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Assessing the longitudinal association between sleep, diet quality and BMI *z*-score among Black adolescent girls

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Summary

Background: Cross-sectional research has suggested associations between diet, sleep and obesity, with sparse longitudinal research.

Objectives: To identify longitudinal mechanistic associations between sleep, diet and obesity.

Methods: We used longitudinal data from a sample of Black adolescent girls. At T1 (enrolment), 6 months (T2) and 18 months (T3), we estimated sleep duration and quality (7-day accelerometry), diet quality (Healthy Eating Index [HEI-2020]) and body mass *z*-scores (zBMI) from measured height and weight. Longitudinal mediation using structural equation models examined the mechanistic roles of sleep, diet quality and zBMI.

Results: At enrolment, girls ($n = 441$) were mean age 12.2 years (±0.71), 48.3% had overweight/obesity, and mean HEI 55.8 (±7.49). The association between sleep and diet quality did not vary over time. Sleep duration at T1 was not associated with diet quality at T2 nor was diet associated with zBMI at T3. The bootstrapped indirect effect was not significant. Sleep quality at T1 was not associated with diet quality at T2 nor was diet associated with zBMI at T3. The bootstrapped indirect effect was not significant.

Conclusions: Diet was not a mediator between sleep and obesity. Study strengths are the longitudinal design and direct measures of sleep and zBMI among a homogeneous sample.

KEYWORDS
adolescence, diet quality, longitudinal design, mediation analysis, obesity, sleep

1 | INTRODUCTION

Obesity, a major public health challenge worldwide, disproportionally affects low-income urban communities of colour.[1] Low-quality diet, low physical activity levels, high sedentary behaviours (i.e., sitting for long periods and screen time)[2] and, recently, short sleep duration (<6 h/night) and night-time awakenings (a measure of sleep efficiency: percentage of time spent asleep vs. awake in bed) are the most proximal drivers of obesity.[3] In the United States, due to a history of structural racism,[4] socioeconomically disadvantaged adolescent girls often live in communities with high levels of deprivation (i.e., high poverty and unemployment rates),[5] experience disproportionally higher rates of obesity,[1] have lower diet quality[6] and physical activity levels,[7] report shorter sleep durations[8] and have poorer quality sleep compared with adolescents of other races/ethnicities and boys.

Examining the interplay of health behaviours and obesity risk is crucial among Black adolescent girls who are particularly vulnerable to lifelong cardiometabolic health disparities.[9] Non-Hispanic

Black individuals have a 34%–50% age-adjusted prevalence of obesity, which is a higher rate than any other racial/ethnic group.[10] Black women are also less likely to lose weight in weight loss interventions, compared to other marginalized subpopulations.[11] This increased obesity prevalence, in addition to a higher burden of modifiable risk factors and clinical comorbidities, contributes to increased rates of cardiovascular disease-related (CVD) mortality among Black women compared with White women.[12] CVD often manifests during younger ages for Black women (i.e., young and middle adulthood) compared to women in other racial/ethnic groups.[13]

Sleep duration and sleep quality are important dimensions of pediatric sleep health.[14] However, most middle school-age (60%) and high school-age adolescents (78%) report sleeping less than the recommended 8 h per night[15] and experience a sharp decline in sleep quality with age.[16] Recent systematic reviews and meta-analyses highlight how short sleep duration and poor sleep quality are prospectively associated with higher overweight/obesity risk in children and adolescents.[17–19] However, the explanatory mechanisms by which sleep duration affects weight gain are unclear.[3,17,19] A proposed mechanism is through poor diet quality.[3] Cross-sectional studies have shown associations between decreased sleep duration and higher energy-dense food intake, which may be seen as a normal physiological response to provide the energy needed for the brain to sustain additional wakefulness.[20] Restricted sleep might affect food intake, appetite traits and energy balance by modifying responses to hormones, such as decreased leptin and increased ghrelin levels.[3] To identify potential mechanisms between sleep, diet and body mass index (BMI), longitudinal studies are needed, particularly among Black adolescents and girls, who are at the highest risk for disparate health outcomes and under-represented in extant literature.[3,19]

Understanding the mechanisms for the development of obesity is especially needed in the transitional life stage of adolescence, where self-regulatory and lifestyle changes are colliding. At the biological level, adolescents experience diminishing sleep pressure and a circadian phase delay, making it more difficult to fall and stay asleep at night.[21] This risk for short sleep duration is further compounded by early school start times and a '24/7' lifestyle of competing social, extra-curricular and familial obligations making prioritization of health behaviours difficult.[22] This constellation presents a missed opportunity as adolescence may be a prime developmental period to address health behaviours given adolescent's emerging agency over their health and lifestyle choices.

Examination of the longitudinal interrelations between sleep and diet with obesity among a population at risk for racial health outcomes can identify the mechanism linkages and provide potential points for behavioural interventions. This study aimed to examine the longitudinal association between sleep, diet quality and BMI *z*-scores (zBMI) among Black adolescent girls. The present study hypothesizes that sleep quantity (duration) and quality (efficiency) influence adolescent girls' dietary intake, leading to increased zBMI. This paper addresses the following research questions:

1. How are sleep (quantity and quality), longitudinally related to diet quality and zBMI among Black adolescent girls?
2. Does diet quality mediate the sleep and zBMI longitudinal association?

2 | METHODS

2.1 | Study design and participants

We obtained longitudinal data from individuals participating in *Challenge! In Middle Schools*, a multilevel randomized controlled trial implemented in urban schools serving low-income communities. The trial aimed to enhance both diet and physical activity through a mentorship model and enrolled 22 middle schools in a large public school district with predominantly Black students (>70%) and the majority eligible for free or reduced-priced school meals (>75%). Sleep was not an intervention target, and there were no significant changes in zBMI, prevalence of obesity, body composition or diet quality due to the intervention. The intervention builds on a previous intervention conducted among early adolescent youth,[23] and spanned four years, from 2009 to 2013.

Briefly, within each school, recruitment targeted 20–40 adolescent girls in 6th and 7th grades who could participate in a physical education class and whose parent or caregiver was proficient in English. Recruitment efforts were conducted through mailings or in-person interactions during lunch shifts. A total of 789 girls were successfully enrolled in the study, and there were no specific weight-related inclusion or exclusion criteria.

2.2 | Data collection

Data collection occurred at three specific time points within each cohort (2009 to 2013): T1 (enrolment in the fall), T2 (6-month follow-up in the spring) and T3 (18-month follow-up in the spring). We collected data in-person during school days on diet (food frequency), accelerometry and anthropometry. Trained data collectors who were unaware of the intervention assignment conducted evaluations.

To participate, adolescent girls provided written informed consent, and their primary caregivers signed a written informed consent. The study protocol received approval from the Institutional Review Boards (HP-00040540) at both the University and the city public school system where the study was conducted.

2.3 | Diet quality

We estimated dietary quality using the 2020 Healthy Eating Index generated from the Youth/Adolescent Food Frequency Questionnaire (YAQ), an instrument developed and validated for use with adolescents.[24] The YAQ was a self-administered instrument, and girls reported foods consumed over the past 12 months. The questionnaire contained 131 total items, and response categories differed by food groups reflecting standard food portions available and familiar to adolescents. Based on responses on the YAQ, we estimated scores on energy, macronutrients, micronutrients and mean daily servings of foods consumed. We considered values <500 or >7000 calories per day invalid and marked as missing.

The HEI-2020 assesses adherence to the 2020–2025 Dietary Guidelines for Americans (DGA) with high validity and reliability.[25] The HEI scoring algorithm method adjusted for total energy consumption to derive HEI thirteen components and HEI total score. Nine food components assessed the adequacy of intake, where higher scores indicate higher consumption: total fruit (ranging from 0–5), whole fruit (0–5), total vegetables (0–5), greens and beans (0–5), whole grains (0–10), dairy (0–10), total protein foods (0–5), seafood and plant proteins (0–5) and fatty acids (0–10). Four food components assessed limited intake, where higher scores indicate lower consumption, each ranging from 0 to 10: refined grains, sodium, saturated fats and added sugars. The 13 components were summed to generate the HEI total score for adolescents (0–100 points), where a greater score indicates higher adherence to the DGA.

2.4 | Sleep quantity and quality

We estimated sleep and wake measures from Actical accelerometer data using the validated American Sleep Association (ASA) Sadeh algorithm for adolescents[26] based on a wake threshold of 40 (medium sensitivity).[27] Due to the limited number of accelerometers, data were collected on a subsample of the parent study determined through a randomization procedure. Only complete days (i.e., full 24-h periods) with a daily average of 80 counts/minute and at least three nights/assessment[28] were included in the analysis. We truncated data after seven days (totalling a maximum of 6 nights). Participants wore accelerometers on their non-dominant ankle, secured in place with non-removable hospital bands. Actical accelerometer placed on the wrist or ankle yields a similar or high intraclass correlation compared to PSG in sleep latency, total sleep time and sleep efficiency.[29] Removal of the accelerometer necessitated cutting off the band. Once participants removed the band, they were unable to reattach it. Our analyses accounted for days of wear to measure wear time.

Quantity of sleep is represented by the total nocturnal sleep hours, which was the sum of minutes scored as sleep from sleep onset (first of ten continuous minutes scored as sleep occurring between 7:00 p.m. and 5:00 a.m. next day) and sleep offset (last minute of sleep followed by ten continuous minutes of awake, and could occur anytime between 5:00 a.m. and 1:00 p.m.) minus the number of wake minutes between sleep onset and offset (i.e., total sleep time). Quality of sleep is represented by sleep efficiency, calculated as the percentage of nocturnal sleep minutes out of the total sleep period.

2.5 | Body mass index *z*-score

We measured height and weight at three-time points using a portable stadiometer (Shorr Productions, Olney, MD) and a weight scale (Tanita TBF-410, Arlington Heights, IL) to the nearest 0.1 cm and 0.1 kg in triplicate and then averaged. We measured participants with their shoes removed and light clothing. We calculated BMI-for-age and sex-specific *z*-scores and compared them to the CDC growth charts.[30] We defined overweight or obese as BMI-for-age >1 *z*-score.

2.6 | Covariates

We collected sociodemographic characteristics at T1. We calculated age in years using dates of birth and interview and treated age as a continuous variable. Caregivers reported household size and annual household income (US$) via a self-administered questionnaire sent by mail. We generated the income-to-poverty ratio using the weighted average poverty thresholds by the U.S. Census Bureau specific to each cohort (2009–2013). A poverty ratio below 1 indicates that the average income for each respective family size is below the federal poverty threshold.

At every time point, we used the two-item Tanner scale to assess puberty development. Girls reported sexual maturation on a scale from 1 to 5 for breast and pubic. Higher scores denoted more maturation.[31] The average of the two-item Tanner scale was used as a time-varying covariate.

Moderate-to-vigorous physical activity (MVPA) data were extracted using Actical accelerometer and reduced using Actiware 9.0. Validated thresholds for adolescent's MVPA from ankle accelerometry were applied,[27] yielding minutes of MVPA per day.

Covariates were chosen based on associations reported previously and significant associations with sleep, diet and obesity.

2.7 | Data analyses

We employed multilevel mixed-effect models to assess the association between parameters of sleep (quantity and quality) in relation to adolescent diet quality and the association between diet quality and zBMI using maximum likelihood estimation. We used random intercepts to account for the clustering of the repeated measures over time within the same individual and at the school level. We assessed the association between sleep (time-varying predictor) and diet quality over time. We also assessed the association between diet quality (time-varying predictor) and zBMI over time. To assess whether the association varied over time, we included an interaction term between the time of assessment and independent variables, in separate models. All models controlled for grade in school, study wave, Tanner score, moderate-to-vigorous physical activity minutes and intervention group. Because there might be variations in the adolescent girl's age at each time point, we conducted sensitivity analyses that assessed the adolescent's age (continuous) instead of the time point in the interaction.

We employed structured equation models (SEM) with the observed variables to assess associations and statistical mediations among sleep, diet quality and zBMI. We calculated bootstrapped indirect effects with a product of the coefficients approach using the nonlinear combination of parameters in Stata, following the contemporary approach for assessing mediation proposed by Preacher & Hayes.[32] We conducted a sensitivity analysis of bootstrapped indirect effects fitting a seemingly unrelated regression (SUR) model, a special case of generalized regression model where error terms are correlated. In line with Jose (2016), we constructed a complete mediation model accounting for all possible paths between observed variables at all three-time points.[33] We also considered more parsimonious models that focused on the main relations tested in our sensitivity analysis, but present here the complete mediation model as they yielded similar results. We conducted two models: one using sleep duration (Model 1) and a second model with sleep efficiency (Model 2). In both Model 1 and Model 2, we tested the mediating effect of (i) diet on the association between sleep and zBMI and (ii) the mediating effect of sleep on the association between diet and zBMI. We did not test zBMI as a mediator. Goodness of fit statistics such as Tucker–Lewis index (TLI), comparative fit index (CFI), root mean square error of approximation (RMSEA) and the coefficient of determination (CD) were used to determine model fit.[34]

3 | RESULTS

Figure S1 illustrates the participant flow from the baseline sample, reasons for excluding sleep and diet data and the final analytical sample. Table 1 shows the baseline characteristics of the 441 adolescent girls who were included in the analysis. The mean age of adolescent girls was 12.1 (SD 0.7) years, 48% had obesity, with a mean HEI total score of 55.8 (SD 7.5), mean hours of sleep 7.9 (SD 1.2) and sleep efficiency of 87%. Adolescent girls who completed all evaluation data points were younger (mean age − 0.13 SE 0.07, 95% CI −0.27; −0.01) and had lower sleep efficiency (−1.9 SE 0.9, 95% CI 0.15; 3.69) than those who had one or two missing points.

3.1 | Longitudinal association between sleep (duration and quality) and diet quality

The association between sleep (quantity and quality) and diet quality did not vary over time (unstandardized b for sleep hours × time interaction = −0.09, 95% CI [−0.47, 0.29]; b for sleep

TABLE 1 Descriptive information on adolescent girls' characteristics of the analytical sample ($n = 441$).

	T1		T2		T3	
Adolescent characteristics	***n***	**Mean (SD)**	***n***	**Mean (SD)**	***n***	**Mean (SD)**
Age (years)	441	12.15 (0.71)	326	12.60 (0.71)	259	13.21 (0.68)
School grade	441	6.44 (0.51)	326	6.45 (0.50)	259	7.43 (0.50)
Tanner score[a]	413	3.19 (0.96)	314	3.52 (0.90)	253	3.60 (0.90)
Race Black (%)	441	100.00	326	100.00	259	100.0
Poverty ratio (<1.0) (%)[b]	101	48.10	168	47.00	135	50.00
Body mass index						
Body mass index (z-score)[c]	441	0.98 (1.04)	326	1.01 (1.08)	259	0.93 (1.06)
Overweight or obese (%)[d]	217	49.20	166	50.92	124	47.88
Healthy eating index						
Total score	441	55.84 (7.49)	326	54.76 (7.79)	259	54.94 (7.34)
Sleep parameters						
Total number of nights	441	4.84 (0.70)	326	4.72 (0.60)	259	4.67 (0.54)
Night-time sleep (hours)	441	7.93 (1.25)	326	7.63 (1.59)	259	7.77 (1.63)
Sleep efficiency (%)[e]	441	87.75 (9.09)	326	85.28 (11.00)	259	84.61 (11.07)

Note: T1 (enrolment), T2 (6-month follow-up) and T3 (18-month follow-up).
Abbreviation: SD (standard deviation).
[a]Mean score of the two Tanner items breast and pubic.
[b]The income-to-poverty ratio was calculated according to the U.S. Census Bureau, Weighted Average Poverty Thresholds, 2009–2012. Annual household income was reported by the adolescent girl's caregiver ($n = 221$).
[c]Body mass index was calculated for age- and sex-specific z-score according to the CDC Child Growth Reference for girls.
[d]Overweight and obese categories were calculated using the World Health Organization standard for children 5–19 years and their respective zBMI cut-off points. Using the International Obesity Task Force (IOTF) cut-off yielded the same prevalence.
[e]% of the time asleep during the sleep period.

TABLE 2 Longitudinal associations between sleep behaviour and diet quality among adolescents across 3 time points.

Model[a]	Healthy Eating Index (95% CI)[b]	p-value	Difference in beta between T1 and T2 (95% CI)[b]	p-value	Difference in beta between T1 and T3 (95% CI)[b]	p-value	Difference in beta between T2 and T3 (95% CI)[b]	p-value	Cohen's f^2 effect size
Sleep duration (hours)									
T1	−0.410 (−1.114, 0.294)	0.254	Reference		Reference				−0.069
T2	0.169 (−0.376, 0.714)	0.543	0.579 (−0.258, 1.416)	0.175			Reference		
T3	−0.577 (−0.934, −0.221)	0.001			−0.167 (−0.968, 0.633)	0.682	−0.747 (−1.332, −0.161)	0.012	
Sleep efficiency									
T1	−0.037 (−0.122, 0.049)	0.399	Reference		Reference				−0.070
T2	0.008 (−0.081, 0.097)	0.867	0.044 (−0.076, 0.165)	0.470			Reference		
T3	−0.065 (−0.148, 0.019)	0.128			−0.028 (−0.159, 0.104)	0.677	−0.072 (−0.160, 0.016)	0.108	

Note: T1 (enrolment), T2 (6-month follow-up) and T3 (18-month follow-up).
Abbreviation: CI, confidence interval.
[a]Hierarchical models controlled for adolescent's grade in school (6th–7th), minutes of moderate-to-vigorous physical activity, Tanner score, cohort of data collection (2009–2012) and intervention group. ($n = 441$).
[b]Represents the average marginal effect of change in Healthy Eating Index that is produced by a 1-unit increase in sleep at a given time point.

efficiency × time interaction = −0.01, 95% CI [−0.08, 0.05]). There was a negative cross-sectional association between sleep duration and diet quality at T3 3 ($b = -0.57$, 95% CI [−0.93, −0.22]), but not at T1 and T2 (Table 2).

3.2 | Longitudinal association between diet quality, sleep and zBMI

The association between diet quality and zBMI did not vary over time (b for HEI*time interaction = 0.001, 95% CI [−0.002, 0.004]) (Table 3). The association remained null when including time as a covariate in the model to restrict the association to be equal over time (b for HEI = −0.001, 95% CI [−0.01, 0.01]). Similarly, sleep duration did not predict zBMI. However, greater sleep efficiency was cross-sectionally associated with lower zBMI ($b = -0.004$ [95% CI −0.006, −0.001]) at T1, but not at T2 and T3.

3.3 | Diet quality as a mediator of the longitudinal association between sleep and zBMI

SEM Model 1 examined the longitudinal mediation association between sleep duration, diet quality and zBMI and presented a good model fit (RMSEA = 0.048, CFI = 0.993). Sleep duration at T1 was not associated with diet at T2 (a_1 path), nor was diet associated with zBMI at T3 (b_1 path) (Figure 1). Likewise, the bootstrapped indirect effect was not significant, indicating that diet was not a significant longitudinal mediator between sleep duration and zBMI ($a_1 \times b_1$ path = −0.001, bootstrapped 95% CI = −0.002; 0.001, p-value = 0.718).

Diet at T1 was not associated with sleep duration at T2 (a_2 path) nor was sleep duration at T2 associated with zBMI at T3 (b_2 path). The bootstrapped indirect effect was not significant, indicating that sleep duration was not a significant longitudinal mediator between diet and zBMI ($a_2 \times b_2$ path = −0.001, bootstrapped 95% CI = −0.002; 0.001, p-value = 0.598).

Model 1 examined the longitudinal mediation association between sleep quality, diet quality and zBMI and presented a good model fit (RMSEA = 0.049, CFI = 0.993). Similar to Model 1, sleep quality at T1 was not associated with diet at T2 (a_3 path) nor was diet associated with zBMI at T3 (b_3 path) (Figure 2). Likewise, the bootstrapped indirect effect was not significant, indicating that diet was not a significant longitudinal mediator between sleep quality and zBMI ($a_3 \times b_3$ path = −0.001, bootstrapped 95% CI = −0.002; 0.001, p-value = 0.811).

When testing sleep quality as the mediator, diet at T1 was not associated with sleep quality at T2 (a_4 path) nor was diet associated with zBMI at T3 (b_4 path). Likewise, the bootstrapped indirect effect was not significant indicating that sleep quality was not a significant longitudinal mediator between diet and zBMI ($a_4 \times b_4$ path = −0.001, bootstrapped 95% CI = −0.002; 0.001, p-value = 0.649).

4 | DISCUSSION

Two major findings emerged from this longitudinal investigation of associations between sleep parameters, diet and body mass index among a homogenous sample of Black adolescent girls from a

TABLE 3 Longitudinal associations between diet quality, sleep and body mass index (BMI) *z*-score among adolescents across 3 time points.

Model[a] Time-variant predictors	Body mass index *z*-score (zBMI) as outcome (95% CI)[b]	*p*-value	Difference in beta between T1 and T2 (95% CI)[b]	*p*-value	Difference in beta between T1 and T3 (95% CI)[b]	*p*-value	Difference in beta between T2 and T3 (95% CI)[b]	*p*-value	Cohen's f^2 effect size
Healthy eating index									
T1	−0.001 (−0.005, 0.003)	0.699	Reference		Reference				18.094
T2	−0.000 (−0.004, 0.003)	0.846	0.000 (−0.004, 0.005)	0.840			Reference		
T3	0.002 (−0.005, 0.008)	0.653			0.002 (−0.004, 0.009)	0.473	0.002 (−0.004, 0.008)	0.553	
Sleep durations (hours)									
T1	−0.022 (−0.103, 0.059)	0.589	Reference		Reference				22.400
T2	−0.029 (−0.106, 0.049)	0.466	−0.006 (−0.032, 0.019)	0.620			Reference		
T3	−0.051 (−0.121, 0.020)	0.162			−0.028 (−0.065, 0.008)	0.127	0.000 (−0.003, 0.004)	0.808	
Sleep efficiency									
T1	−0.004 (−0.006, −0.001)	0.004	Reference		Reference				25.336
T2	−0.001 (−0.003, 0.001)	0.335	0.003 (−0.000, 0.006)	0.082			Reference		
T3	−0.001 (−0.004, 0.003)	0.685			0.003 (−0.000, 0.006)	0.058	−0.022 (−0.050, 0.006)	0.127	

Note: T1 (enrolment), T2 (6-month follow-up) and T3 (18-month follow-up).
Abbreviations: CI, confidence interval; zBMI, body mass index *z*-score.
[a]Hierarchical models controlled for adolescent's grade in school (6th-7th), minutes of moderate-to-vigorous physical activity, Tanner score and cohort of data.
[b]Represents the average marginal effect of change in zBMI that is produced by a 1-unit increase in healthy eating index or sleep at a given time point.

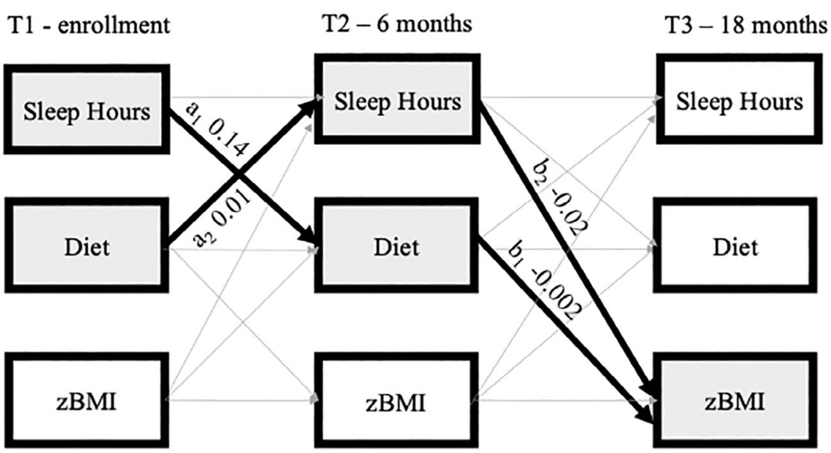

FIGURE 1 Model 1 longitudinal associations between sleep hours, diet quality and body mass index *z*-score. zBMI, body mass index *z*-score. Data shown are coefficients, and all associated p values are above 0.05. T1 (enrolment), T2 (6-month follow-up) and T3 (18-month follow-up).

predominantly low-income background. First, neither diet nor sleep was longitudinally associated with zBMI over time. However, greater sleep quality was associated with lower zBMI cross-sectionally (T1). Second, diet quality was not a longitudinal mediator of the association between sleep quantity or quality and zBMI. A longitudinal investigation of sleep, diet and body mass index in a homogenous sample is an important contribution to the current body of literature, as there has been limited attention to possible differences in sex, age, race and socioeconomic levels with inconsistent findings partially due to participants' heterogeneity.[18,19]

Most studies examining the association between sleep and obesity among adolescents have been cross-sectional[17] and used self-reported sleep,[35] which could explain the discrepancy with our findings of the null association between sleep and obesity

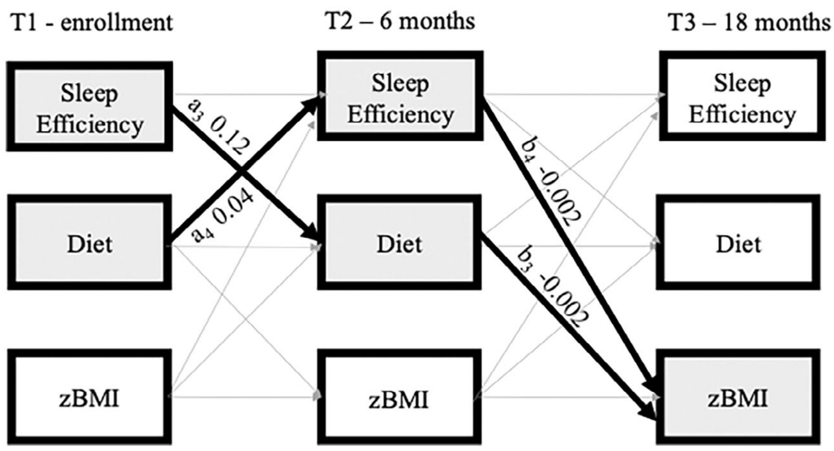

FIGURE 2 Model 2 longitudinal associations between sleep efficiency, diet quality and body mass index *z*-score. zBMI, body mass index *z*-score. Data shown are coefficients, and all associated p values are above 0.05. T1 (enrolment), T2 (6-month follow-up) and T3 (18-month follow-up).

longitudinally. However, two recent meta-analyses reported small but significant evidence that longer sleep duration and higher sleep quality were related to lower zBMI in adolescence over time.[18,19] Nevertheless, the longitudinal association between sleep and weight gain over time has been stronger for children than adolescents,[18] and few studies have included diverse samples or Black adolescent girls, and more than two time points of assessment.[19] Another possible explanation for the null association between sleep and obesity in this longitudinal study can be attributed to the ambiguous temporal effects in measurement of both variables. That is, sleep behaviour at present might not capture the cumulative effects (or change) of sleep patterns over the next six or 18 months. Similarly, zBMI measured later might reflect changes due to various known and unknown risk factors beyond sleep.

Our longitudinal analysis consistently indicated that diet quality, as defined using the HEI, was not associated with sleep or zBMI among Black adolescent girls over time. Another recent longitudinal analysis reported no association between sleep duration and caloric intake among adolescents in Mexico City,[36] corroborating our findings. Nevertheless, longer sleep duration was statistically significantly associated with lower diet quality at T3 among adolescent girls, disagreeing with the literature. It is possible that adolescent girls with longer sleep patterns may have lower frequency of main meals or irregular mealtimes, which could in turn explain a lower diet quality,[37] although future studies should explore the role of meal timing and meal frequency in the sleep and diet association. Taken together, these analyses contradict the majority of the evidence on the topic stemming from cross-sectional studies[19] and highlight the need for better understanding of daily lifestyle behaviours among adolescent girls. Diet quality had lower variability than observed in a national NHANES sample,[38] which could have precluded our ability to determine statistical probabilities and thus miss large differences if true. Furthermore, there is heterogeneity in the definition of diet quality across studies with most focusing on the consumption of specific food groups or nutrients instead of assessing the quality of the diet overall, contributing to the mixed findings.[19] Data from cross-sectional analysis indicate that frequent consumption of ultra-processed foods is associated with a shorter sleep duration[39] and lower quality among children and adolescents.[40] Conversely, two experimental studies investigated dietary intake among adolescents after shorter sleep duration reporting a null association with macronutrient intake,[41] greater consumption of sweet/dessert foods, but no association with other food groups.[42]

The longitudinal interplay between sleep, diet and zBMI in adolescents is complex. Although diet quality has been suggested to be a plausible mediator between sleep and BMI,[43] other factors could also influence the relationship between sleep and BMI which could have attenuated the mediating effect of diet. Indeed, sleep, physical activity and sedentary behaviours make up 24-h movement behaviours that continually interact with one another. For instance, short sleep duration increases daytime sleepiness and subsequent sedentary behaviour. In turn, activities that promote sedentary behaviour, such as screen time, could decrease the amount of time during the day devoted to physical activity.[44] Eventually, increased sedentary behaviour and decreased physical activity will contribute to obesity development.[44] Although our longitudinal mediation analysis controlled for physical activity, future studies could explore its role as a mediator of the association between sleep and zBMI among adolescents. Furthermore, biological factors may also influence the complex relation between sleep, diet and BMI, including circadian phase delays common during adolescence. The delay in circadian rhythm with the onset of puberty[45] paired with social factors such as increased autonomy over bedtimes[46] and early school start times may result in shorter sleep duration on school nights. Adolescents often compensate for this short weekday sleep by delaying their wake-up time on weekends, a phenomenon known as social jetlag, which has been consistently associated with obesity risk and metabolic syndrome.[47] Further research is warranted to investigate the influence of behavioural and social factors on the association between sleep and weight gain, particularly among adolescents.

Adding to the complexity of the interplay between sleep, diet and BMI is their potential bidirectional relationships. Few studies have assessed bidirectionality, with those that have, presenting mixed findings. Among adult samples, few studies have supported obesity as a risk factor for daytime sleepiness and wakefulness at night.[48] Among adolescents, more studies have shown the absence of a bidirectional relationship between sleep and BMI[19] than those that support a bidirectional relationship.[49] Even less work has explored the bidirectionality between sleep and diet quality. Cross-sectional studies have found that adolescents who consume more processed foods, skip breakfast and have irregular eating schedules are more likely to have poorer sleep quality compared to their counterparts.[40] One longitudinal study has explored the bidirectionality between sleep and diet and found sleep quantity to be positively associated bidirectionally with junk food over time.[49] Our analysis adds to this scarce body of literature, as it investigates the potential bidirectional association between sleep and diet and how it further influences weight gain longitudinally among Black adolescent girls. Future work is needed to clarify the directionality of these relationships and how they may affect one another in a feedback loop.[19]

A major strength of this study was the inclusion of a large homogenous sample characterized by race, sex, age and socioeconomic status, as it inherently controls for known confounding variables and reduces variability. To date, few studies have examined the longitudinal association between sleep, diet and obesity among early adolescents with no or limited attention to racial, sex or socioeconomic differences, potentially explaining discrepancies in findings. Future studies should investigate effect modification by sex, age, race and socioeconomic levels. The use of objectively measured sleep duration and quality, and height and weight (zBMI), along with a validated food frequency questionnaire to estimate diet quality analysed longitudinally are additional strengths. Furthermore, this study focused on an understudied population, although the generalizability of the findings does not extend beyond Black adolescent girls with similar demographics. The study has potential limitations that must be acknowledged. First, participant loss over time is a limitation, although the attrition rate was typical of expectations in community- and school-based intervention trials. Second, meal timing was not assessed in this study but should be objectively measured in future studies as the literature suggests that it may play a role in sleep. Third, accelerometry data collection was not paired with a sleep log or other measures of subjective sleep quality, since sleep was not a focus of the parent study. Nevertheless, differences between pediatric sleep data with and without a sleep log are minimal.[50] The ankle placement of the accelerometry might have generated sleep measures that may differ from other studies using wrist and waist accelerometry, which could in turn explain discrepant findings with the literature. Although the placement of the accelerometry on the ankle is somewhat novel in adolescent populations, the validity, reliability and feasibility of ankle placement in adolescents have been demonstrated.[27] Excellent interunit reliability and good criterion validity have been shown when ankle placement was compared to wrist and waist placement in adults, particularly for walking.[51] Furthermore, the high compliance rate of using accelerometry on the ankle in this study is a strength and adds to the rationale for the choice of accelerometry placement. The Sadeh algorithm has been validated on the ankle for adults;[52] thus, future validation studies are warranted for sleep and wake estimation using the Sadeh algorithm among adolescents wearing ankle accelerometry. Fourth, the timing of data assessment points in this longitudinal study may not reflect the time lag necessary for sleep to influence diet. For example, sleep may have a more immediate effect on food consumption, as opposed to a long-term effect (6 months or more). Future studies should assess the short-term effect of sleep on diet with multiple assessments along with a long-term assessment of BMI, as health outcomes are more likely to take time to change following behavioural outcomes. It is possible that seasonality could have impacted the outcomes, as youth diet and physical activity are known to change across school and summer. However, the role of school structure is unlikely to have played a role in our study, as data were collected only when school was in session and not during periods of extreme weather conditions (Summer or Winter) or holiday breaks. Lastly, data from this study were collected more than 10 years ago, when media and smartphone use among adolescents were not as prevalent as nowadays, negatively influencing contemporary declines in sleep time. This study provides an opportunity to understand the effect of media use in the sleep, diet and obesity associations. Subsequent studies could further explore the impact of smartphones and media.

5 | CONCLUSIONS

In conclusion, our study suggests that there was no longitudinal association between sleep (duration and quantity) or diet quality with zBMI and that diet quality was not a longitudinal mediator of sleep and zBMI among Black adolescent girls of low-income communities. Our findings add to the scarce body of literature that explores longitudinal associations between objectively and validated measured behavioural and health outcomes among underserved populations.

AUTHOR CONTRIBUTIONS

ACBT designed and conceived the study, did the data analyses and data interpretation and wrote the manuscript. LC, BA and MMB formulated the clinical question, reviewed the statistical analyses and interpreted the data. ACBT, LC, BA, MMB and GMV drafted the manuscript and critically reviewed the report.

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CONFLICT OF INTEREST STATEMENT

No conflict of interest was declared.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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