Sleep, Lifestyle Behaviors, and Cardiometabolic Health Markers in Overweight/Obese Young Adults: A Pilot Study Using the SenseWear[®] Armband

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Abstract

Aim: This pilot study examined associations between sleep quality and metabolic risk profiles, underlying hormones, inflammatory markers, and behaviors in overweight and obese young adults, aged 18–29 years. **Design:** Cross-sectional, descriptive, correlational study design. **Method:** A partial sample ($n = 29$) was re-recruited from a parent study on screening for risk of early-onset diabetes. BodyMedia's SenseWear[®] armband was used to assess sleep quality. Based on the percentage of consolidated sleep days during the past week, participants were classified as poor, fair, or good sleepers. Multiple multivariate general linear models were used to examine group differences in study variables after adjusting for obesity impact. **Results:** There were no significant differences among groups in age (mean 23.5 ± 2.9 years) or body mass index (mean 38.0 ± 8.9 kg/m²). Good sleepers ($n = 12$, 41.4%) showed the longest nocturnal sleep duration ($7:53 \pm 1:00$ hr), followed by fair ($n = 12$, 41.4%, $7:23 \pm 1:34$ hr) and poor sleepers ($n = 5$, 17.2%, $5:34 \pm 0:56$ hr). Poor sleepers showed the highest plasma resistin ($\eta^2 = .103$) and ghrelin ($\eta^2 = .205$) levels and caloric ($\eta^2 = .255$) and sodium ($\eta^2 = .156$) intakes. No differences in clinical metabolic markers or serum leptin or adiponectin were observed. **Conclusions:** Overweight/obese young adults had irregular sleep schedules and patterns, indicators of poor sleep quality, that were possibly associated with changes in dietary behaviors and underlying plasma hormones. In addition to traditional clinical cardiometabolic markers, plasma resistin and ghrelin may be good predictors of heightened vulnerability to cardiometabolic diseases in overweight/obese young adults with poor-quality sleep.

Keywords

sleep quality, cardiometabolic health, overweight/obese young adults, hormonal changes

Sleep quality has long been considered an important predictor of health; however, the defining characteristics of good-quality sleep have only recently been identified (Ohayon et al., 2017; Reed & Sacco, 2016). In 2017, the National Sleep Foundation released age-specific guidelines for assessing sleep quality using a total of 12 indicators. Variables related to sleep continuity (sleep latency, frequency of awakenings lasting greater than 5 min, wake after sleep onset, and sleep efficiency), sleep architecture (rapid eye movement, Stages 1–3, arousal), and napping (frequency of naps per 24 hr, nap duration, and days per week with at least one nap) were included (Ohayon et al., 2017).

Young adults, aged 18–29 years, transitioning between adolescence and adulthood often voluntarily forgo sleep to meet the challenges of changing roles and perceived mature expectations (Nelson, Story, Larson, Neumark-Sztainer, & Lytle, 2008; Unwin, Goodie, Reamy, & Quinlan, 2013). Their “night owl” habits are closely associated with behaviors influencing sleep: binge drinking (≥ 5 drinks per day); misuse of substances

like tobacco, alcohol, and caffeine; excessive electronic media use; and daytime physical inactivity (National Sleep Foundation, 2014; Schoenborn & Adams, 2008). Not surprisingly, these behaviors influence young adults' sleep quality. Young adults, aged 18–29 years, show the highest prevalence of sleep disturbance (68%), unintended daytime sleep (44%),

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medication use to stay awake (11%), and the lowest overall sleep quality (37%) of any age-group, according to the 2002 “Sleep in America” poll, the most recent available sleep data in this age-group (National Sleep Foundation, 2002).

Poor-quality sleep increases the levels of proinflammatory markers (C-reactive protein and tumor necrosis factor- α [TNF- α]; Engin, 2017). Sleep quality also affects levels of adipocyte-derived hormones (leptin, adiponectin, and resistin), gut-derived hormones (ghrelin, glucagon-like peptide-1 [GLP-1]), and hormones that directly participate in carbohydrate metabolism like insulin (Guarino, Nannipieri, Iervasi, Taddei, & Bruno, 2017; Morselli, Guyon, & Spiegel, 2012).

When young adults have poor-quality sleep, their tissues exhibit resistance to the effects of insulin and leptin (DeBoer, 2013; Lopez-Jaramillo et al., 2014). Leptin resistance activates the sympathetic nervous system, leading to increased cortisol release, endothelial dysfunction, altered lipid metabolism, and decreased insulin but not diminished appetite (Engin, 2017; Morselli et al., 2012). Insulin insensitivity leads to inappropriately regulated glucose metabolism and early B-cell exhaustion, resulting in early-onset type 2 diabetes and left ventricular hypertrophy (Carnethon et al., 2013).

Overweight and obese young adults with poor-quality sleep, therefore, have a heightened risk of hypertension, diabetes, and cardiovascular diseases due to the independent, additive risk factors of obesity, sleep, and unhealthy dietary habits induced by appetite-related hormonal imbalances and changes in adipokines (Engin, 2017; Morselli et al., 2012). However, sleep is rarely included in cardiometabolic prevention strategies (Coughlin & Smith, 2014), and many overweight and obese young adults with poor-quality sleep are unaware of their heightened vulnerability to cardiometabolic diseases. Consequently, there are few sleep studies of community-dwelling overweight and obese young adults and even fewer studies assessing sleep quality using objective measures.

In the present pilot study, we examined the associations between sleep quality, measured using BodyMedia's SenseWear[®] armband, and clinical metabolic risk profiles, underlying hormones, physical activity, and dietary behaviors in overweight and obese young adults. Specifically, we compared sleep duration and patterns (i.e., frequency of awakening > 5 min, duration of awakening during sleep, nap frequency, and ranges of nap time), cardiometabolic risk factors (body mass index [BMI], blood pressure, lipids, fasting glucose and insulin, glycated hemoglobin [HbA1C]), underlying hormones (leptin, adiponectin, resistin, ghrelin, GLP-1), inflammatory markers (C-reactive protein, TNF- α), dietary behaviors, and physical activity in overweight or obese young adults who were poor-, fair-, or good-quality sleepers.

Method

Design

We used a cross-sectional, descriptive correlational design for this pilot study.

Participants

Prior to beginning the study, we obtained approval from the university's institutional review board. We re-recruited a subsample ($n = 30$) from a parent study aimed at screening for early-onset type 2 diabetes risk in young adults, aged 18–29 years (K01NR012779). Those eligible for the parent study were community-dwelling young adults who were overweight or obese ($\text{BMI} \geq 25 \text{ kg/m}^2$) and sedentary (leisure-time physical activity < 90 min/week during the past month). After a phone screening, we invited eligible young adults ($n = 106$) to the Clinical Research Unit of Atlanta Clinical & Translational Science Institute (ACTSI) to complete anthropometric assessments, behavioral surveys, and fasting blood draws. The details of the participant recruitment for the parent study are described in a previous publication (Cha et al., 2014).

Data Collection

Once participants completed data collection for the parent study, those interested in participating in the present substudy received a SenseWear armband model MF-SW (BodyMedia, Inc., Pittsburgh, PA) and a self-addressed stamped mailer, with instructions to wear the armband for 7 days continuously, except when taking a shower or swimming. The research team called participants on the first day to remind them how to accurately wear the armband and answer any questions. After 7 days, we called the participants again to remind them to return the armband.

Variables and Measures

During the ACTSI visit, a trained nurse from the ACTSI collected anthropometric data and blood samples, and research staff collected survey data.

Anthropometric data and biomarkers. To determine BMI (kg/m^2), we used height and weight measurements. Seated blood pressure (BP) was measured using American Heart Association standard guidelines. Waist circumference (WC) was measured with a tape measure to assess visceral fat. Fasting blood samples were collected to measure plasma glucose, insulin, lipids (total cholesterol, high-density lipoprotein [HDL], low-density lipoprotein [LDL], triglycerides, and free fatty acids), hormones affecting glucose metabolism (GLP-1, leptin, ghrelin, resistin, and adiponectin), proinflammatory markers (TNF- α , C-reactive protein), and HbA1c. Plasma samples were processed, and results were reported by a certified Biomarker Core Laboratory. We calculated insulin resistance using the homeostasis model of assessment formula: fasting insulin ($\mu\text{U/ml}$) $\times$ fasting glucose (mg/dl)/405 (Matthews et al., 1985). We also calculated the ratio of insulin to glucose levels. We provide a brief review of the impacts obesity and poor-quality sleep have on hormones in Table 1.

Table 1. Hormonal Functions and Effects in Persons With Obesity or Poor Sleepers.

Hormone	Function	In Obesity	In Poor-Quality Sleep
Insulin (a peptide released from beta cells of the pancreas)	<ul style="list-style-type: none"> • ↓ Blood glucose • ↑ Protein synthesis • ↑ FFA and triglycerides formation 	<ul style="list-style-type: none"> • ↑ Insulin release (hyperinsulinemia) • ↓ Insulin sensitivity 	<ul style="list-style-type: none"> • ↑ Insulin release • ↓ Insulin sensitivity
Glucagon-like peptide-I (GLP-I, a peptide that is released from cells in the intestine)	<ul style="list-style-type: none"> • ↑ Insulin secretion and sensitivity • ↓ Blood glucose and glucagon secretion • Satiety signal in response to food intake, decreased hunger 	<ul style="list-style-type: none"> • ↓ Secretion 	<ul style="list-style-type: none"> • ↓ Secretion in persons with fragmented sleep
Adiponectin (a protein released from adipocytes)	<ul style="list-style-type: none"> • ↓ Appetite • ↑ Insulin sensitivity • ↓ Atherosclerosis • Anti-inflammatory and anti-atherogenic effects 	<ul style="list-style-type: none"> • ↓ Secretion • ↓ Expression • ↓ Level in plasma 	<ul style="list-style-type: none"> • OSA is linked to ↓ expression and/or ↓ secretion
Leptin (a peptide released from adipocytes; satiety hormone)	<ul style="list-style-type: none"> • ↓ Appetite due to effects at hypothalamic receptors • Activates descending autonomic pathways that control energy expenditure 	<ul style="list-style-type: none"> • ↑ Secretion • ↑ Circulating levels of leptin • ↓ Leptin sensitivity 	<ul style="list-style-type: none"> • ↓ Leptin levels • ↓ Amplitude and duration of leptin diurnal rhythm in healthy males
Ghrelin (a polypeptide released from the stomach; counter-regulatory hormone to leptin; hunger hormone)	<ul style="list-style-type: none"> • ↑ Appetite, growth hormone secretion, initiates feeding activity and prepares body for food intake • ↑ Gastric acid secretion and gastric motility • ↓ Proatherogenic changes in cells, ↓ proinflammatory mechanisms; • ↑ Anti-inflammatory mechanisms 	<ul style="list-style-type: none"> • ↓ Circulating levels of ghrelin • ↑ Ghrelin sensitivity, obesity may result in ghrelin supersensitivity 	<ul style="list-style-type: none"> • ↓ Total ghrelin levels • ↑ Ghrelin levels in short sleepers • ↑ Ghrelin levels in patients with OSA
Resistin (a polypeptide cytokine believed to be released from macrophages in humans)	<ul style="list-style-type: none"> • ↑ Expression of suppressor of cytokine signaling-3 • Inhibits insulin signaling and leads to insulin resistance • ↑ LDL and ↑ risk of heart disease • A positive correlation between serum resistin and proinflammatory markers 	<ul style="list-style-type: none"> • ↑ Resistin in patients with diabetes • A positive correlation between serum resistin and inflammatory factors 	<ul style="list-style-type: none"> • Resistin is not correlated with poor sleep quality
TNF- α (a cytokine involved in systemic inflammation, primarily produced by activated macrophages)	<ul style="list-style-type: none"> • Positive relationship with resistin • Suppresses appetite (in response to perceived infection) • ↑ Insulin resistance • May act on adipocytes to regulate release of leptin 	<ul style="list-style-type: none"> • Overexpression of TNF-α in the adipose tissue of obese persons 	<ul style="list-style-type: none"> • ↑ TNF-α following acute sleep deprivation (one night of sleep loss)

Note. This table was created based on the articles of Engin (2017), Guarino, Nannipieri, Iervasi, Taddei, and Bruno (2017), and Morselli, Guyon, and Spiegel (2012). FFA = free fatty acids; LDL = low-density lipoprotein; OSA = obstructive sleep apnea; TNF- α = tumor necrosis factor- α .

Sleep measures. Subjective assessments of participants' sleep duration were made with 3 items (participants' bedtime, time awakened, and average nocturnal sleep duration during the past month) in the sociodemographic questionnaire. Objective sleep quantity and quality were assessed with the SenseWear armband (armband model MF-SW, software V8.0), a triaxial actimetry monitor of sleep and physical activity. Prior research has validated use of the SenseWear armband in both children and adults (O'Driscoll, Turton, Copland, Strauss, & Hamilton, 2013; Soric et al., 2013). Skin sensors (for temperature, heat flux, and electrical resistance) and time recordings coupled with accelerometer readings allowed researchers to differentiate between participants' sleep and inactivity conditions.

Using the SenseWear Professional Software V8.0 guide and guidance from sleep experts experienced with the SenseWear armband (the last author and a consultant), we calculated the durations of sleep fragments. Body position (lying down), energy expenditure (metabolic equivalent task [MET] < 2), step counts (<10 steps/min over 10 or more consecutive minute), and time stamp were used to determine when patients were lying down and sleeping and to calculate the frequency and duration of these conditions. We then calculated the durations and frequency of naps and nocturnal sleep, the frequency of awakening for >5 min, and the duration of time awake during the sleep period.

We classified sleep-quality groups based on sleep patterns and the number of sleep fragments. Since many of the young

adults had sleep schedules characterized by going to bed very late at night (e.g., 4:42 a.m.) and getting up very late (e.g., 12:40 p.m.) and/or taking long naps (>2 hr), we included both daytime naps and nocturnal sleep durations as sleep fragments. To determine how consolidated the participants' sleep patterns were, we considered the number of sleep fragments. Adjacent sleep fragments were defined as consolidated if intervening awake times were less than 30 min, but treated as distinct sleep fragments if intervening awake times were greater than 30 min. We distinguished naps from the nocturnal sleep period using the following criteria: (1) a period of sleep occurring at least 3 hr before the upcoming nocturnal sleep period or 3 hr past the end of the previous nocturnal sleep period, and (2) daytime sleep according to time stamp.

Survey Questionnaires

Sociodemographics. We collected sociodemographic characteristics, including age, ethnicity, years of education, employment status, smoking, alcohol consumption, and binge drinking habits during the past month, using a sociodemographic questionnaire developed by the first author.

Dietary behaviors. We measured dietary behaviors with the Youth/Adolescent Food Frequency Questionnaire (FFQ), a youth version of the Willett FFQ from the Nurses' Health Study (Rockett et al., 1997), as recommended by Harvard University School of Public Health. Once we obtained preliminary data from the Harvard University School of Public Health that included a nutritional breakdown of the participants' FFQs, we calculated a Dietary Quality Index Revised for young adults score to assess overall dietary quality, caloric intake, caffeine intake, added sugar intake, and serving sizes (Cha et al., 2014; Newby et al., 2003).

Physical activity. We assessed physical activity with the 7-Item Modifiable Activity Questionnaire, which researchers used in the Diabetes Prevention Program (Kriska, 1997). Then, we calculated the activity as metabolic equivalents (MET)-hour per week (Kriska, 1997).

Data Analysis

Only participants who wore the SenseWear armband for a minimum of 15 hr per day (including the 9:00 p.m. to 9:00 a.m. time period) and at least 3 days of 7 were included in our analysis. We excluded one participant due to inadequate duration of time spent wearing the armband. Thus, 29 participants remained for final data analysis.

Descriptive summaries are reported for demographic data. Continuous variables are reported as means and standard deviations; categorical variables are reported as frequencies and percentages. Based on the percentage of days during the past 7 days in which participants reported consolidated sleep (i.e., sleep was not fragmented), the participants were assigned to poor (<33.3% consolidated sleep), fair (33.4–66.6%

consolidated sleep), or good ($\geq 66.7\%$ consolidated sleep) sleep groups. Age, BMI, dietary and physical activity variables, hormones, cardiometabolic risk profiles, and inflammatory makers were compared among groups using Kruskal–Wallis nonparametric test (a one-way analysis of variance on rank). This approach allows the researchers to test whether samples originate from the same distribution and compare the group differences despite different sample sizes. The Levene's test was also performed to evaluate the equality of variance. A multiple general linear model was constructed to adjust for the obesity effect and thus examine the sleep groups and their associations with anthropometric measures and biomarkers after adjusting for BMI. We used BMI instead of waist circumference to assess the impact of obesity on the study variables because we recognized that accurate assessment of waist circumference in severely obese ($\text{BMI} > 35 \text{ kg/m}^2$) persons is very challenging due to varying distribution of body fat. A p value of $<.05$ was set, and due to the small sample size, the effect size (partial eta square [η^2]: .02 for small, .13 for medium, and .26 for large) was also reported to indicate potentially significant variables for a larger study in the future. All data analyses were performed using IBM SPSS version 23.0.

Results

Sociodemographics

The majority of participants in this study were African American ($n = 22$, 75.9%) and female ($n = 21$, 72.4%). The mean age and mean BMI were 23.5 ± 2.9 years and $38.0 \pm 8.7 \text{ kg/m}^2$, respectively. Half of the participants were full-time students ($n = 15$, 51.7%) and 65.5% ($n = 19$) never smoked. Mean self-reported daily sleep time during the past month was 6.3 ± 1.4 hr (range: 4–10 hr). Since all participants were obese, metabolic aberration was common. The most prevalent metabolic aberration was low HDL ($n = 24$, 82.8%), followed by elevated systolic blood pressure ($n = 10$, 34.5%), HbA1C ($n = 9$, 31.0%), and LDL ($n = 8$, 27.6%).

Sleep Duration, Quality, and Patterns

Table 2 provides the normal or recommended values for sleep, clinical, behavioral, and cardiometabolic variables and compares the values observed by group in the present study. The most common bedtimes were between 12:01 a.m. and 2:00 a.m. (66 of 144 observations; 45.8%), followed by bedtimes after 2:01 a.m. (43 of 144 observations; 29.9%). Good sleepers went to bed at consistent and earlier times throughout the week (34.92% had bedtimes before 12:00 a.m.), as compared to fair (21.54% had bedtimes before 12:00 a.m.) and poor (10.53% had bedtimes before 12:00 a.m.; 10.53%) sleepers.

While good sleepers in the present study did not awaken during the nocturnal sleep period (median awakenings = 0), the fair and poor sleepers experienced awakenings almost every night (median awakenings = 3.5 and 4 per night, respectively). Fair and good sleepers had similar nocturnal sleep durations, while poor sleepers obtained less sleep at night. Poor

Table 2. Comparisons of Sleep, Clinical, Behavioral, and Cardiometabolic Variables by Sleep-Quality Group.

Variable	Normal/ Recommended Values	Good Sleepers (n = 12)	Fair Sleepers (n = 12)	Poor Sleepers (n = 5)	Kruskal- Wallis Test p Value	Partial η^2	Adjusted p Value
Age (years)	—	23.7 $\pm$ 3.1	23.9 $\pm$ 2.8	22.1 $\pm$ 2.8	.473	0.054	.468
BMI (kg/m ²)	18.5–24.9	37.9 $\pm$ 8.7	35.5 $\pm$ 7.4	44.5 $\pm$ 10.2	.193	0.117	
Physical activity, median (MET-hour/week)	>10	9.3	2.33	9.69	.067	0.193	
Observed nights (n)	—	5.73	5.0	3.80	—		
Frequency of awakening for >5 min/night	<1	0.50 $\pm$ 0.8	3.7 $\pm$ 0.7	3.40 $\pm$ 1.0	—		
Nocturnal sleep (hour:min)	—	7:53 $\pm$ 1:03	7:23 $\pm$ 1:34	5:34 $\pm$ 0:56	—		
Nap duration, range (hour:min)	0:00	0:46–1:04	0:24–1:24	0:11–2:13	—		
Total caloric intake (Kcal)	1,800–2,000 (W); 2,400–2,600 Kcal (M)	1,594.4 $\pm$ 377.5	1459.9 $\pm$ 496.6	2291.0 $\pm$ 663.3	.028	0.255	.023
Diet quality (DQIR-Y score)	—	60.6 $\pm$ 7.5	53.7 $\pm$ 10.6	63.6 $\pm$ 7.4	.085	0.176	.120
Carbohydrates (g/day)	45–65% of total caloric intake	216.2 $\pm$ 55.6	186.0 $\pm$ 64.5	287.5 $\pm$ 77.6	.058	0.203	.027
Added sugar (g/day)	<25 (W); <37.5 (M)	54.4 $\pm$ 18.4	58.3 $\pm$ 38.5	68.6 $\pm$ 25.0	.686	0.027	.564
Caffeine (mg/day)	—	30.2 $\pm$ 36.8	46.5 $\pm$ 34.5	28.3 $\pm$ 27.8	.411	0.064	.530
Sodium (mg/day)	<1,500–2,300 mg	1,843.6 $\pm$ 565.5	1686.4 $\pm$ 572.5	2832.5 $\pm$ 1007.8	.112	0.156	.039
Alcohol consumption (g/day)	—	1.8 $\pm$ 1.5	1.9 $\pm$ 2.4	3.5 $\pm$ 3.9	.855	0.011	.682
Cholesterol intake (mg/day)	<300	202.1 $\pm$ 82.0	200.2 $\pm$ 114.5	286.1 $\pm$ 115.1	.353	0.074	.471
Fasting glucose (mg/dl)	<100	83.5 $\pm$ 7.5	88.2 $\pm$ 11.3	92.2 $\pm$ 7.0	.090	0.172	.166
Fasting insulin (mIU/ml)	<25	23.4 $\pm$ 7.9	18.8 $\pm$ 5.4	21.7 $\pm$ 4.8	.391	0.067	.243
Ratio of insulin to glucose $\times$ 10	—	2.8 $\pm$ 1.0	2.1 $\pm$ 0.6	2.4 $\pm$ 0.7	.169	0.127	.095
Insulin resistance (HOMA-IR)	—	4.8 $\pm$ 1.7	4.1 $\pm$ 1.5	4.9 $\pm$ 0.8	.404	0.065	.615
Free fatty acid (NEFA; mmol/L)	0.00–0.72	0.7 $\pm$ 0.9	0.8 $\pm$ 1.0	0.4 $\pm$ 0.2	.550	0.043	.917
HbA1C (%)	<5.7	5.5 $\pm$ 0.4	5.4 $\pm$ 0.6	5.3 $\pm$ 0.6	.896	0.008	.635
Systolic blood pressure (mmHg)	<120	125.3 $\pm$ 12.4	118.5 $\pm$ 20.2	127.8 $\pm$ 22.5	.436	0.060	.786
Diastolic blood pressure (mmHg)	<80	75.3 $\pm$ 5.2	69.3 $\pm$ 7.1	73.2 $\pm$ 9.6	.107	0.160	.150
Total cholesterol in serum (mg/dl)	<200	140.3 $\pm$ 31.5	170.1 $\pm$ 35.3	140.8 $\pm$ 27.3	.086	0.175	.116
Triglycerides (mg/dl)	<150	93.1 $\pm$ 79.3	99.6 $\pm$ 57.0	61.2 $\pm$ 20.7	.310	0.084	.592
HDL (mg/dl)	$\geq$ 60	38.7 $\pm$ 10.6	43.3 $\pm$ 8.4	39.7 $\pm$ 5.4	.362	0.073	.563
LDL (mg/dl)	<100	96.9 $\pm$ 31.0	114.1 $\pm$ 33.7	92.0 $\pm$ 24.4	.401	0.065	.514

Note. N = 29. Values are reported as mean $\pm$ SD unless otherwise indicated. The Kruskal–Wallis test was performed in light of the small and unequal sample sizes. Adjusted p values are from the general linear models (GLMs), which were adjusted for BMI effect. Partial η^2 refer to the effect size. If the adjusted p value is significant but the Kruskal–Wallis test is not, it may indicate the need for a larger sample. If the Kruskal–Wallis test is significant but the GLM result is not, it indicates that there was a group difference but that the BMI may be responsible for the difference. BMI = body mass index; DQIR-Y = Dietary Quality Index Revised for young adults; HbA1C = glycated hemoglobin; HDL = high-density lipoprotein cholesterol; HOMA-IR = homeostasis model of assessment-insulin resistance; LDL = low-density lipoprotein cholesterol; M = men; NEFA = nonesterified fatty acids; W = women.

and fair sleepers frequently used daytime naps to compensate for insufficient nocturnal sleep.

Clinical Profiles and Behaviors

There were no differences in mean age (23.5 $\pm$ 2.9, p = .755) or BMI (38.0 $\pm$ 8.9, p = .534) among the three groups. There were also no significant differences among groups with respect

to physical activity although the fair sleepers exhibited the lowest MET-hour/week.

Poor sleepers showed the highest caloric (p = .023, η^2 = .255), carbohydrate (p = .027, η^2 = .203), and sodium (p = .039, η^2 = .156) intakes after adjusting for BMI. However, there were no statistical group differences among the three groups related to alcohol, added sugar, or caffeine consumption and no differences with regard to cardiometabolic risk profiles.

Table 3. Comparisons of Hormones and Inflammatory Markers by Sleep-Quality group.

Variable	Good Sleepers (<i>n</i> = 12)	Fair Sleepers (<i>n</i> = 12)	Poor Sleepers (<i>n</i> = 5)	K-W Test <i>p</i> Value	Partial η^2	Adjusted <i>p</i> Value
Hormones						
Resistin (ng/ml)**	26.2 $\pm$ 18.2	31.3 $\pm$ 19.5	49.8 $\pm$ 31.1	.236	.103	.045
Leptin (pg/ml)	48.2 $\pm$ 33.0	47.4 $\pm$ 35.6	71.3 $\pm$ 31.2	.287	.089	.851
Ghrelin(ng/ml)*	1.3 $\pm$ 0.2	1.3 $\pm$ 0.3	1.6 $\pm$ 0.2	.057	.205	.064
Adiponectin (ng/ml)	6.9 $\pm$ 2.8	8.6 $\pm$ 2.9	7.1 $\pm$ 2.5	.321	.081	.416
GLP-1 (ng/ml)	4.6 $\pm$ 1.4	3.9 $\pm$ 1.3	5.4 $\pm$ 0.8	.062	.199	.126
Inflammatory markers						
C-reactive protein (mg/L)	5.9 $\pm$ 3.9	6.0 $\pm$ 6.1	2.8 $\pm$ 1.2	.450	.057	.320
TNF- α (ng/ml)	2.7 $\pm$ 0.5	3.8 $\pm$ 2.2	3.4 $\pm$ 1.9	.724	.080	.252

Note. (*N* = 29). Values are reported as mean $\pm$ SD unless otherwise indicated. The Kruskal–Wallis (K–W) test was performed in light of the small and unequal sample sizes. Adjusted *p* values are from the general linear models (GLMs), which were adjusted for BMI effect. Partial η^2 refer to the effect size. If the adjusted *p* value is significant but the Kruskal–Wallis test is not, it may indicate the need for a larger sample. If the Kruskal–Wallis test is significant but the GLM result is not, it indicates that there was a group difference but that the BMI may be responsible for the difference. GLP-1 = glucagon-like peptide 1; TNF- α = tumor necrosis factor- α .

p* < .010. *p* < .05.

While fasting insulin level was the highest in good sleepers, there was no group difference. Good sleepers also showed the highest ratio of insulin to glucose, indicating the best insulin secretion and a good response to glucose. Serum cholesterol levels (total cholesterol, triglyceride, and LDL) were highest in fair sleepers but not statistically different from those of other groups (see Table 2).

Comparisons of Underlying Markers: Hormones and Inflammatory Markers

Table 3 lists levels of hormones and inflammatory markers by group. Poor sleepers showed the highest plasma resistin (*p* = .045, η^2 = .103) and ghrelin (*p* = .064, η^2 = .205) values after adjusting for BMI. GLP-1 (*p* = .126, η^2 = .199) showed medium effect size, though the difference was not statistically significant. There were no group differences in adiponectin (*p* = .416) or TNF- α (*p* = .252). Leptin was highest in poor sleepers; however, there was no group difference (*p* = .287 to *p* = .851, η^2 = .089).

Discussion

Curtailed sleep increases metabolic risk, which can lead to the development of cardiometabolic diseases (Bayon, Leger, Gomez-Merino, Vecchierini, & Chennaoui, 2014). In the present study, we addressed the additional importance of the effects of sleep quality on metabolic aberration (Morselli et al., 2012), changes in behaviors, underlying hormones, and inflammatory responses (Engin, 2017; Guarino et al., 2017). Poor-quality sleep in the participating overweight or obese young adults was not significantly associated with traditional clinical cardiometabolic biomarkers (i.e., fasting serum glucose, insulin, lipids, leptin, adiponectin, and blood pressure); however, we observed potentially detrimental differences in behaviors and levels of metabolic hormones and inflammatory markers.

Overweight and obese young adults who were poor sleepers showed the highest plasma levels of resistin and ghrelin as well as the highest consumption of calories, carbohydrates, and sodium. Plasma resistin may be useful in predicting metabolic aberration when used in conjunction with traditional cardiometabolic clinical markers in overweight and obese persons with leptin resistance. Emphasizing appropriate sodium and carbohydrate intake may be a straightforward dietary intervention that could be implemented in this population to improve cardiometabolic health. Future research with a larger sample and over a longer time period is warranted to further investigate the associations among resistin, ghrelin, and dietary behaviors and to examine when, how, and why these small changes are associated with pathophysiologic processes.

Young adults with good-quality sleep will awaken less than one time per night (Ohayon et al., 2017). Troublingly, the majority of participants in the present study had more frequent awakenings (mean awakenings/night = 2.31 $\pm$ 2.69), waking 60–120 min after sleep onset and not returning to sleep for up to 3 hr. The long intervening awake time was associated with a late morning awakening and long (2–3 hr) or frequent (≥ 3 /day) naps. These sleep behaviors might generate a vicious cycle of irregular bedtimes, daytime sleepiness, and delayed sleep over consecutive nights. Educating young adults about appropriate sleep behaviors and good sleep hygiene practices may help to improve their sleep quality.

Poor sleepers showed the highest BMI (although it was not significantly different from other sleep groups) and experienced the most frequent awakenings during sleep in the current study. This finding is consistent with those of previous studies that demonstrated that overweight and obese young adults are prone to more fragmented sleep and are vulnerable to obstructive sleep apnea (OSA; Bayon et al., 2014; Schwartz et al., 2008), with the severity of OSA positively correlated with BMI and neck circumference (Schwartz et al., 2008; Seetho & Wilding, 2014). More work is needed to establish associations

among OSA, sleep quality, and metabolic aberrations in overweight and obese young adults.

The current pilot study is limited in multiple aspects. First, the small sample size and homogeneity of participants (all participants were overweight and obese young adults) make generalizations to normal-weight young adults problematic. Second, while self-report measures have been determined to be valid and reliable, they may be subject to misrepresentation by respondents due to their nature. Third, there was variability in the length of time participants wore the SenseWear armband, despite instructions to wear it 24 hr/day for 7 days, leading to partial or incomplete records of their sleep patterns. Additionally, objective assessment of physical activity (e.g., steps/day) requires measuring sleep and physical activity together over a full 24-hr period of wearing SenseWear armband. Since many participants did not wear the armband for a continuous 24-hr period, the sample size was further reduced, and the evidence regarding physical activity is inconclusive. Finally, it was difficult to distinguish inactivity from a genuine sleep period. Sleep diaries maintained by participants combined with an objective measure like the SenseWear armband might provide more complete pictures of sleep patterns.

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Declaration of Conflicting Interests

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References

Bayon, V., Leger, D., Gomez-Merino, D., Vecchierini, M. F., & Chennaoui, M. (2014). Sleep debt and obesity. *Annals of Medicine*, 46, 264–272. doi:10.3109/07853890.2014.931103

- Carnethon, M. R., Ning, H., Soliman, E. Z., Lewis, C. E., Schreiner, P. J., Sidney, S., & Lloyd-Jones, D. M. (2013). Association of electrocardiographically determined left ventricular mass with incident diabetes, 1985-1986 to 2010-2011: Coronary Artery Risk Development in Young Adults (CARDIA) study. *Diabetes Care*, 36, 645–647. doi:10.2337/dc12-1359
- Cha, E., Kim, K. H., Lerner, H. M., Dawkin, C. R., Bello, M. K., Umpierrez, G., & Dunbar, S. B. (2014). Health literacy, self-efficacy, food label use, and diet in young adults. *American Journal of Health Behavior*, 38, 331–339. doi:10.5993/AJHB.38.3.2
- Coughlin, J. W., & Smith, M. T. (2014). Sleep, obesity, and weight loss in adults: Is there a rationale for providing sleep interventions in the treatment of obesity? *International Review of Psychiatry*, 26, 177–188. doi:10.3109/09540261.2014.911150
- DeBoer, M. D. (2013). Obesity, systemic inflammation, and increased risk for cardiovascular disease and diabetes among adolescents: A need for screening tools to target interventions. *Nutrition*, 29, 379–386. doi:10.1016/j.nut.2012.07.003
- Engin, A. (2017). The pathogenesis of obesity-associated adipose tissue inflammation. *Advances in Experimental Medicine and Biology*, 960, 221–245. doi:10.1007/978-3-319-48382-5_9
- Guarino, D., Nannipieri, M., Iervasi, G., Taddei, S., & Bruno, R. M. (2017). The role of the autonomic nervous system in the pathophysiology of obesity. *Frontiers in Physiology*, 8, 665. doi:10.3389/fphys.2017.00665
- Kriska, A. M. (1997). Modifiable Activity Questionnaire. *Medicine and Science in Sports and Exercise*, 29, S73–S78.
- Lopez-Jaramillo, P., Gomez-Arbelaiz, D., Lopez-Lopez, J., Lopez-Lopez, C., Martinez-Ortega, J., Gomez-Rodriguez, A., & Triana-Cubillos, S. (2014). The role of leptin/adiponectin ratio in metabolic syndrome and diabetes. *Hormone Molecular Biology and Clinical Investigation*, 18, 37–45. doi:10.1515/hmbci-2013-0053
- Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: insulin resistance and β -cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*, 28, 412–419.
- Morselli, L. L., Guyon, A., & Spiegel, K. (2012). Sleep and metabolic function. *Pflügers Archiv*, 463, 139–160. doi:10.1007/s00424-011-1053-z
- National Sleep Foundation. (2002). 2002 “Sleep in America” poll. Retrieved from <http://sleepfoundation.org/sites/default/files/2002SleepInAmericaPoll.pdf>
- National Sleep Foundation. (2014). 2014 Sleep in America poll: Sleep in the modern family: Summary of findings. Retrieved from <http://sleepfoundation.org/sites/default/files/2014-NSF-Sleep-in-America-poll-summary-of-findings—FINAL-Updated-3-26-14-.pdf>
- Nelson, M. C., Story, M., Larson, N. I., Neumark-Sztainer, D., & Lytle, L. A. (2008). Emerging adulthood and college-aged youth: An overlooked age for weight-related behavior change. *Obesity*, 16, 2205–2211. doi:10.1038/oby.2008.365
- Newby, P. K., Hu, F. B., Rimm, E. B., Smith-Warner, S. A., Feskani, D., Sampson, L., & Willett, W. C. (2003). Reproducibility and validity of the diet quality index revised as assessed by use of a food-frequency questionnaire. *American Journal of Clinical Nutrition*, 78, 941–949.

- O'Driscoll, D. M., Turton, A. R., Copland, J. M., Strauss, B. J., & Hamilton, G. S. (2013). Energy expenditure in obstructive sleep apnea: Validation of a multiple physiological sensor for determination of sleep and wake. *Sleep and Breathing*, 17, 139–146. doi:10.1007/s11325-012-0662-x
- Ohayon, M., Wickwire, E. M., Hirshkowitz, M., Albert, S. M., Avigan, A., Daly, F. J., . . . Vitiello, M. V. (2017). National Sleep Foundation's sleep quality recommendations: First report. *Sleep Health: Journal of the National Sleep Foundation*, 3, 6–19. doi:10.1016/j.sleh.2016.11.006
- Reed, D. L., & Sacco, W. P. (2016). Measuring sleep efficiency: What should the denominator be? *Journal of Clinical Sleep Medicine*, 12, 263–266. doi:10.5664/jcsm.5498
- Rockett, H. R., Breitenbach, M., Frazier, A. L., Witschi, J., Wolf, A. M., Field, A. E., & Colditz, G. A. (1997). Validation of a youth/adolescent food frequency questionnaire. *Preventive Medicine*, 26, 808–816. doi:10.1006/pmed.1997.0200
- Schoenborn, C. A., & Adams, P. F. (2008). Sleep duration as a correlate of smoking, alcohol use, leisure-time physical inactivity, and obesity among adults: United States, 2004–2006. *NCHS Health e-Stats*. Retrieved from <http://www.cdc.gov/nchs/data/hestat/sleep04-06/sleep04-06.pdf>
- Schwartz, A. R., Patil, S. P., Laffan, A. M., Polotsky, V., Schneider, H., & Smith, P. L. (2008). Obesity and obstructive sleep apnea: Pathogenic mechanisms and therapeutic approaches. *Proceedings of the American Thoracic Society*, 5, 185–192. doi:10.1513/pats.200708-137MG
- Seetho, I. W., & Wilding, J. P. (2014). Sleep-disordered breathing, Type 2 diabetes and the metabolic syndrome. *Chronic Respiratory Disease*, 11, 257–275. doi:10.1177/1479972314552806
- Soric, M., Turkalj, M., Kucic, D., Marusic, I., Plavec, D., & Misigoj-Durakovic, M. (2013). Validation of a multi-sensor activity monitor for assessing sleep in children and adolescents. *Sleep Medicine*, 14, 201–205. doi:10.1016/j.sleep.2012.11.003
- Unwin, B. K., Goodie, J., Reamy, B. V., & Quinlan, J. (2013). Care of the college student. *American Family Physician*, 88, 596–604.