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RESEARCH ARTICLE

Associations of actigraphy-assessed sleep variables with adiposity and serum cardiometabolic outcomes in emerging adults

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Summary

This study assessed associations of actigraphy-assessed sleep with adiposity and serum cardiometabolic outcomes in emerging adults, and whether sex and race modified these associations. Data on 147 emerging adults (age = 19.4 ± 1.3 years; body mass index = 26.4 ± 7.0 kg m^{-2}; 59% female; 65% White) from RIGHT Track Health were used. Actigraphy-based sleep measures included sleep duration, sleep efficiency, sleep timing midpoint, day-to-day sleep duration and sleep timing midpoint variability. Combined sleep duration and sleep timing behaviours were also derived (early-bed/late-rise, early-bed/early-rise, late-bed/late-rise, late-bed/early-rise). Outcomes included body mass index and BodPod-assessed fat mass index, fasting serum leptin, C-reactive protein, and homeostatic model assessment-insulin resistance. Sleep duration was 5.4 h per night. We noted an inverse association between sleep duration and homeostatic model assessment-insulin resistance. The early-bed/early-rise group had greater body mass index, C-reactive protein and homeostatic model assessment-insulin resistance compared with the early-bed/late-rise group (referent). Sex modified associations of sleep efficiency with C-reactive protein; stratified results revealed positive association between sleep efficiency and C-reactive protein in males, but not females. Race modified associations of sleep duration with body mass index and leptin, and of sleep duration variability with C-reactive protein. Stratified analyses revealed inverse associations between sleep duration with body mass index and leptin in Black, multiracial/other race individuals only. Positive association between sleep duration variability and C-reactive protein was noted in White individuals only. Shorter sleep duration, particularly when combined with earlier sleep timing, is associated with greater adiposity and serum cardiometabolic outcomes. Additional studies are needed to assess individual- and contextual-level factors that may contribute to sex and race differences in sleep health and cardiometabolic risk in emerging adults.

KEYWORDS
adiposity, cardiometabolic health, emerging adulthood, sleep

1 | INTRODUCTION

Emerging adulthood (approximately ages 18–28 years) represents a developmental period defined by "frequent change and exploration" (Arnett, 2000). Emerging adults often face competing demands (e.g. school, work, personal and social relationships) as they aim to establish their identities, independence, relationships, and career and life goals (Arnett, 2000). Adult health behaviours, including sleep, are often established during emerging adulthood, shaping the lifelong trajectory of cardiometabolic risk and wellbeing (Gooding et al., 2020). Furthermore, evidence suggests that sleep duration gradually declines during emerging adulthood (Maslowsky & Ozer, 2014). Concurrent with this decline in sleep duration, obesity rates double from adolescence through early emerging adulthood, and then double again by late emerging adulthood (Barbour-Tuck et al., 2018; Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010).

Short sleep duration (typically < 7 h of sleep per night in adults) and poor sleep quality (typically self-reported or based on actigraphy-measured sleep efficiency < 85%) are consistently associated with higher body weight, fat mass (FM) and/or a greater obesity risk (Antza et al., 2021; Bacaro et al., 2020; Fatima et al., 2016). Recent reviews also reported that having a later sleep timing midpoint (i.e. wake-time – ½ sleep duration), as well as greater day-to-day sleep duration and sleep timing variability are associated with greater adiposity and adverse cardiometabolic outcomes (Chaput et al., 2020; Morales-Ghinaglia & Fernandez-Mendoza, 2023). Consistent evidence suggests that emerging adults and college students with shorter sleep duration and/or poorer sleep quality have greater adiposity (Bailey et al., 2014; Fernström et al., 2020; Kahlhöfer et al., 2016; Krističević et al., 2018; Meyer et al., 2012; Peltzer & Pengpid, 2017; Quick et al., 2014; Sa et al., 2020; Vargas et al., 2014; Yang et al., 2020), but much of this evidence is limited to self-reported sleep variables and/or using body mass index (BMI) as a single indicator of adiposity/obesity.

Short sleep duration and poor sleep quality are also consistently associated with disruptions in serum metabolic and inflammatory outcomes (Irwin et al., 2016; Koren & Taveras, 2018; Leproult & Van Cauter, 2010; Singh et al., 2022), which increases cardiometabolic risk. In emerging adults, there is initial evidence to suggest that short sleep duration, poor sleep quality and sleep duration variability are associated with worse serum cardiometabolic outcomes, including greater insulin resistance (as assessed by homeostatic model assessment of insulin resistance [HOMA-IR]; Fernström et al., 2020) and elevated C-reactive protein (CRP; Bakour et al., 2017; Okun et al., 2009; Park et al., 2020). Further evidence on associations of multiple actigraphy-assessed sleep variables and serum cardiometabolic outcomes is needed to compliment these findings in emerging adults.

The primary aim of this cross-sectional analysis was to assess associations between actigraphy-assessed sleep duration, sleep efficiency, sleep timing midpoint, as well as day-to-day sleep duration and sleep timing midpoint variability with adiposity (BMI and FM index [FMI]), and serum cardiometabolic (leptin, CRP, HOMA-IR) outcomes in emerging adults. Our secondary aim was to assess effect modification of these associations by sex and race as these demographic factors may influence associations between sleep and obesity risk (Koren & Taveras, 2018). We hypothesized that shorter sleep durations, lower sleep efficiencies, later sleep timing midpoints, and greater day-to-day sleep duration and sleep timing midpoint variability would be associated with BMI, FMI, as well as fasting serum leptin, HOMA-IR and CRP. We also hypothesized that sex and race will modify these associations, indicating stronger associations in females, as well as in Black and multiracial/other race individuals.

2 | METHODS

2.1 | Study design and participants

Cross-sectional data were collected during emerging adulthood from the RIGHT Track Health study (Dollar et al., 2020; Wideman et al., 2016). Participants completed an in-laboratory visit following a 10-h overnight fast from food; ad libitum water intake was allowed. Participants were also asked to refrain from smoking/vaping the morning of the visit, as well as alcohol intake and vigorous-intensity physical activity participation for at least 24 h prior to this visit. The visit was re-scheduled if participants reported: (1) illness/injury in the past week or surgery in the past month; (2) immunizations within the past 2 weeks; and (3) use of antibiotics, corticosteroids or other prescription anti-inflammatories within the past 10 days. During this visit, after completing a fasting blood draw, participants were offered a small snack. Then, they completed questionnaires, resting heart rate and blood pressure measures, body composition measures, resting orthostatic challenge, maximal exercise testing, and post-exercise orthostatic challenge. Following this visit, participants were given an accelerometer to wear for 7 consecutive days. The Institutional Review Board at the University of North Carolina at Greensboro approved all study procedures (IRB #11-0360). Written informed consent was obtained from all participants. A total of 319 participants completed at least one component of data collection during the emerging adulthood timepoint. Actigraphy data were available in 165 participants. Of these, 147 participants had at least 3 valid days of accelerometry wear time (≥ 13 h per day of wear time) from which sleep variables could be derived and were thus included in the present analyses. All valid accelerometry days for each participant were used in the sleep calculations and analyses. Twelve participants (8%) had 3–4 valid accelerometry days, 44 participants (30%) had 5–6 valid accelerometry days and 91 participants (62%) had 7–10 valid accelerometry days. Details on the original participant recruitment and the emerging adulthood health assessments are described elsewhere (Dollar et al., 2020; Wideman et al., 2016). A flow chart presenting the selection of participants for these analyses is presented in Figure 1. Measures relevant to these analyses are described in more detail below.

2.2 | Actigraphy-derived sleep variables

Participants were asked to wear an Actigraph GT9X Link accelerometer (Actigraph LLC, Pensacola, FL, USA) on their non-dominant wrist

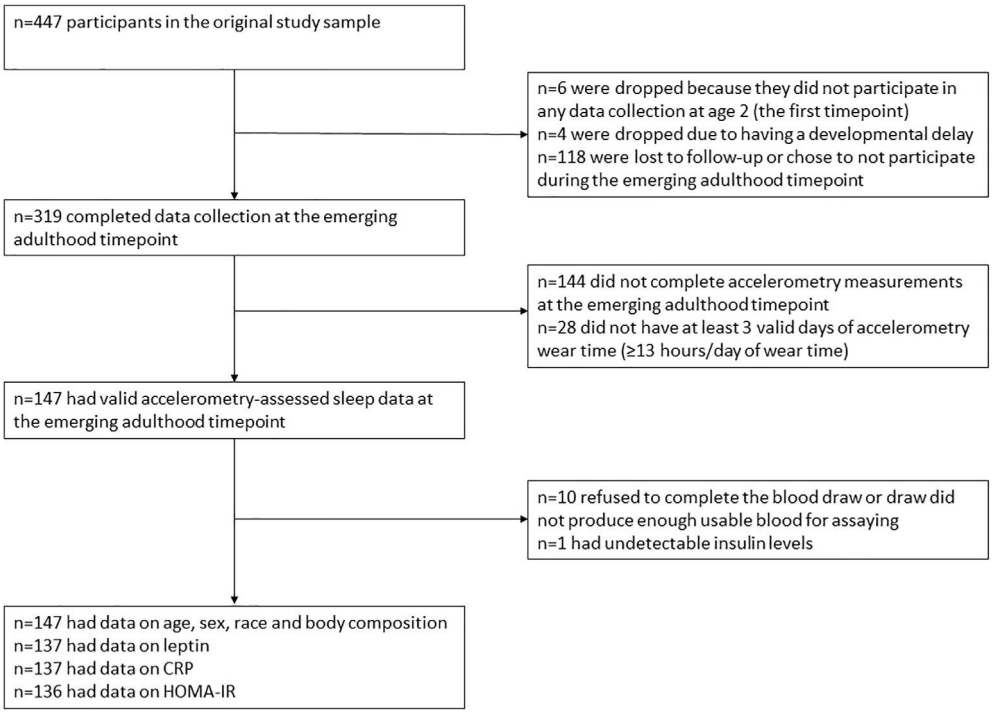

FIGURE 1 Study flow chart and selection of participants for analyses focused on associations between multiple actigraphy-assessed sleep variables with adiposity and serum cardiometabolic outcomes in emerging adults, The RIGHT TRACK Health study, North Carolina, USA, 2014-2018. CRP, C-reactive protein; HOMA-IR, homeostatic model assessment-insulin resistance.

for 24 h per day over 7 consecutive days. Some participants wore the device for more than 7 days prior to mailing it back to the research team. Participants were instructed to remove the device for water-based activities, and when required by sporting competition or occupation (Wideman et al., 2016). Periods of non-wear time were reported using a log sheet. Accelerometers collected data at a sampling rate of 30 Hz and aggregated to 60-s epoch files for analysis by the Actilife software (version 6.13.4). Sleep duration (hr per day) and sleep efficiency (sleep duration/time in bed; %) were derived from the Sadeh algorithm because it was originally validated on a sample of adolescents and emerging adults (Sadeh et al., 1994). Sleep duration was based on total sleep time (including multiple bouts of sleep in 1 night and daytime naps) accumulated during a 24-h period. Following visual inspection of the daily sleep data for each participant after applying the Sadeh algorithm, some participants had multiple bouts of sleep in 1 night (e.g. two bouts of sleep separated by < 3 h). In these instances, the elapsed time between bedtime for the first sleep bout and waketime for the last sleep bout (when < 3 h separated each bout) was used as the denominator in the sleep efficiency calculation. This approach has been recommended by Reed and Sacco (2016) to account for periods of sleep discontinuation (i.e. time out of bed during nighttime awakenings). A nap was logged/identified if a prior sleep bout had occurred, and this nap was separated from that prior sleep bout by ≥ 3 h. A total of six naps were identified in six different participants, hence we do not expect that the inclusion of the nap data into the total sleep duration calculation will meaningfully impact the results. The Sadeh algorithm was also used to derive bed- and wake-times, from which sleep timing midpoint (clock time) was calculated as wake-time − ½ sleep duration (Roenneberg et al., 2003). Sleep timing midpoint is meant to be an indicator of endogenous circadian phase (or when a person prefers to sleep), and has been referred to as the best phase anchor point for melatonin onset (Roenneberg et al., 2003). The sleep timing midpoint calculation was only applied to prolonged night- or daytime sleep behaviours (i.e. excluded naps) to have a single sleep timing midpoint value per day for each participant. Lastly, day-to-day sleep duration and sleep timing midpoint variability were calculated as the coefficient of variation in sleep duration and sleep timing midpoint (i.e. standard deviation [SD] of sleep duration or sleep timing midpoint across all valid accelerometry days divided by mean sleep duration or sleep timing midpoint × 100), as previously described by Lemola et al. (2013).

2.3 | Adiposity outcomes

Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer (SECA, Chino, CA, USA); weight was measured to the nearest 0.1 kg with a balance-beam scale (Detecto-Medic, Brooklyn, NY, USA). BMI was calculated as body weight (kg)/height (m^2). FM (kg) was assessed via air displacement plethysmography with a BOD POD (Cosmed, Concord, CA, USA). Participants entered the BOD POD chamber wearing minimal, skintight clothing (e.g. spandex shorts, sports bra), and a swim cap to cover their hair when possible. Standard manufacturer calibration and measurement procedures were followed. Participants' thoracic lung volume was measured using the BOD POD breathing circuit system, and FM was calculated using age- and race-appropriate algorithms built into the BOD POD system. FMI was calculated as FM (kg)/height (m^2). We chose to use BMI and FMI in these analyses to account for participants' height. Previous research has found that FMI helps to classify obesity more accurately when

compared with body fat percentage in men and women (Peltz et al., 2010).

2.4 | Serum cardiometabolic outcomes

Fasting serum leptin (ng ml^{-1}), insulin (pg ml^{-1}) and CRP (mg L^{-1}) were analysed at the University of North Carolina at Greensboro Exercise Physiology Laboratory, using multiplex ELISA kits (EMD Millipore Sigma, Burlington, MA, USA) and the Luminex 200s (Luminex, Austin, TX, USA) plate reader. Fasting glucose (mg dl^{-1}) was measured using a colorimetric assay (Cayman Chemical, Ann Arbor, MI, USA) and the EPOCH plate reader (Biotek, Santa Clara, CA, USA). All samples were analysed in duplicate with appropriate quality controls, and all samples from a single participant were analysed in the same ELISA plate to minimize inter-assay variability (Wideman et al., 2016). Fasting serum glucose and insulin levels were used to calculate HOMA-IR as follows: fasting insulin (μU ml^{-1}) × fasting glucose (mg dl^{-1})/405. Insulin unit conversions followed standardized procedures, with a molecular weight of 5808 g mol^{-1} for insulin and the 1 μIU ml^{-1} equal to a 6 pmol L^{-1} conversion, as outlined by Knopp et al. (2019).

2.5 | Statistical analyses

All analyses were performed using STATA software version 17 (StataCorp, College Station, Texas, USA). Descriptive data are presented as mean ± SD. Differences in descriptive data between sex (male versus female) and race (White versus Black and multiracial/other race) were assessed using an independent sample *t*-test. Stem-and-leaf and Q-Q plots were visually inspected to assess normality of data distribution for all study outcomes. BMI, FMI, leptin, CRP and HOMA-IR were not normally distributed, and log transformed. A log $(x + 1)$ transformation was also used for HOMA-IR and CRP because of the presence of negative log transformed values. Stem-and-leaf and Q-Q plots were repeated for the log transformed data to confirm that the normal distribution of these data was improved.

Linear regression models were used to assess associations between continuous sleep duration, sleep efficiency, sleep timing midpoint, day-to-day sleep duration variability and day-to-day sleep timing midpoint variability with adiposity and serum cardiometabolic outcomes. As previously described (Mikulovic et al., 2014; Zerón-Rugerio et al., 2020), combined sleep duration and sleep timing behaviours were defined using the following categories: 1 – earlier sleep timing midpoint and longer sleep duration (early-bed/late-rise); 2 – earlier sleep timing midpoint and shorter sleep duration (early-bed/early-rise); 3 – later sleep timing midpoint and longer sleep duration (late-bed/late-rise); 4 – later sleep timing midpoint and shorter sleep duration (late-bed/early-rise). To do so, a median split for sleep timing midpoint (04:39 hours) was used to define the "earlier versus later" sleep timing midpoint categories. Participants within each of these sleep timing groups were further subdivided into two categories (shorter versus longer sleep durations) to have an equal number of participants across the four abovementioned groups. Linear regression models were also used to assess associations between these combined sleep duration and sleep timing midpoint categories with adiposity and serum cardiometabolic markers. The early-bed/late-rise category was used as the referent group. Covariates included age, sex, race, number of valid accelerometry/sleep days and actigraphy-assessed total activity time (total number of minutes per day spent ≥ 100 counts per minute; Troiano et al., 2008). Continuous sleep duration, sleep efficiency, sleep timing midpoint, as well as sleep duration variability and sleep timing midpoint variability were considered as covariates when not the predictor of interest. For the combined sleep duration and sleep timing midpoint categories, only sleep efficiency, sleep duration variability and sleep timing variability were considered as sleep-related covariates. To determine the final model of best fit for each linear regression model, a stepwise backwards elimination linear regression model set at $p < 0.1$ that included all covariates was first used to generate a sequence of covariates to be added one at a time to subsequent models and assess the Akaike information criterion (AIC) associated with each of these models. The AIC balances both the fit and simplicity of the model by considering "improvements in model fit" following the addition of each covariate one at a time. The model with the lowest AIC value is considered to have the best "goodness of fit" and was selected as the final model for each analysis (i.e. the AIC was used to identify the most appropriate covariates to include in each linear regression model). Variance inflation factor (VIF) assessed multicollinearity; all final models had a VIF < 2 indicating no evidence of multicollinearity.

We tested for effect modification by sex (male versus female) and race (White versus Black and multiracial/other race) by adding interaction terms for each sleep variable with sex or race categories (one at a time) to each final model. Black and multiracial/other race categories were combined because only eight participants identified as multiracial/other racial descent, and interpretation of the effect modification results did not change when we removed these eight multiracial/other race participants (results not shown). Stratified analyses were conducted if the interaction term reached $p < 0.10$. Statistical significance was set at $p < 0.05$.

3 | RESULTS

A total of 147 participants were included in the present analyses, which included 61 males (41.5%) and 86 females (58.5%). Racial breakdown of the sample included 96 White individuals (65.3%) and 51 Black, multiracial/other race individuals (34.7%). The proportion of males ($n = 39$; 41%) and females ($n = 57$; 59%) among White individuals was comparable to the entire sample. Similarly, the proportion of males ($n = 22$; 43%) and females ($n = 29$; 57%) among Black, multiracial/other race individuals was comparable to the entire sample. Descriptive data are presented in Table 1. Sleep duration was only 5.4 h per night in this sample, which did not differ between sex and race categories. Despite these short sleep durations, sleep efficiency

TABLE 1 Participant characteristics in all participants, and based on sex (male versus female) and race (White versus Black, multiracial/other) from the RIGHT Track Health Study, North Carolina, USA, 2014–2018.

	All participants		Males only		Females only			White racial descent only		Black and multiracial/other racial descent only		Differences between racial groups
	n	Mean ± SD	*n*	Mean ± SD	*n*	Mean ± SD	Differences between sex	*n*	Mean ± SD	*n*	Mean ± SD	
Age (months)	147	233 ± 16	61	233 ± 16	86	232 ± 15	$t\,(145) = 0.35, p = 0.73$	96	233 ± 16	51	233 ± 15	$t\,(145) = 0.002, p = 0.99$
Age (years)	147	19.4 ± 1.3	61	19.4 ± 1.3	86	19.3 ± 1.3		96	19.4 ± 1.3	51	19.4 ± 1.3	
BMI (kg m^{-2})	147	26.4 ± 7.0	61	26.1 ± 6.9	86	26.6 ± 7.1	$t\,(145) = -0.44, p = 0.66$	96	24.8 ± 5.1	51	29.4 ± 8.9	$t\,(145) = -4.00, p = 0.0001$
FMI (kg m^{-2})	147	7.8 ± 5.8	61	5.8 ± 5.6	86	9.3 ± 5.4	$t\,(145) = -3.81, p = 0.0002$	96	6.8 ± 4.4	51	9.8 ± 7.4	$t\,(145) = -3.07, p = 0.003$
Leptin (pg ml^{-1})	137	14,231 ± 17,402	59	5378 ± 6973	78	20,927 ± 19,819	$t\,(135) = -5.76, p < 0.0001$	92	12,198 ± 14,037	45	18,388 ± 22,410	$t\,(135) = -1.98, p = 0.05$
HOMA-IR	136	7.3 ± 7.8	58	7.5 ± 6.3	78	7.2 ± 8.8	$t\,(134) = 0.16, p = 0.88$	91	6.1 ± 4.9	45	9.9 ± 11.4	$t\,(134) = -2.73, p = 0.01$
CRP (mg L^{-1})	137	1.7 ± 3.7	59	1.3 ± 2.7	78	2.0 ± 4.3	$t\,(135) = -1.13, p = 0.26$	92	1.2 ± 1.9	45	2.7 ± 5.8	$t\,(135) = -2.32, p = 0.02$
Total activity time (minutes per day)	147	777 ± 93	61	790 ± 91	86	769 ± 94	$t\,(145) = 1.36, p = 0.18$	96	768 ± 92	51	794 ± 95	$t\,(145) = -1.63, p = 0.11$
Sleep duration (hr per day)	147	5.4 ± 1.3	61	5.2 ± 1.5	86	5.4 ± 1.1	$t\,(145) = -0.83, p = 0.41$	96	5.4 ± 1.1	51	5.3 ± 1.5	$t\,(145) = 0.11, p = 0.91$
Sleep efficiency (%)	147	87 ± 4	61	87 ± 4	86	87 ± 4	$t\,(145) = -0.33, p = 0.74$	96	86 ± 4	51	88 ± 4	$t\,(145) = -2.99, p = 0.003$
Sleep timing midpoint (clock time ± hours)	147	04:47 ± 2.1	61	04:52 ± 1.4	86	04:43 ± 2.5	$t\,(145) = 1.72, p = 0.09$	96	04:43 ± 1.4	51	04:53 ± 3.1	$t\,(145) = 1.17, p = 0.24$
Sleep duration variability (%)	147	38 ± 15	61	38 ± 18	86	37 ± 13	$t\,(145) = 0.27, p = 0.79$	96	36 ± 13	51	42 ± 19	$t\,(145) = -2.25, p = 0.03$
Sleep timing midpoint variability (%)	147	45 ± 26	61	40 ± 24	86	49 ± 27	$t\,(145) = -1.94, p = 0.05$	96	42 ± 26	51	51 ± 26	$t\,(145) = -1.86, p = 0.06$

Abbreviations: BMI, body mass index; CRP, C-reactive protein; FMI, fat mass index; HOMA-IR, homeostatic model assessment-insulin resistance.

TABLE 2 Associations between sleep duration, sleep efficiency, sleep timing midpoint, sleep duration variability and sleep timing midpoint variability with adiposity and serum cardiometabolic outcomes, the RIGHT Track Health study, North Carolina, USA, 2014–2018.

	Multivariable-adjusted linear regression model results, β (95% CI); p-value	Included covariates
Sleep duration		
Log-transformed BMI	−0.03 (−0.05, 0.003); 0.08	Race
Log-transformed FMI	−0.02 (−0.10, 0.06); 0.69	Number of sleep days, Sex, Race
Log-transformed leptin	−0.09 (−0.25, 0.08); 0.30	Sleep timing midpoint, Sex, Race, Sleep timing midpoint variability
Log-transformed CRP	−0.03 (−0.12, 0.06); 0.49	Race, Sex
Log-transformed HOMA-IR	**−0.10 (−0.19, −0.01); 0.02**	Race
Sleep efficiency		
Log-transformed BMI	0.01 (−0.004, 0.01); 0.25	Race, Sleep duration
Log-transformed FMI	0.01 (−0.01, 0.04); 0.25	Number of sleep days, Sex, Race
Log-transformed leptin	0.01 (−0.04, 0.06); 0.58	Sleep timing midpoint, Sex, Race
Log-transformed CRP	0.01 (−0.02, 0.04); 0.46	Sex, Race
Log-transformed HOMA-IR	−0.01 (−0.03, 0.02); 0.63	Sleep duration, Race
Sleep timing midpoint		
Log-transformed BMI	-5.19^{-09} (-1.30^{-08}, 2.63^{-09}); 0.19	Race, Sleep duration, Sleep timing midpoint variability
Log-transformed FMI	-1.33^{-08} (-3.54^{-08}, 8.84^{-09}); 0.24	Number of sleep days, Sex, Race, Sleep timing midpoint variability
Log-transformed leptin	-3.71^{-08} (-7.52^{-08}, 1.02^{-09}); 0.06	Race, Sex
Log-transformed CRP	-1.41^{-08} (-3.62^{-08}, 7.97^{-09}); 0.21	Sex, Race
Log-transformed HOMA-IR	-8.05^{-09} (-2.89^{-08}, 1.27^{-08}); 0.45	Race, Sleep duration
Sleep duration variability		
Log-transformed BMI	−0.002 (−0.003, 0.002); 0.88	Race, Sleep duration
Log-transformed FMI	0.001 (−0.01, 0.01); 0.81	Number of sleep days, Sex, Race
Log-transformed leptin	−0.01 (−0.02, 0.01); 0.34	Sleep timing midpoint, Sex, Race
Log-transformed CRP	0.003 (−0.005, 0.01); 0.49	Sex, Race
Log-transformed HOMA-IR	−0.003 (−0.01, 0.004); 0.37	Sleep duration, Race
Sleep timing midpoint variability		
Log-transformed BMI	−0.001 (−0.002, 0.001); 0.33	Race, Sleep duration
Log-transformed FMI	−0.003 (−0.01, 0.002); 0.22	Number of sleep days, Sex, Race
Log-transformed leptin	−0.01 (−0.02, 0.004); 0.23	Sleep timing midpoint, Sex, Race
Log-transformed CRP	0.002 (−0.002, 0.01); 0.32	Sex, Race
Log-transformed HOMA-IR	0.001 (−0.003, 0.01); 0.65	Sleep duration, Race

Note: Bold indicates statistically significant based on the p-value cut-off of 0.05.
Abbreviations: BMI, body mass index; CRP, C-reactive protein; FMI, fat mass index; HOMA-IR, homeostatic model assessment-insulin resistance.

was good (87%), and was significantly higher in Black, multiracial/other race individuals compared with White individuals. Sleep duration variability was also significantly greater in Black, multiracial/other race individuals compared with White individuals. No significant differences in sleep timing midpoint and sleep timing midpoint variability were noted between sex and race categories. Regarding adiposity and serum cardiometabolic outcomes, significantly greater FMI and leptin were noted in females compared with males. Black, multiracial/other race individuals had significantly greater BMI, FMI, HOMA-IR and CRP compared with White individuals.

3.1 | Associations between sleep variables with adiposity and serum cardiometabolic outcomes

Results from adjusted linear regression models for associations between continuous sleep variables with adiposity and serum cardiometabolic outcomes are presented in Table 2. A significant inverse association was noted between sleep duration and HOMA-IR, suggesting that participants with lower sleep durations have greater HOMA-IR. No other significant associations were noted between continuous sleep variables with adiposity and serum cardiometabolic

outcomes. Associations between combined sleep duration and sleep timing midpoint groups with adiposity and serum cardiometabolic outcomes are presented in Table 3. BMI, CRP and HOMA-IR were significantly greater in the early-bed/early-rise group compared with the early-bed/late-rise group (referent). No significant differences in adiposity and serum cardiometabolic markers were noted between the late-bed/late-rise and late-bed/early-rise groups when compared with the early-bed/late-rise group (referent).

3.2 | Effect modification results

Sex modified associations between sleep efficiency with FMI ($p_{interaction} = 0.03$), leptin ($p_{interaction} = 0.03$) and CRP ($p_{interaction} = 0.02$). Sex also modified associations between sleep duration variability with CRP ($p_{interaction} = 0.095$). Results from stratified analyses are presented in Table 4, and revealed a significant positive association between sleep efficiency and CRP in males, but not females.

Race modified associations between sleep duration with BMI ($p_{interaction} = 0.07$), FMI ($p_{interaction} = 0.096$) and leptin ($p_{interaction} = 0.06$). Stratified analyses revealed significant inverse associations between sleep duration with BMI and leptin in Black, multiracial/other race individuals, but not White individuals (Table 4). Additionally, race modified the association between sleep duration variability with CRP ($p_{interaction} = 0.08$), and stratified analyses revealed a significant positive association between sleep duration variability and CRP in White individuals, but not Black, multiracial and other race individuals (Table 4).

4 | DISCUSSION

This study examined cross-sectional associations between multiple actigraphy-assessed sleep variables with adiposity and serum cardiometabolic outcomes in a sample of emerging adults with a very high prevalence of short sleep duration (~90% had < 7 h of sleep per night). We also explored potential effect modification of these

TABLE 3 Associations between combined sleep duration and sleep timing midpoint categories with adiposity and serum cardiometabolic outcomes, the RIGHT Track Health study, North Carolina, USA, 2014–2018.

	Mean ± SD	Multivariable-adjusted linear regression model results, β (95% CI); p-value	Included covariates
Log-transformed BMI			Race
Early-bed/late-rise ($n = 37$)	3.18 ± 0.18	Referent	
Early-bed/early-rise ($n = 37$)	3.31 ± 0.25	**0.13 (0.03, 0.23); 0.01**	
Late-bed/late-rise ($n = 37$)	3.24 ± 0.27	0.06 (−0.04, 0.16); 0.23	
Late-bed/early-rise ($n = 36$)	3.24 ± 0.22	0.07 (−0.03, 0.18); 0.16	
Log-transformed FMI			Number of sleep days
Early-bed/late-rise ($n = 37$)	1.79 ± 0.64	Referent	Sex
Early-bed/early-rise ($n = 37$)	1.90 ± 0.69	0.25 (−0.05, 0.55); 0.10	Race
Late-bed/late-rise ($n = 37$)	1.88 ± 0.82	0.14 (−0.15, 0.43); 0.35	Sleep efficiency
Late-bed/early-rise ($n = 36$)	1.68 ± 0.74	0.21 (−0.11, 0.53); 0.19	
Log-transformed leptin			Race
Early-bed/late-rise ($n = 34$)	8.97 ± 1.40	Referent	Sex
Early-bed/early-rise ($n = 35$)	8.89 ± 1.63	0.32 (−0.26, 0.90); 0.28	
Late-bed/late-rise ($n = 33$)	8.94 ± 1.34	0.04 (−0.54, 0.63); 0.88	
Late-bed/early-rise ($n = 35$)	8.18 ± 1.56	−0.11 (−0.71, 0.48); 0.70	
Log-transformed CRP			Race
Early-bed/late-rise ($n = 34$)	0.50 ± 0.50	Referent	Sex
Early-bed/early-rise ($n = 35$)	0.81 ± 0.81	**0.35 (0.02, 0.68); 0.04**	
Late-bed/late-rise ($n = 33$)	0.66 ± 0.71	0.17 (−0.15, 0.50); 0.29	
Late-bed/early-rise ($n = 35$)	0.57 ± 0.70	0.17 (−0.16, 0.51); 0.31	
Log-transformed HOMA-IR			Race
Early-bed/late-rise ($n = 33$)	1.69 ± 0.55	Referent	
Early-bed/early-rise ($n = 35$)	2.09 ± 0.88	**0.38 (0.07, 0.69); 0.02**	
Late-bed/late-rise ($n = 33$)	1.85 ± 0.60	0.17 (−0.15, 0.48); 0.30	
Late-bed/early-rise ($n = 35$)	1.84 ± 0.52	0.17 (−0.14, 0.48); 0.29	

Note: Bold indicates statistically significant based on the p-value cut-off of 0.05.
Abbreviations: BMI, body mass index; CRP, C-reactive protein; FMI, fat mass index; HOMA-IR, homeostatic model assessment-insulin resistance.

TABLE 4 Associations between continuous sleep variables with adiposity and serum cardiometabolic outcomes, stratified by sex and race categories,[a] the RIGHT Track Health study, North Carolina, USA, 2014–2018.

	Multivariable-adjusted linear regression model results, *β* (95% CI); *p*-value			Multivariable-adjusted linear regression model results, *β* (95% CI); *p*-value	
Sleep efficiency	**Males**	**Females**	**Sleep duration**	**White racial descent**	**Black and multiracial/other racial descent**
FMI	0.04 (−0.005, 0.09); 0.08	−0.003 (−0.03, 0.02); 0.85	BMI	0.0004 (−0.03, 0.04); 0.98	**−0.05 (−0.10, −0.003); 0.04**
Leptin	0.07 (−0.02, 0.17); 0.12	−0.04 (−0.09, 0.02); 0.18	FMI	0.06 (−0.04, 0.16); 0.24	−0.07 (−0.22, −0.07); 0.30
CRP	**0.05 (0.01, 0.08); 0.01**	−0.02 (−0.06, 0.02); 0.43	Leptin	0.05 (−0.17, 0.28); 0.62	**−0.29 (−0.53, −0.05); 0.02**
Sleep duration variability			Sleep duration variability		
CRP			CRP		
	−0.01 (−0.01, 0.004); 0.23	0.01 (−0.002, 0.02); 0.12		**0.01 (0.0002, 0.02); 0.04**	−0.01 (−0.02, −0.01); 0.42

Note: Bold indicates statistically significant based on the *p*-value cut-off of 0.05.
Abbreviations: BMI, body mass index; CRP, C-reactive protein; FMI, fat mass index.
[a]Stratified analyses were only conducted if the interaction term added to the final multivariable model reached $p < 0.10$. Statistical significance for the stratified results was set at $p < 0.05$.

associations by sex and race. While population-based data from the National Health Interview Survey (NHIS) in 2017 noted that ~67% of US adults reported sleeping on average ≥ 7 h per night (Sheehan et al., 2019), accumulating evidence in adolescents and emerging adults suggests that these individuals are more likely to have shorter sleep durations (Casper-Gallop, 2022; Maslowsky & Ozer, 2014) and greater day-to-day variability in sleep patterns (Morales-Ghinaglia & Fernandez-Mendoza, 2023). Our findings add to this literature by suggesting that sleep habits in emerging adults fall short of minimum guidelines, making this an essential developmental period to examine and intervene on sleep behaviours to help reduce obesity and cardiometabolic disease risk.

A significant inverse association between sleep duration and HOMA-IR was noted in the entire sample, suggesting that those with shorter sleep durations have less advantageous HOMA-IR/greater insulin resistance. These results corroborate prior findings in emerging adults (Fernström et al., 2020), and provide further evidence of an association between short sleep duration and HOMA-IR as a risk factor for cardiometabolic disease in this population.

Additionally, BMI, HOMA-IR and CRP were significantly greater in the early-bed/early-rise group compared with the early-bed/late-rise group, whereas no significant differences were noted with the "late-bed" groups. Recent studies have also reported that having an early-bedtime/early-wake-time was associated with higher BMI and waist circumference in female emerging adults (Zerón-Rugerio et al., 2020), and that adolescents with earlier waketime and sleep timing midpoint had higher FM 1 year later (LeMay-Russell et al., 2021). Chronotype, which varies with age, may partially explain these associations in adolescents and emerging adults. Indeed, children have earlier chronotypes, which gradually delay until reaching a peak of "lateness" during emerging adulthood and then gradually shift earlier with advancing age (McMahon et al., 2018; Roenneberg et al., 2007). While chronotype was not assessed in the present study, better aligning sleep timing with chronotype (which tends to be delayed during adolescence and emerging adulthood) in addition to promoting adequate sleep durations may be associated with lower obesity and cardiometabolic risk.

Our effect modification results revealed a significant positive association between sleep efficiency and CRP in males, but not females. The reason for these sex differences or for the positive association between sleep efficiency and CRP are unclear. Richardson and Churilla (2017) did note that males who reported sleeping ≤ 6 h per night had higher odds of elevated CRP when compared with males reporting 7–8 h of sleep per night, whereas this association was non-significant in females. Although the association between sleep duration and CRP was non-significant, and sleep duration was considered as a covariate in the sleep efficiency-CRP model, it is possible that higher sleep efficiencies may be due to sleep deprivation or prolonged wakefulness prior to bed (i.e. individuals with shorter sleep durations may have greater sleep efficiencies because of prolonged wakefulness/tiredness prior to sleeping). Furthermore, the very high prevalence of short sleep durations in our sample likely undermined this underlying sleep duration-CRP association.

Our effect modification results also revealed that the inverse association between sleep duration with BMI and leptin was significant in Black, multiracial/other race individuals, but not White individuals. These results complement findings from the NHIS that reported higher odds of obesity among Black short duration sleepers compared with White short duration sleepers (Donat et al., 2013), as well as a recent study that noted that both short and long sleep durations were significantly associated with obesity in Black college students only (Sa et al., 2020). We also noted a significant positive association between sleep duration variability and CRP in White individuals, but not Black, multiracial/other race individuals. These results corroborate those reported by Park et al. (2020), where the positive association between sleep duration variability and CRP was present in European American youth, but not in Asian and Latino youth. The reasons for these racial differences in sleep-obesity/inflammation associations are not well understood, and are likely multifactorial and systemic (Donat et al., 2013; Koren & Taveras, 2018). For instance, Park et al. (2020) suggested that additional sources of stress that are more common in minoritized populations (e.g. poverty, discrimination) may blunt the influence of sleep on inflammation. Additional studies are needed to focus on individual- and systemic-level "stress" factors that may contribute to these racial differences in sleep-obesity and cardiometabolic risk associations.

This study had several strengths and limitations. Strengths included a focus on multiple actigraphy-assessed sleep variables in a population-based sample of emerging adults with a very high prevalence of short sleep duration and the consideration of potential effect modification by sex and race. Limitations include the cross-sectional nature of the analyses, which limits our ability to identify sleep variables as a potential cause of obesity/weight gain and/or cardiometabolic risk, particularly during a transitive period of continued weight change such as emerging adulthood (Barbour-Tuck et al., 2018). The homogeneity of sleep duration patterns in this sample (i.e. ~90% of this sample had < 7 h of sleep per night) likely limited the strength of associations between sleep duration with adiposity and serum cardiometabolic outcomes. Information on self-reported bed- and wake-times was not collected with the accelerometer log, meaning that this information could not be used to verify bed- and wake-times detected with the Sadeh algorithm. To help mitigate this issue, all individual days with < 13 h of wear time were removed from the analyses. Furthermore, menstrual cycle phase was not reported or accounted for during data collection in females, which may undermine some of the associations between sleep and serum cardiometabolic markers (leptin and CRP) that vary across the menstrual cycle. Lastly, we had to combine Black racial descent with multiracial/other race categories because only eight participants identified as multiracial/other racial descent.

5 | CONCLUSION

Our results add to accumulating evidence that short sleep duration is prevalent in emerging adults (Casper-Gallop, 2022; Maslowsky & Ozer, 2014), and indicate that shorter sleep duration, particularly when combined with earlier sleep timing midpoint (< 04:39 hours), is associated with greater adiposity and serum cardiometabolic outcomes. Our results also suggest that the association between shorter sleep duration and these outcomes may be stronger in Black, multiracial/other race individuals, whereas greater sleep duration variability and sleep efficiency are associated with higher CRP in White individuals and males, respectively. Additional longitudinal evidence is needed to corroborate these findings in emerging adults, with a particular need to focus on individual- and contextual-level factors that may influence both sleep health and cardiometabolic disease risk in emerging adults.

AUTHOR CONTRIBUTIONS

Jessica McNeil: Writing – original draft; writing – review and editing; formal analysis. **Nathaniel T. Berry:** Formal analysis; writing – review and editing. **Jessica M. Dollar:** Conceptualization; investigation; funding acquisition; writing – review and editing. **Lenka H. Shriver:** Conceptualization; investigation; funding acquisition; writing – review and editing. **Susan P. Keane:** Writing – review and editing; funding acquisition; investigation; conceptualization. **Lilly Shanahan:** Conceptualization; investigation; funding acquisition; writing – review and editing. **Laurie Wideman:** Conceptualization; investigation; funding acquisition; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declared no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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