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Resting metabolic rate varies by race and by sleep duration

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Abstract

Objective—Short sleep duration is a significant risk factor for weight gain, particularly in African Americans and men. Increased caloric intake underlies this relationship but it remains unclear whether decreased energy expenditure is a contributory factor. The current study assessed the impact of sleep restriction and recovery sleep on energy expenditure in African American and Caucasian men and women.

Methods—Healthy adults participated in a controlled laboratory study. After two baseline sleep nights, subjects were randomized to an experimental (n=36; 4h sleep/night for 5 nights followed by 1 night 12h recovery sleep) or control condition (n=11; 10h sleep/night). Resting metabolic rate and respiratory quotient were measured using indirect calorimetry in the morning after overnight fasting.

Results—Resting metabolic rate—the largest component of energy expenditure—decreased after sleep restriction (−2.6%, p=0.032) and returned to baseline levels after recovery sleep. No changes in resting metabolic rate were observed in control subjects. Relative to Caucasians (n=14), African Americans (n=22) exhibited comparable daily caloric intake but a lower resting metabolic rate (p=0.043) and higher respiratory quotient (p=0.013) regardless of sleep duration.

Conclusions—Sleep restriction decreased morning resting metabolic rate in healthy adults, suggesting that sleep loss leads to metabolic changes aimed at conserving energy.

Keywords

Sleep restriction; energy balance; race differences

Epidemiological and laboratory studies have consistently found that short sleep duration is a significant risk factor for weight gain and obesity[1–4], particularly in African Americans and

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men[5–8]. Increased caloric intake underlies the relationship between short sleep duration and weight gain[3,4,9,10], but it remains unclear whether decreased energy expenditure—an important contributor to energy balance and weight management[11,12]—also plays a role.

Sleep loss can change energy expenditure by affecting each of its components: resting metabolic rate (RMR), diet-induced thermogenesis (DIT) and physical activity[13]. Because energy expended during sleep is less than energy expended during wake[14], the increased energy requirement associated with sleep restriction produces negative energy balance and weight loss over time if diet remains constant. To protect against this state, it is hypothesized that the body employs compensatory responses to increase energy intake and conserve energy[14]. If more energy is consumed and conserved than needed (overcompensation), positive energy balance and weight gain occurs.

Several studies support this compensatory hypothesis. Additional energy required for extended wakefulness from acute total sleep deprivation and sleep restriction is 135 kcal/day[15] and ~100 kcal/day[4,16], respectively. Under ad libitum feeding conditions, subjects consume ~500 additional daily calories when sleep restricted[3]; thus, during extended wakefulness adults overcompensate for increases in energy cost with marked increases in energy intake. Some laboratory studies, but not all[15–18], have observed decreased RMR[19–21], DIT[20], and physical activity[22,23] during the day following sleep loss, suggesting a metabolic adaptation to conserve energy in response to the previous day's extended wakefulness.

Substrate utilization, the type of fuel used for metabolism, is assessed using respiratory quotient (RQ; CO_2 produced/O_2 consumed). RQ ranges from 0.63 (0.7: fat metabolism) to 1.3 (1.0: carbohydrate metabolism) with higher RQ values associated with overfeeding and weight gain[12,24,25]. Some studies have found that sleep loss increases RQ[17,21], others have found it has no effect[15,18].

Few studies have assessed the impact of recovery sleep on energy expenditure. Sleeping metabolic rate was lower during recovery sleep from acute total sleep deprivation than during baseline sleep in a small sample of young adults[15]. RMR decreased following 3 weeks of sleep restriction combined with circadian disruption and returned to baseline levels after 9 nights of recovery sleep in young adults[19]. Previous studies examining the effect of sleep loss and recovery sleep on energy expenditure have controlled caloric intake which does not mimic what occurs when adults are sleep restricted outside of the laboratory. Therefore, an experiment examining the impact of sleep restriction and recovery sleep on energy expenditure under ad libitum feeding conditions is critically needed to more closely approximate real world conditions.

No studies to date have examined race and gender differences in the energy expenditure response to sleep restriction. We found that men increased caloric intake to a greater degree than women during sleep restriction, whereas African Americans and Caucasians exhibited comparable daily caloric intake levels[26]. Given that African Americans gained more weight than Caucasians during sleep restriction[3] but did not have greater caloric intake, sleep restriction may affect energy expenditure differently in these racial groups.

The aim of the current study was to investigate the impact of sleep restriction and subsequent recovery sleep on energy expenditure in a sample of healthy African American and Caucasian men and women. We hypothesized resting metabolic rate would be reduced and respiratory quotient would be increased following sleep restriction and these measures would show a compensatory return to baseline levels after a night of recovery sleep. Considering that African Americans gain more weight but exhibit a similar increase in caloric intake compared to Caucasians when sleep restricted, we hypothesized resting metabolic rate would be lower and respiratory quotient would be higher following sleep restriction in African Americans.

Methods

Subjects

Healthy adults (21–50y) were recruited in response to study advertisements. They reported habitual nightly sleep durations between 6.5h–8.5h, bedtimes between 2200h-0000h and wake-times between 0600h–0900h; reports were confirmed using actigraphy. They had no evidence of habitual napping, sleep disturbances, or extreme morningness/eveningness (assessed by questionnaire[27]). Subjects were free of acute/chronic medical/psychological conditions, as established by interviews, clinical history, questionnaires, physical examinations, and blood (including a fasting glucose test) and urine tests. They were nonsmokers and did not participate in shift work, transmeridian travel, or irregular sleep-wake routines in the 60 days prior to the study. Enrolled subjects were monitored at home with actigraphy, sleep-wake diaries, and time-stamped call-ins to assess bedtime and waketime during the week before and after the in-laboratory phase. They were not permitted to use caffeine, alcohol, tobacco, and medications (except oral contraceptives) in the week before the laboratory study, as verified by urine screenings. Sleep disorders were excluded by a night of laboratory polysomnography and oximetry measurements.

The studies were approved by the Institutional Review Board of the University of Pennsylvania and carried out in accordance with approved guidelines. All subjects provided written informed consent before enrollment and were compensated for participation.

Experimental design

Subjects participated in one of two protocols in the Sleep and Chronobiology Laboratory at the Hospital of the University of Pennsylvania and were studied for 14 or 18 consecutive days continuously, in a laboratory protocol with daily clinical checks of vital signs and symptoms by nurses (with a physician on call). Only data collected during the first 8 days of both protocols were used in the current study. A subset of subjects in the current cohort were part of larger cohorts in two previously published studies (n=6 control subjects and n=15 sleep-restricted subjects). Subjects were randomized to either the experimental or control condition (Figure 1). In both protocols, the experimental condition consisted of two baseline nights of 10h/12h time-in-bed (TIB) per night (BL1-2; 2200h-0800h/1000h) followed by five nights of sleep restricted to 4h TIB per night (SR1-5; 0400h–0800h) and one night of 12h TIB recovery sleep (R1; 2200h-1000h). This dose of sleep restriction produces neurobehavioral deficits in healthy adults[28] and is within the range of sleep loss occurring

due to lifestyle factors[29]. The control condition consisted of 10h TIB per night (2200h–0800h) each night.

Subjects were not permitted to leave the laboratory during the protocol. Subjects were ambulatory and were permitted to watch television, read, play video/board games, and perform other sedentary activities between test bouts but they were not allowed to exercise. Subjects wore a wrist actigraph throughout the in-laboratory protocol. On certain protocol days, subjects wore ambulatory electroencephalography and electrocardiography recording equipment for 24h intervals. The light levels were held constant at <50 lux during scheduled wakefulness and <1 lux during sleep periods. Ambient temperature was maintained 22°–24°C. Subjects were monitored by trained staff continuously to ensure adherence. Food/drink was ad libitum throughout the protocol (caffeine was prohibited).

Body composition and metabolic measurements were collected after an overnight fast in the morning following BL1, the fifth night of sleep restriction or control sleep and the night of recovery sleep or sixth night of control sleep (Figure 1). To control for morning-evening effects, measurements occurred during the two hours following waketime (BL1, SR5/CD5, CD6: 0800h–1000h, R1: 1000h–1200h). Prior to the SR5 measurement, subjects were awake for 6h of the 10h fast (2200h-0359h) and could only consume water during this time. Upon awakening, subjects were instructed to use the restroom. Each subject's body composition was assessed and then he/she remained in bed in a supine position for the remainder of the testing period.

Measures

Each subject's body composition was measured using bioelectrical impedance analysis (Omron HBF-510W, 4-Limb device), following voiding. Body weight, fat percentage, and fat free mass (FFM) were collected.

Subjects selected meals/snacks by choosing from menu options, selecting food/drink available in the kitchen within the laboratory suite and making requests to the study staff. Subjects were allowed to consume food/drink at any time during the protocol other than when they were completing neurobehavioral tests or sleeping. All food was weighed and recorded prior to being provided to the subjects. Each day, a detailed description of the items, the amount consumed and intake time were recorded by trained monitors. Additionally, food/drink that was leftover after each meal was weighed and recorded. Intake data were entered into The Food Processor SQL program (ESHA Research, Salem, OR), a validated[30] professional nutrition analysis software that provides components of food/drink intake including calories and macronutrients.

Metabolic rate (kcal/day) was measured using indirect calorimetry with a validated[31,32] ventilated hood system (TrueOne 2400 metabolic cart, Parvo Medics, Sandy, UT, USA). Before each measurement day, a flow meter calibration was conducted and before each use, the metabolic cart was calibrated with reference gas. After achieving steady state (5 minutes, Figure 2), expired gases were collected for 10 minutes and used to calculate metabolic rate. To test the reliability of this measurement, metabolic rate was assessed in a subset of subjects (n=13, 4 women) on two occasions separated by at least 3 months. During both

assessments metabolic rate was measured after a baseline night of sleep in the laboratory and after an overnight fast. The measurement of resting metabolic rate was stable (Assessment 1: 1561.2 kcal/day, Assessment 2: 1571.0 kcal/day; Cronbach's alpha=0.79, Intraclass Correlation Coefficient=0.80) between measurements. Trained technicians monitored subjects and instructed them to keep their eyes open to ensure they remained awake during the tests.

Statistical analysis

Independent sample t-tests compared demographic and sleep variables between sleep-restricted and control subjects and between sleep-restricted gender and race groups. Wilcoxon signed rank tests compared the percent of African Americans, percent of women, as well as pre-study sleep timing between sleep-restricted and control subjects and between sleep-restricted gender and race groups. Mixed model ANOVAs compared changes across protocol days for RMR and RQ between sleep-restricted and control subjects and between sleep-restricted gender and race groups. Planned comparisons were conducted using repeated measures ANOVA and Wilcoxon signed rank tests. All subjects were included in all analyses except for in-laboratory sleep duration; polysomnography data was unavailable for n=10 sleep-restricted subjects. Statistical analyses were completed using IBM SPSS Statistics for Windows, Version 20.0 (Armonk, NY). Effect sizes were calculated using Cohen's d[33].

Results

Subject characteristics

Sleep-restricted subjects (n=36) did not differ from control subjects (n=11) in age, BMI, FFM, or in the percentage of African Americans or women (ps>0.66; Table 1). Sleep duration and timing during the week prior to the in-laboratory study were assessed using wrist actigraphy; there were no differences in pre-study sleep duration, onset, offset or midpoint between sleep-restricted and control subjects (ps>0.13). During the in-laboratory study, sleep duration (assessed using polysomnography) did not differ between sleep-restricted subjects and control subjects during baseline sleep (sleep-restricted subjects: 529.9±55.6 min, control subjects: 494.4±64.3 min, p=0.09). Compared to control subjects, sleep-restricted subjects exhibited a shorter sleep duration during sleep restriction (SR1/CD1: sleep-restricted subjects: 223.5±14.5 min, control subjects: 482.8±84.6 min, p<0.001 and SR5/CD5: sleep-restricted subjects: 229.0±10.9 min, control subjects: 450.4±93.3 min, p<0.001) and a longer sleep duration during recovery sleep (sleep-restricted subjects: 661.5±40.2 min, control subjects: 481.6±71.8 min, p<0.001).

Sleep-restricted African Americans and Caucasians did not differ in age, BMI, FFM or the percentage of women (ps>0.26, Table 1). Sleep-restricted men and women did not differ in BMI or the percentage of African Americans (ps>0.49, Table 1). However, compared to women, men were older (p=0.008) and had greater FFM (p<0.001). In sleep-restricted subjects, there were no gender or race differences in pre-study sleep duration, onset, offset or midpoint (all p>0.15, Table 1) or in sleep duration during the in-laboratory study (ps>0.12).

Weight Gain and Caloric Intake

Sleep-restricted subjects gained 1.31±1.22 kg from baseline day 1 (BL1) to sleep restriction day 5 (SR5; t_{35}=6.42, p<0.001), whereas the change in weight in control subjects (0.62±1.33 kg) from BL1 to control day 5 (CD5) was not significant (p=0.15). The change in weight from BL1 to SR5/CD5 was not significantly different between sleep-restricted and control subjects (covariates: age, BMI, gender and race, p=0.097).

Sleep-restricted subjects and control subjects did not differ in caloric or macronutrient intake during BL1, SR5/CD5 or recovery day 1/control day 6 (R1/CD6; ps>0.05). However, sleep-restricted subjects consumed more calories per day ($F_{1,42}$=5.30, p=0.026), a higher percentage of calories from fat ($F_{1,42}$=5.25, p=0.027) and a lower percentage of calories from protein ($F_{1,42}$=7.71, p=0.008), on average, from baseline day 2 (BL2, when kept awake until 0400h) to sleep restriction day 4 (SR4, the day prior to the metabolic measurement that occurred upon waking on SR5) than control subjects on corresponding days (BL2 to CD4) (covariates: age, gender and race).

Although the difference was not significant, sleep-restricted African Americans gained more weight from BL1 to SR5 than Caucasians (African Americans: 1.57±1.08 kg, Caucasians: 0.89±1.34 kg, covariates: age, BMI, gender, p=0.084). African Americans and Caucasians showed comparable daily caloric intake during the study (BL1-R1; covariates: age and gender, ps>0.24). Sleep-restricted men and women did not significantly differ in weight gain from BL1 to SR5 (men: 1.47±1.13 kg, women: 1.10±1.33 kg, p=0.58; covariates: age, BMI, race); however, men consumed more calories throughout the study than women (BL1: p=0.091, BL2-SR4: p=0.021, SR5: p=0.002, and R1: p=0.002).

Resting metabolic rate

A mixed-model ANOVA, with RMR measurement day as the repeated-measures variable (BL1, SR5/CD5 and R1/CD6), condition (control versus sleep-restriction) as the independent variable, and age, gender, race, FFM, and caloric intake during sleep restriction/corresponding control condition days (BL2-SR4/CD4) as covariates, was conducted. There was a significant day X condition interaction ($F_{2,80}$=6.10, p=0.003); RMR varied across the three measurements in sleep-restricted subjects ($F_{2,70}$=7.79, p=0.001) but not in control subjects (p=0.42). In sleep-restricted subjects, RMR was lower on SR5 compared to BL1 (p=0.032, d_z=0.37, raw change: −41.72 kcal/day, −2.6%), and higher on R1 compared to SR5 (p<0.001, d_z=0.84, raw change: 60.02 kcal/day, +3.8%) but did not differ between BL1 and R1 (p=0.24) (Figure 3A). Comparisons were also significant when using non-parametric tests (BL1 to SR5: p=0.021, SR5 to R1: p<0.001).

In sleep-restricted subjects, a mixed-model ANOVA, with RMR measurement day as the repeated-measures variable (BL1, SR5 and R1), gender and race as independent variables and age, FFM, and BL2-SR4 caloric intake as covariates was conducted. There were no significant day X race (p=0.25) or day X gender interactions (p=0.85). There was a significant main effect of race ($F_{1,29}$=4.50, p=0.043, d=0.60) such that African Americans exhibited a lower RMR than Caucasians during the study (Figure 3B) and no main effect of gender (p=0.38).

Respiratory quotient

A mixed-model ANOVA, with RQ measurement day as the repeated-measures variable (BL1, SR5/CD5 and R1/CD6), condition (control versus sleep-restriction) as the independent variable, and age, gender, race, FFM, and caloric intake during sleep restriction/control condition (BL2-SR4/CD4) as covariates, was conducted. The day X condition interaction was not significant ($p=0.22$). For both groups, RQ was higher on SR5/CD5 (sleep-restricted subjects: $p<0.001$; control subjects: $p=0.019$) and R1/CD6 (sleep-restricted subjects: $p<0.001$; control subjects: $p=0.012$) compared to BL1, but did not differ between SR5/CD5 and R1/CD6 (sleep-restricted subjects: $p=0.57$; control subjects: $p=0.22$; Table 2).

In sleep-restricted subjects, a mixed-model ANOVA, with RQ measurement day as the repeated-measures variable (BL1, SR5 and R1), gender and race as independent variables and age, FFM, and BL2-SR4 caloric intake as covariates was conducted. There was not a significant day X race interaction ($p=0.89$) but there was a significant day X gender interaction ($p=0.034$). RQ varied across the three measurements in men ($F_{2,38}=14.93$, $p<0.001$) but not in women ($p=0.097$). Although both gender groups exhibited an increase in RQ from BL1 to SR5 and R1, the increase was more marked in men (BL1 to SR5: $p<0.001$, BL1 to R1: $p<0.001$) and not significant in women (BL1 to SR5: $p=0.07$, BL1 to R1: $p=0.15$; Table 2).There was a significant main effect of race ($F_{1,29}=7.00$, $p=0.013$, $d=0.70$) such that African Americans exhibited a higher RQ than Caucasians during the study (Table 2) but no main effect of gender ($p=0.88$).

Discussion

Resting metabolic rate (RMR)—the largest (60–70%) component of energy expenditure—was significantly reduced after sleep restriction and rebounded to baseline levels after one night of recovery sleep. Our finding is consistent with a study reporting reductions in RMR following three weeks of sleep restriction (5.6h sleep/24h) and forced desynchrony (28h “days”), and a return in RMR to baseline levels following 9 nights of recovery sleep with circadian re-entrainment (10h sleep/24h) under controlled caloric intake conditions[19]. Our results demonstrate that sleep restriction, without significant circadian disruption and under ad libitum feeding conditions, decreased RMR. Furthermore, RMR returned to baseline levels after only one night of recovery sleep, consistent with the time course of the caloric intake response[3].

Other studies have found no effect of sleep restriction on RMR[9,10] when assessed the day following sleep loss. Discrepant findings may be due to differences in study design. For example, studies allowed subjects to leave the laboratory to walk outside, consume caffeine daily during the study, or use a gym at their leisure, thereby potentially confounding the assessment of RMR[9,10].

The compensatory hypothesis posits that sleep restriction produces weight gain due to the overcompensation of neuroendocrine, metabolic and behavioral responses aimed to increase energy intake and conserve energy in order to counteract the additional energy requirement associated with extended wakefulness[14]. Although the observed change in RMR was small (~50 kcal/day), our study provides further support for this hypothesis. Previous research has

found that under non-sleep restriction conditions, short-term (2–7 days) overfeeding leads to weight gain (+0.5–1.8 kg) and increased RMR (+1.1–11.7%)[34,35]. In the current study, food/drink was ad libitum. We estimated the amount of daily calories each subject would need to consume in order to maintain weight during the study (each subject's measured RMR multiplied by 1.4, to account for energy needed for sedentary activity levels in the laboratory environment)[38]. Using this estimation, subjects consumed 35% more calories than needed during sleep restriction (BL2-SR4). For example, if it was estimated that a subject needed to consume 2000 calories to maintain weight, he/she consumed 700 additional calories during sleep restriction. Despite this recent history of overconsumption, subjects exhibited a *decrease* in RMR measured in the morning after the fifth night of sleep restriction. Thus, sleep restriction produces metabolic responses to substantially increase caloric intake and to conserve energy which is conducive to weight gain over time. By contrast, recovery sleep reverses these responses.

It remains unknown which mechanisms may underlie the effect of sleep loss on changes in resting metabolic rate. In contrast to humans, rodents exhibit marked increases in energy expenditure in response to sleep deprivation which prevents weight gain despite increased caloric intake[37]. More research in humans is needed to better understand what mechanisms may underlie the decrease in resting metabolic rate that we observed the morning following sleep restriction; decreases in energy used for immune, endocrine and sympathetic nervous system functions are areas that should be examined.

Consistent with previous studies[17,21], sleep restriction increased RQ, which has been associated with weight gain[12,24]. However, this increase was also observed in control subjects, suggesting that factors other than sleep restriction, such as overeating[25] and living in a sedentary laboratory environment, contributed to the RQ effect.

Relative to Caucasians, African Americans exhibited a lower RMR and a higher RQ regardless of sleep duration but had comparable caloric intake during the study. Although there is debate whether or not race differences in RMR or RQ are associated with weight gain/obesity[38], it is possible that these differences led to greater weight gain during our in-laboratory study. African Americans—who consistently report shorter sleep durations[39] and have a higher prevalence of overweight/obesity (particularly women) in population studies[40]—may need to compensate for a lower RMR by reducing caloric intake to a greater degree than Caucasians or by increasing physical activity in order to prevent weight gain.

The current study had several limitations. Because subjects needed to remain in the laboratory for the duration of the protocol, body composition was assessed using bioelectrical impedance analysis which has limited sensitivity; therefore, we were unable to examine changes in body composition after sleep loss. Due to time restrictions related to our protocol, we used a metabolic cart with 15-minute test duration to assess metabolic rate. Whole-room indirect calorimetry systems with continuous recordings for multiple hours provide a more complete measure of changes in energy expenditure[13]. Physical activity was not directly assessed in the current study and was kept sedentary within the confines of our laboratory; future studies are needed to assess if there are race or gender differences in activity levels. Finally, in our subjects we did not systematically monitor the menstrual cycle

and allowed the use of oral contraceptives. Only n=2 women were taking oral contraceptives at the time of the study and all women except for two reported experiencing regular menstrual cycles (one woman was not actively menstruating due to oral contraceptive use and one woman had a partial hysterectomy).

Conclusion

Sleep restriction decreased morning resting metabolic rate in healthy adults, suggesting that sleep loss leads to metabolic changes aimed at conserving energy. A lower resting metabolic rate—combined with the increased caloric intake that occurs during sleep restriction—places adults who are habitual short sleepers at heightened risk for weight gain and obesity.

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What is already known about this subject?

- Habitual short sleep duration is consistently associated with weight gain and increased risk for obesity
- Sleep restriction leads to weight gain and increased caloric intake

What does your study add?

- Resting metabolic rate is decreased after sleep restriction under ad libitum feeding conditions in healthy diverse men and women

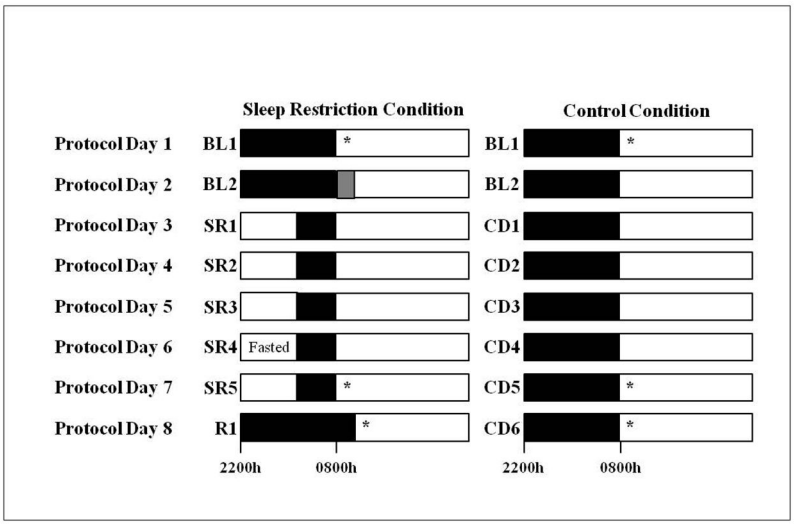

Figure 1.

Protocol schedule

The sleep restriction condition consisted of two baseline nights of 10h or 12h TIB per night (2200h–0800h/1000h) followed by five nights of sleep restricted to 4h TIB per night (0400h–0800h) and one night of 12h TIB recovery sleep (2200h-1000h). The control condition consisted of 8 consecutive nights of 10h TIB per night (2200h-0800h). Food/drink intake was ad libitum; however, prior to the measurement on SR5, sleep-restricted subjects were awake for 6h of the 10h overnight fast (2200h-0359h) and were only allowed to consume water during this time. *****Body composition and metabolic measurements were collected during the morning after the first night of baseline sleep, after the fifth night of sleep restriction and after the night of recovery sleep, or after corresponding nights in the control condition. To control for morning-evening effects, measurements occurred during the two hours following waketime (between 0800h–1000h on BL1, SR5/CD5 and CD6 and between 1000h–1200h on R1). Upon awakening, subjects used the restroom. Each subject's body composition was assessed and then subjects remained in bed in a supine position for the remainder of the testing period. After the body composition measurement, each subject's metabolic rate was assessed. Black bars=sleep periods; white bars=wake periods; gray bar=additional 2h sleep period for subjects participating in one of the protocols.

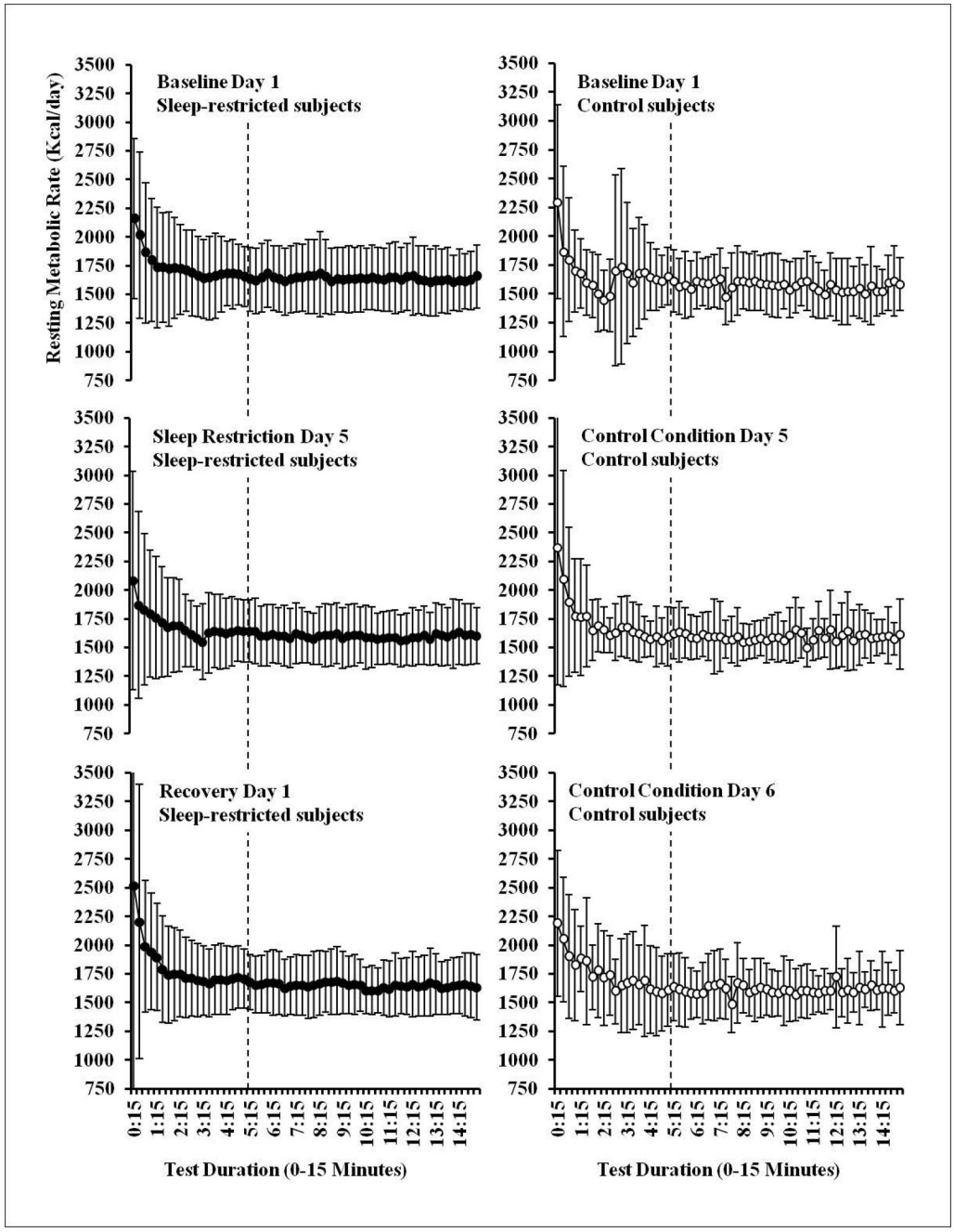

Figure 2.

Metabolic measurements during each protocol day

Metabolic rate (kcal/day) was measured using indirect calorimetry with a validated ventilated hood system (TrueOne 2400 metabolic cart, Parvo Medics, Sandy, UT, USA). Before each measurement day, a flow meter calibration was conducted and before each use, the metabolic cart was calibrated with reference gas. After achieving steady state (5 minutes, denoted with the dotted line), expired gases were collected for 10 minutes and used to calculate metabolic rate. Trained technicians monitored subjects and instructed them to keep their eyes open to ensure they remained awake during the tests. Data shown as mean ± SD.

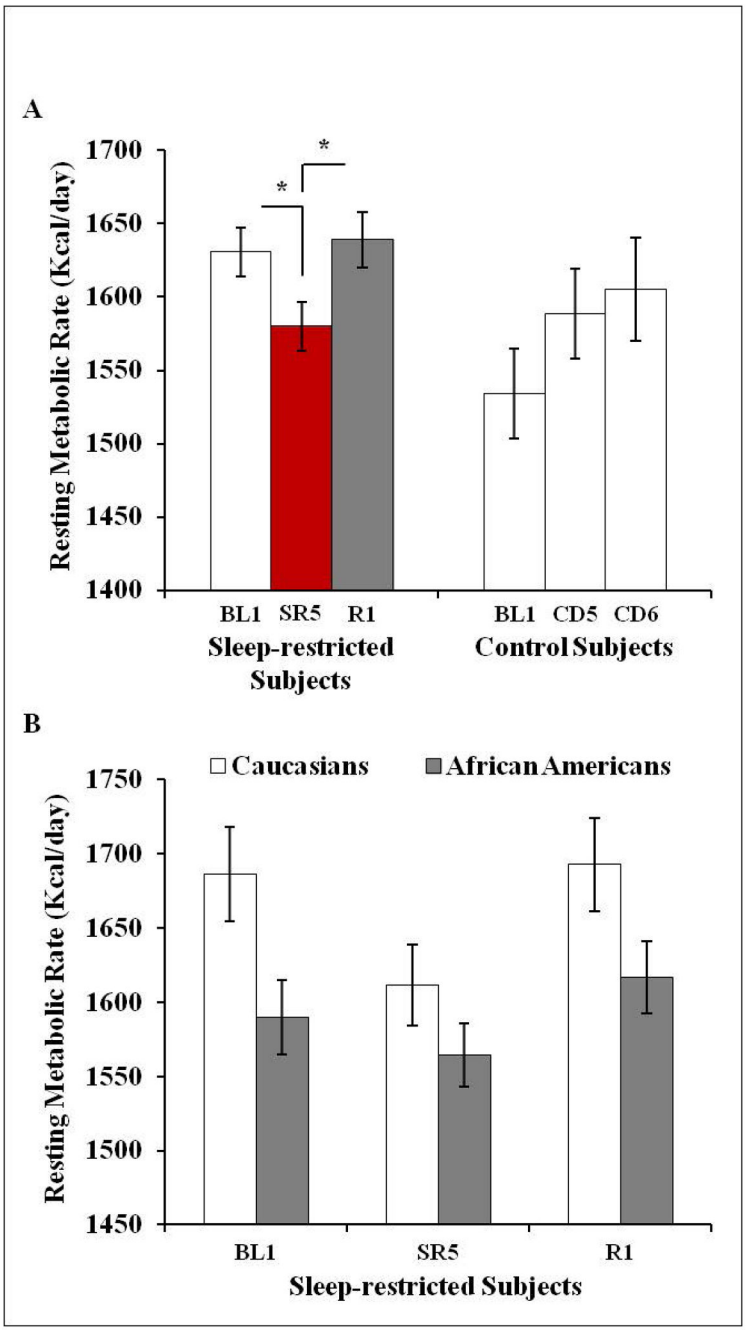

Figure 3.

Resting metabolic rate across protocol days

(A) A mixed-model ANOVA, with RMR measurement day as the repeated-measures variable (BL1, SR5/CD5 and R1/CD6), condition (control versus sleep restriction) as the independent variable and age, gender, race, FFM, and caloric intake during sleep restriction/ corresponding control condition days (BL2-SR4/CD4) as a covariates, revealed that there was a significant day X condition interaction (p=0.003). RMR varied across the three measurements in sleep-restricted subjects (p=0.001) but not in control subjects. In sleep-

restricted subjects, RMR was lower on SR5 compared to BL1 ($p=0.032$, raw change: −41.72 kcal/day, −2.6%), and higher on R1 compared to SR5 ($p<0.001$, raw change: 60.02 kcal/day, +3.8%) but did not differ between BL1 and R1. **(B)** In sleep-restricted subjects, a mixed-model ANOVA, with RMR measurement day as the repeated-measures variable (BL1, SR5 and R1), gender and race as independent variables and age, FFM, and BL2-SR4 caloric intake as covariates, revealed no significant day X race or day X gender interactions. There was a significant main effect of race ($p=0.043$) such that African Americans exhibited a lower RMR than Caucasians during the study; however, there was no main effect of gender ($p=0.38$). Data shown as mean ± SEM, *$p<0.05$.

Table 1

Mean (SD) Subject Demographic Characteristics

	Age y	Weight kg	BMI kg/m^2	Fat-Free Mass kg	% Body Fat	Pre-Study Sleep Duration	Pre-Study Sleep Midpoint
Control Subjects (n=11)	34.0 (9.4)	73.4(11.5)	24.3 (3.4)	52.6 (9.8)	28.3(10.5)	7h 57m(30.6 m)	03:53(33.0 m)
Sleep-Restricted Subjects (n=36)	33.8 (9.2)	74.0(12.3)	24.8 (3.1)	52.2 (11.5)	30.1(9.4)	7h 55m(26.4 m)	03:41(43.2 m)
Men (n=20)	37.4(9.5)	79.4(12.4)	24.7(3.1)	60.8(7.8)	23.7(6.1)	7h 53m(21.6 m)	03:40(42.6 m)
Women (n=16)	29.4 (6.7)	67.2(8.4)	25.0(3.3)	41.6(3.6)	38.1(6.2)	7h 57m(32.4 m)	03:44(45.0 m)
Caucasians (n=14)	34.1(7.1)	75.8(12.4)	25.0(3.1)	55.0(12.0)	28.1(8.8)	7h 53m(24.0 m)	03:50(37.8 m)
African Americans (n=22)	33.7(10.5)	72.9(12.4)	24.7(3.2)	50.5(11.1)	31.4(9.8)	7h 56m(28.8 m)	03:35(46.8 m)

Body composition was assessed using bioelectrical impedance analysis.

Pre-study sleep duration and timing were assessed using wrist actigraphy.

Table 2

Mean (SD) RQ across Protocol Days

	BL1	SR5/CD5	R1	Change in RQ (SR5 – BL1)
Control Subjects (n=11)	0.86 (0.04)	0.91 (0.07)*	0.93 (0.09)*	0.05 (0.06)
Sleep-Restricted Subjects (n=36)	0.85 (0.05)	0.89 (0.06)*	0.90 (0.07)*	0.05 (0.06)
Men (n=20)	0.84 (0.05)	0.91 (0.06)*	0.92 (0.07)*	0.06 (0.05)
Women (n=16)	0.85 (0.04)	0.88 (0.05)	0.87 (0.06)	0.03 (0.06)
Caucasians (n=14)	0.83 (0.04)	0.87 (0.06)*	0.88 (0.06)*	0.04 (0.06)
African Americans (n=22)	0.86 (0.05)	0.91 (0.05)*	0.91 (0.07)*	0.05 (0.06)

* $p<0.05$, compared to BL1