from adolescence to young adulthood

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Abstract

Research suggests that sleep duration and obesity are related, but the direction of this association remains uncertain. We applied autoregressive cross-lag models to evaluate the directionality of the relationship between sleep duration and BMI from adolescence through emerging and young adulthood, life stages where the risk for developing obesity are particularly high. Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), we examined sex-stratified associations between sleep duration and BMI in this cohort from adolescence (ages 12–18, year 1996), to emerging adulthood (ages 18–24, 2001–2002), to young adulthood (ages 24–32, 2008), controlling for key confounders. For both males and females, higher BMI during an earlier developmental stage was associated with shorter sleep duration in the subsequent stage (both *B*s= −0.02, *p*s<0.01). However, sleep duration at an earlier developmental stage was not associated with BMI at the subsequent stage. Findings suggest that researchers should be cautious when interpreting cross-sectional relationships between sleep and BMI, as higher BMI may precede shorter sleep during adolescence to young adulthood. Researchers may also wish to account for potential bi-directional associations when modeling sleep and BMI using longitudinal data.

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Introduction

In the United States, nearly one third of children and more than two thirds of adults have overweight or obesity (1, 2), a risk factor for chronic diseases (3). A growing body of evidence suggests sleep duration may be an important factor in the development of obesity. However, much of the evidence is based on cross-sectional research and research designed to examine how poor sleep behaviors may impact obesity risk (4). In fact, the directionality of the relationship between sleep duration and obesity risk is unclear (5) and it is plausible that obesity predisposes one to poor sleep (6). Autoregressive cross-lag (ARCL) models may help disentangle the directionality of the relationship between sleep and body mass by examining how these variables move together through time without specifying which construct is the primary independent and dependent variable (7).

To understand the potential bi-directional relationship between sleep and body mass, we applied ARCL models to examine longitudinal associations between sleep and body mass index (BMI) in a national sample followed in adolescence, emerging adulthood, and young adulthood—three key life stages for the development of obesity (8, 9).

Methods

Study Sample

We used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; www.cpc.unc.edu/projects/addhealth), a nationally representative sample of 20,745 individuals recruited from middle and high schools in 1994–95 and followed into adulthood. We used data from Wave II (collected in 1996, ages 12–18), Wave III (2001–2002, ages 18–24), and Wave IV (2008, ages 24–32), the three waves in which BMI was objectively measured.

Measures

We used measured height and weight from Waves II-IV to calculate respondent BMI at each wave. In Wave II, respondents self-reported usual sleep duration in hours per night. In Waves III and IV, respondents reported usual bedtime and waketime for work/school days and nonwork/non-school days; from those responses we calculated sleep duration for work and nonwork days in decimal hours (e.g., thirty minutes equals 0.5 decimal hours), then calculated a weighted average for usual sleep duration. Similar methods for self-reporting sleep have demonstrated strong validity when compared to objective measures including actigraphy (10).

We included variables that might confound the relationship between sleep and BMI as covariates. We controlled for depressive symptoms (11) at each developmental stage with a latent variable comprised of five items from the Center for Epidemiologic Studies Depression Scale, as previous analyses have found these items (or similar items) have stronger measurement invariance across racial/ethnic groups compared to the full 19 items (12). Additionally, we included the following time-invariant controls: age and pubertal status at study onset; race/ethnicity; parent education; birthweight; exclusive breastfeeding for 6+ months; and obesity in the biological mother or father (1, 13–15).

Statistical Analysis

Our analytic sample included respondents with complete information on key variables, excluding pregnant women. We excluded the most extreme 1% of the sample for sleep duration to omit implausible values (specifically, less than 4 hours per night and greater than 12 hours [Wave II] or 20 hours [Wave III & IV] per night). Because BMI was assessed by study staff, all measured BMI were within a plausible range, and so we did not omit any extreme BMI cases.

To evaluate the relationship between sleep and BMI across developmental stages, we estimated autoregressive cross-lag (ARCL) models. ARCLs enable us to examine how two variables (i.e., sleep and BMI) move together through time without needing to specify which construct is the primary independent and dependent variable. The autoregressive nature of the ARCL allows the model to account for stability within constructs over time. The cross-lag nature of the ARCL allows us to establish temporality of investigated relationships. To obtain the best fitting ARCL, we conducted likelihood-ratio tests between models of increasing complexity.

Previous studies have found the relationship between sleep and BMI varies by sex (16); therefore, we conducted sex-stratified analyses. All analyses accounted for the complex survey design of Add Health (17). We conducted analyses using Mplus version 8. Mplus code for the final models are available as supplemental materials (Supplemental File 1 and 2). The University of North Carolina Chapel Hill Institutional Review Board granted human subjects' research approval for this study.

Results

The final analytic sample included 11,965 respondents (6,198 females; 5,767 males). We excluded 4,924 respondents who were missing information on predictors, and 515 respondents who were missing information on sleep and/or BMI; the excluded sample and analytic sample were similar on all covariates. Excluded respondents, on average, had a slightly higher BMI compared to the analytic sample. BMI and sleep duration increased over time for males and females (Table 1). Other descriptive statistics appear in Table 1.

Figure 1 presents final ARCL models, which demonstrated good fit to the data (e.g., CFI, TLI ≥ 0.95). Among both males and females, BMI was highly correlated across the three developmental periods; sleep duration was also correlated over time, although less strongly. Sleep during a previous developmental stage did not predict BMI in the subsequent stage for males or females. Higher BMI during the previous developmental stage, however, was associated with shorter sleep in the subsequent stage for both males and females (ps<0.01). The magnitude of the effect, however, was modest: Bs=−0.02, SEs=0.01. For example, an individual with a BMI of 26 kg/m^2 in adolescence would be expected to sleep 1.2 fewer minutes during emerging adulthood compared to an individual with a BMI of 25 kg/m^2 in adolescence.

Because depressive symptoms may serve as a mediator—rather than a confounder—of the relationship between sleep and BMI, we conducted sensitivity analyses that removed this

covariate effect. Results were robust to this omission; the same pattern of significance and directionality emerged.

Discussion

This is one of the first studies to examine the directionality of the relationship between sleep and body mass in the transition between adolescence, emerging adulthood, and young adulthood. We found that BMI during an earlier developmental stage was negatively associated with sleep duration in the subsequent stage for both males and females. We did not find, however, an association between sleep at an earlier developmental stage and BMI at the subsequent stage.

Previous research has, to a large extent, assumed that inadequate sleep is the exposure leading to increased risk of obesity (4, 11). Speculation regarding the mechanism has included the possibility that poor sleep makes one feel tired, resulting in lower activity levels and less energy expenditure; alternately, poor sleep may result in more time awake, providing more opportunity to eat and increasing energy intake (18). In contrast, our findings suggest that BMI may be an exposure that precedes inadequate sleep, or some other cause of inadequate sleep not captured in the present study. The mechanism (or mechanisms) for how BMI may precede sleep patterns remain unknown. These mechanisms may include psychological factors such as anxiety; however, the relationship between psychological factors and BMI remains unclear (19). For example, it could be that anxiety is an antecedent to both BMI and sleep, such that anxiety occurs with (or causes) higher levels of BMI among adolescents and impacts sleep later in life (20). Alternatively, overweight and obesity may give rise to anxiety, which may subsequently alter sleep patterns (20). In addition, hormones involved in sleep patterns, such a melatonin and cortisol, may be influenced by body weight (21, 22). Future longitudinal research is need to clarify potential mechanisms. These longitudinal analyses should account for potential bi-directional association when examining the relationship between sleep and obesity (or vice versa) with techniques from structural equation modeling, including the ARCL and multivariate latent curve model (7, 23).

Limitations to our study include that the Add Health data do not provide detailed diet or physical activity measures that would allow researchers to estimate energy balance, but diet and activity may impact both sleep and body weight. Additionally, sleep duration was assessed using self-report; there have been significant advances in sleep measurement since the Add Health study began. Our analysis controlled for depression at each life stage and a number of other time-invariant confounders such as parental weight status, but there may be other important confounders. Many previous studies have found a prospective relationship between sleep patterns and future body mass (4, 11, 24), and the present study does not discount this large body of evidence. Instead, our findings support the need for future research to also investigate the prospective relationship between body mass and future sleep patterns.

The present findings suggest that researchers should be cautious when interpreting cross-sectional relationships between sleep and BMI, as higher BMI may precede less sleep.

Researchers may also wish to account for these bi-directional associations when modeling sleep and BMI using longitudinal data.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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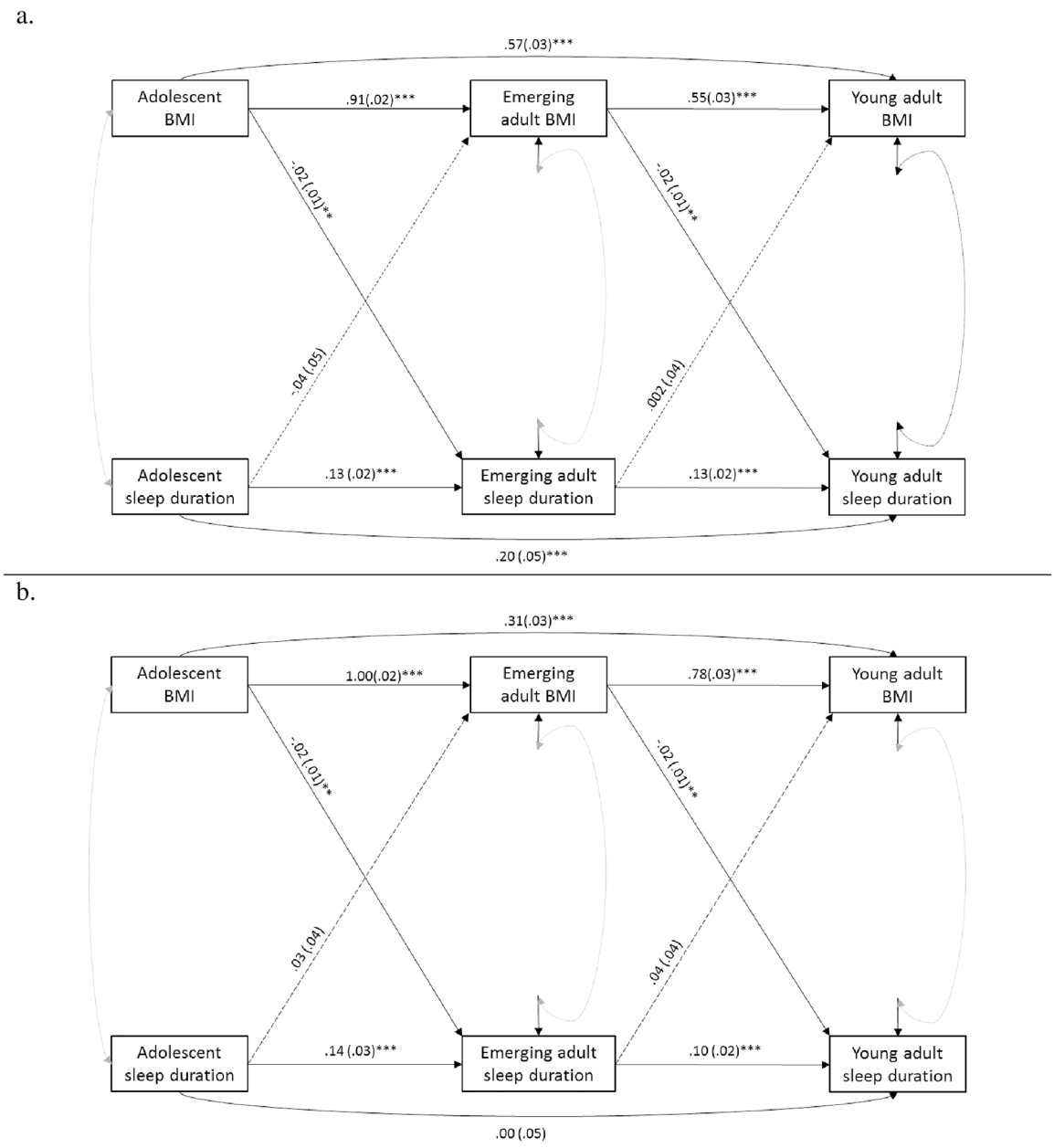

Figure 1.

Autoregressive cross-lag model of sleep and BMI for males (a) and females (b), National Longitudinal Study of Adolescent to Adult Health.

Notes: Covariates and error terms omitted for clarity. Dashed lines indicate cross-lag effects. Gray dotted lines indicate estimated correlations and residual correlations. Male model fit: χ^2 (385, N = 5767) = 948.22, p < 0.0001; RMSEA = 0.016 (0.015, 0.017); CFI = 0.969; TLI = 0.964. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Female model Fit: χ^2 (385, N = 6198) = 1104.29, p < 0.0001; RMSEA = 0.017 (0.016, 0.019); CFI = 0.974; TLI = 0.971. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 1.

Descriptive statistics of the sample from the National Longitudinal Study of Adolescent to Adult Health stratified by biological sex.

Variable	Males, $\bar{x}(sd)$ or %	Females, $\bar{x}(sd)$ or %
Adolescent BMI (kg/m^2)	22.9 (5.0)	22.8 (5.2)
Emerging adult BMI (kg/m^2)	26.3 (5.5)	26.3 (6.6)
Young adult BMI (kg/m^2)	28.9 (7.0)	28.8 (8.1)
Adolescent sleep (hours)	7.8 (1.3)	7.6 (1.3)
Emerging adult sleep (hours)	8.2 (2.3)	8.6 (2.2)
Young adult sleep (hours)	9.1 (2.7)	9.3 (2.6)
Parent with obesity	23.6%	24.3%
Birthweight (ounces)	122.0 (19.6)	116.9 (18.5)
Breastfed ≥ 6 months	20.8%	20.5%
Pubertal status (scale, 1–5)	3.2 (1.1)	3.3 (1.1)
Race/ethnicity		
White	70.6%	70.4%
Hispanic	10.4%	10.2%
Black	12.8%	14.1%
Other	6.2%	5.3%
Parent education		
< High school	10.0%	9.2%
High school	25.4%	27.7%
Some college	32.2%	30.9%
College graduate	32.4%	32.2%
n	5,767	6,198

Note: Pubertal status was measured with the question: How advanced is your physical development compared to other (boys/girls) your age? Higher values indicate more mature development.