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Sleep duration during the school week is associated with C-reactive protein Risk Groups in healthy adolescents

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Abstract

Background—The prevalence of short sleep duration in adolescence and the relevance of early risk factors to cardiovascular disease in adulthood suggest that adolescence is an opportune time to evaluate links between sleep duration and cardiovascular disease risk. We examined associations among actigraphy-assessed sleep duration and sleep debt with elevated C-Reactive Protein (CRP), a known risk factor for cardiovascular disease.

Methods—Participants were 244 (137 Blacks, 116 males) healthy high school students, each of whom wore wrist actigraphs for one week and provided a fasting blood draw. CRP was examined as both a continuous and categorical outcome, with CRP > 3 mg/L identifying a High Risk Group.

Results—Sleep duration and sleep debt were significantly associated with CRP High Risk Group in covariate-adjusted analyses. Shorter sleep duration on school nights was associated with a greater likelihood of being in the High Risk CRP Group. Likelihood of being in the High Risk CRP Group was doubled in students who obtained an average of two or more hours of “catch up” sleep on weekend nights.

Conclusions—Reduced weekday sleep duration and sleep debt were both associated with CRP Risk Group in adolescence. That these relationships may be observed prior to the onset of clinical disease suggests that adolescence may provide an opportune period for disease prevention.

Keywords

Sleep duration; Sleep debt; C-reactive protein; adolescence; sex; race

Introduction

Short sleep duration, which is highly prevalent in industrialized nations, is a significant risk factor for cardiovascular disease (CVD) [1;2]. Chronic low-grade inflammation, including elevated circulating levels of C-reactive protein (CRP), represents one pathway through

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which short sleep duration may influence both the pathogenesis and clinical course of cardiovascular disease [3]. C-reactive protein contributes to multiple aspects of atherogenesis and plaque vulnerability across the lifespan and elevated CRP has been prospectively associated with cardiovascular disease [4;5]. For instance, individuals with CRP values > 3 mg/L are at increased risk for cardiovascular events [6].

Several epidemiological studies have reported that short sleep duration is associated with increased circulating levels of plasma CRP in adults, although discrepant findings have been reported by some [7–12]. Important to questions of causality, a number of studies have reported that CRP levels increase in response to experimental sleep restriction and sleep deprivation in healthy young adults, although some have reported null effects [13–16]. Discrepant findings in the published literature may be related to methodological differences across studies and/or differences in important effect modifiers such as sex, race, medication use, co-morbidities including undiagnosed cardiovascular disease, and survivor bias.

Several factors suggest that adolescence is an opportune time to evaluate links between short sleep duration and cardiovascular disease risk, including markers of inflammation. First, sleep restriction is endemic in high school students due, in part, to a constellation of biological and social factors that favor later bedtimes in spite of societally-mandated early school start times [17;18]. Numerous studies, including our own, have reported that most high school students do not obtain the recommended 8 to 9 hours of sleep suggested by the Centers for Disease Control (CDC) [19–21]. For instance, nearly 40% of high school students who completed the 2007 CDC National Youth Behavior Survey reported sleeping 6 hours or less on school nights [22]. Although high school students sleep an average of two hours longer on weekend nights, most studies suggest their weekly sleep duration averages are still generally below 8 hours [19–21]. Second, longitudinal studies have established that risk factors for cardiovascular disease, including chronic low-grade inflammation, can be identified in adolescents, decades before the onset of clinical disease [23–27]. Among identified early risk factors, elevated CRP in adolescents has been linked to the structural and functional vascular abnormalities as well as the metabolic syndrome [28–30]. Finally, adolescence allows examination of the association between short sleep duration and elevated CRP in the relative absence of underlying disease and without the confounding of survivor bias present in older samples.

Modest inverse correlations between sleep duration and circulating CRP levels have been observed in high school students in the United States and Spain [31;32]. Both studies averaged sleep duration values across the week, despite evidence that many high school students sleep an average of 2 hours longer on weekend compared to week nights [33]. Although neither study evaluated the influence of race, others have reported increased CRP in Black short sleeper adults [34;35]. Results for sex as an effect modifier in adult samples have been mixed [8;32;34;36;37].

The present study evaluated relationships among actigraphy-assessed sleep duration and fasting high-sensitivity CRP (hsCRP) in White and Black high school students, about half of whom were female. These data extend the published literature on sleep duration and CRP in adolescents by examining weekday and weekend sleep duration independently and by

evaluating the possible influence of race and sex on these associations. These issues are critical to both risk stratification and development of evidence-based interventions to reduce cardiovascular risk early in the development of this prevalent and costly disease.

Methods

Participants

Participants were 250 adolescents between the ages of 14 and 19 enrolled in a public high school that serves a racially diverse (42% Black, 56% White, 2% other), lower to middle socioeconomic status community near Pittsburgh, PA. Recruitment and study procedures took place between November 2008 and May 2011, except for summers and school vacations. The study was described to potential participants during regularly scheduled high school physical/health education classes as concerning the relationships among stress, sleep, and cardiovascular risk factors. Students interested in participation contacted the research team to learn more about the study and determine eligibility. Approval of the research project was obtained from the superintendent of the school district, the principal of the high school, and the University of Pittsburgh Institutional Review Board. Participants or legal guardians of students under the age of 18 provided written informed consent prior to commencing the study. A total of 16 students expressed interest in the study but were deemed ineligible due to one or more of the following exclusions: medication use for emotional or psychological disorders, use of diabetes or blood pressure medication, and use of any medication known to affect the cardiovascular system or sleep. Symptoms of sleep apnea including snoring, pauses in breathing, and snorting and gasping during sleep were assessed by parent report. Only one student's parent reported modest symptoms in their child. The parent was subsequently interviewed by a pediatrician and sleep medicine expert, and it was determined that the student did not have sufficient sleep problems to warrant exclusion. The present report includes 244 participants who had complete data available for sleep assessments and CRP.

Measures

Sleep—Sleep measures were derived from rest-activity patterns recorded by wrist actigraphs (Actiwatch-16, Philips Respironics, Bend, OR) worn continuously by participants for a 7-day period during the academic school year. Data were not collected during the summer and over school vacations. Wrist actigraphy has been validated against polysomnography for measuring sleep duration in adolescents [38]. Nocturnal sleep intervals were identified as previously described [39]. Sleep duration was operationalized as the total minutes of sleep between initial sleep onset and final sleep offset, excluding periods of actigraphy-assessed wakefulness during the night. Average sleep duration was calculated separately for week nights (Sun – Thurs nights) and weekend nights (Fri-Sat nights). For comparison to published papers, average weekly sleep duration was calculated over the week-long study period. A fourth variable, sleep debt, was used to evaluate whether the need for "catch up" sleep on weekend nights was associated with CRP. We first subtracted average weeknight sleep duration from average weekend sleep duration. Sleep debt was then labeled as "present" in participants who slept at least 2 hours longer on weekend compared to week nights.[40;41]

C-Reactive Protein—Fasting blood samples were obtained on school days that coincided with study assessments. Blood draws were rescheduled to a later date if participants endorsed any sign of illness (e.g., upper respiratory infection, taking antibiotics, etc.) within the past three days. Once collected, serum was separated by refrigerated centrifuge, aliquoted, and stored at −80 C until assay in the Heinz Lipid Laboratory at the Graduate School of Public Health at the University of Pittsburgh. Each high sensitivity assay (hsCRP) run included duplicate samples, standards and control sera. CRP was measured turbidimetrically by measuring increased absorbance when the CRP in the sample reacts with anti-CRP antibodies. The intra-assay coefficient of variation was 5.5% and inter-assay coefficient of variation was 3.0%. We evaluated hsCRP as both a continuous and categorical outcome, given the prognostic value of evaluating clinically-significant cut-off scores. The Low to Moderate Risk Group included participants with CRP values ≤ 3 mg/L (n=211; 86.5%) and the High Risk Group included participants with CRP values > 3 mg/L (n=33; 13.5%).

Covariates—Age, sex, and race/ethnicity were determined by participant self-report. Height was measured using a stadiometer, and weight was measured on a Tanita digital scale to the 1/10 of a pound. Body mass index (BMI) was calculated using the NHLBI on-line calculator: www.nhlbi.nih.gov/guidelines/obesity/BMI/bmicalc.htm. Parental education was used to characterize household socioeconomic status. Greatest educational attainment by the participant's mother or father in the household was dichotomized as a high school degree/GED (n=113) or less (n=29) versus college-educated, including some college (n=70) or a college degree (n=32). Daytime sleepiness., which may be viewed as an indicator for likely sleep apnea and/or a mechanism to cope with short nighttime sleep, was measured by a ten-item scale focused on situations in which they may have struggled to stay awake or fallen asleep in the past 2 weeks, e.g. attending a performance, in class at school, driving a car. Each item was rated on a four-point scale from no, struggled to stay awake, fallen asleep, or both struggled to stay awake and fallen asleep, and were totaled. Average daytime nap duration was calculated from daily self-reported sleep diaries and recorded as average minutes per day (weekday and weekend). Finally, season was defined as fall (September through November), winter (December through February) and spring (March through June).

Statistical Analysis

Descriptive statistics were used to examine sample characteristics, check for outliers, and evaluate normality. A natural log transformation was used to normalize hsCRP levels. We used Chi^2 tests and analysis of variance (ANOVA) to characterize categorical and continuous sample characteristics as a function of sex and race. In the primary analyses, multivariate linear regression and binary logistic regression were used to evaluate associations among sleep duration and hsCRP levels and CRP Risk Group, respectively. Covariates included in all analyses were sex, race, BMI and parental education. Age, daytime sleepiness, naps and season were not included as covariates as they were unrelated to CRP outcomes in the present sample ($p>.20$). Actigraphy-assessed weekday and weekend sleep duration were included in the same model as these variables were uncorrelated ($r=0.10$, $p>.05$). Separate models were used for average weekly sleep duration and for sleep debt, which was derived from and collinear with weekday ($r=-0.41$, $p<.001$) and weekend

sleep duration (r=−0.61, p<.001). In secondary analyses we examined the extent to which associations among sleep duration and CRP outcomes differed as a function of sex or race, based on the literature in adults [8;34;35]. We evaluated interactions using multivariate models with centering of sleep variables and stratified multivariate analyses explored associations among sleep duration and CRP outcomes by sex and race. Fit statistics (Hosmer & Lemeshow Test for logistic regression and F Test for linear regression) suggested good fit for all models with the exception of interactions and exploratory stratified analyses of effect modifiers. Finally, sensitivity analyses excluding participants with CRP levels > 10 mg/L (n=9) were used to examine possible confounding by undetected acute illness at the time of the blood draw. Results in the reduced sample were unchanged from those in the full sample (sensitivity analyses not shown).

Results

As shown in Table 1, 52.5% of the sample was female, 56.1% were Black, and average BMI was above the 75th percentile for national age and sex norms. Wrist actigraphy revealed that participants obtained just under 6 hours of sleep on school nights and about 7 ½ hours of sleep on the weekends. Actigraphy also indicated that over 1/3 of the sample experienced sleep debt, defined as sleeping at least 2 hours longer on weekend relative to week nights. On average, participants napped over 30 minutes per day during the week and over 15 minutes per day on weekends. As previously reported [20], sleep duration was an average of 30 minutes shorter in males compared to females on school nights (p<.05), and sleep duration was nearly 40 minutes longer on weeknights and weekend nights in Whites compared to Blacks (p<.001 and p<.05, respectively). Other sample characteristics including age, hsCRP levels, BMI, sleep debt and daytime sleepiness were similar for males and females as well as for Whites and Blacks.

Participants in the High Risk CRP Group had significantly higher BMIs (88.09% ± 18.9) compared to participants in the Low to Moderate Risk CRP Group (77.16% ± 22.86; p<.01). In univariate linear analyses, BMI accounted for 21% of the variance in hsCRP levels; higher BMIs were associated with higher hsCRP levels (p<.001). There was a trend for higher parental education in the Low to Moderate Risk CRP Group (44.1% with some college or college degree) compared to 27.3% with education beyond high school in the High Risk CRP Group (Chi^2=3.31, p<.07). Age, daytime sleepiness, naps and season in which the study was conducted did not differ as a function of CRP Risk Group.

Sleep duration and sleep debt were unrelated to hsCRP levels (see Table 2). In contrast, both weekday sleep duration and sleep debt were significantly associated with CRP Risk Group. Multivariate logistic regression revealed that shorter weekday sleep duration was a significant correlate of CRP High Risk Group, independent of weekend sleep duration and other model covariates including sex, race, parental education, and BMI. As shown in Figure 1, students who slept longer on school nights were less likely to be in the High Risk CRP Group (Odds Ratio (OR) = 0.62, 95% Confidence Interval (CI) = 0.39 – 0.98. In contrast, weekend sleep duration was unrelated to CRP Risk Group (OR = 0.96, 95% CI = 0.69 – 1.34). Average sleep duration over the 7-day study period was unrelated to CRP Risk Group. The sleep debt model revealed that students who slept at least 2 hours longer on weekend

compared to weeknights were nearly two and a half times more likely to be in the High Risk CRP Group compared to those without sleep debt (OR = 2.33, 95% CI = 1.01 – 4.89).

Although race-by-sleep duration and gender-by-sleep duration interactions were not significant, exploratory analyses suggest possible differences. In exploratory analyses stratified by race, the association between short weekday sleep duration and CRP Risk Group was observed in Whites (OR = 0.51, 95% CI 0.28 – 0.93) but not in Blacks (OR = 0.83, 95% CI = 0.37 – 1.86). Exploratory analyses stratified by sex revealed that short weekday sleep duration was associated with the High Risk CRP Group in females (OR = 0.43, 95% CI = 0.22 – 0.84) but not in males (OR = 0.88, 95% CI = 0.39 – 1.98).

Discussion

The prevalence of short sleep duration in adolescence and the relevance of early risk factors to cardiovascular disease in adulthood suggest that adolescence is an opportune time to evaluate links between short sleep duration and cardiovascular disease risk. The limited data in adolescents is striking, given that cardiovascular disease develops over years and decades, starting during youth [23–27]. To our knowledge, this study is the first to concurrently examine associations among both weeknight and weekend sleep duration with CRP in adolescents. Students with shorter actigraphy-assessed sleep durations on school nights were more likely to be in the High Risk CRP Group, compared to their classmates who obtained more sleep on school nights. Critically, this association was independent of weekend sleep duration and other factors known to influence circulating CRP levels in healthy adolescents. While weekend sleep duration was not uniquely associated with CRP, likelihood of being in the High Risk CRP Group was doubled in students who obtained an average of two or more hours of “catch up” sleep on weekend compared to weekend nights. We know of no study that has evaluated sleep debt in relation to CRP, although sleep debt in adults has been associated with other adverse cardiometabolic outcomes including increased inflammation (IL-1beta, IL-6) and poor glycemic control [42;43].

Two previous studies in adolescents reported modest inverse linear associations between circulating hsCRP levels and sleep duration averaged across a 7-day period [31;32]. In contrast, we observed a threshold effect in our sample. These divergent results may be a function of sample characteristics. For instance, average sleep duration in our sample across the 7-day study period was 1.4 to 2.2 hours shorter than values observed by Larkin and colleagues and Martinez-Gomez and colleagues, respectively. Given that only three participants in our sample obtained 8 or more hours of sleep on school nights, the sleep duration range may have been too restricted to observe a linear trend between sleep duration and CRP. In addition, our sample was more racially diverse than the previous studies in adolescents. Associations among weekday sleep duration and sleep debt with CRP Risk Group were independent of BMI despite its strong link with study outcomes and the striking number of overweight/obese adolescents in the sample. The average sample BMI was above the 75th age- and sex-normed percentile. Taken as a whole, these studies suggest that sleep duration is a significant correlate of high hsCRP in healthy adolescents, although the nature of this association may differ as a function of sample characteristics.

Although formal tests for interaction effects were not significant, stratified analyses revealed different associations among sleep duration and CRP Risk Group for males and females and for Whites and Blacks. The probability of being in the High Risk CRP Group was increased in females and Whites with shorter weekday sleep durations while sleep duration and CRP Risk Group were unrelated in males and Blacks. Pirkola and colleagues reported similar results in sex-stratified analyses of 16-year-old students in Finland [37]. Girls who reported sleeping less than 8 hours per day on average were more likely to be in a combined High Risk CRP and Low Leukocyte Count group than those who reported habitual sleep durations of 8 or more hours (OR=1.39, 95% CI 1.02 – 1.90); sleep duration was unrelated to hsCRP levels in males. Results have been mixed in adults, where curvilinear associations among sleep duration and adverse health outcomes are more common due, in part, to increased medical and psychiatric morbidity in long-sleeping adults (e.g., sleep durations > 9 h) [44]. One study observed significant associations between short sleep duration (<7 h) and hsCRP levels in adult women only [45], while another found significant associations only in adult men [34]. Although none of the previous studies in adolescents evaluated race as a modifier of the sleep duration-CRP relationship, two studies in adults reported significant associations among short sleep duration and hsCRP levelsin Blacks but not in Whites [46;47]. It is difficult to determine whether sex- and race-specific effects in the present study reflect true differences or are a result of sample characteristics or chance and certainly the absence of significant interaction effects suggest we were not powered to fully examine these possible effect modifiers. Although our sample was roughly comprised of equal numbers of females and males and Blacks and Whites, sleep durations profiles were markedly different – actigraphy-assessed sleep durations of more than 7 hours were observed in 15% of females compared to less than 5% of males and in 30% of Whites compared to 14% of Blacks. Although these results should be interpreted cautiously, the collection of additional data in future studies appears warranted to determine whether risk stratification schemes for sleep duration and cardiovascular risk in adolescents should account for the individuals' sex and race.

Several plausible biological, behavioral, and psychological pathways may lead to increased systemic inflammation, including CRP, in response to insufficient sleep. Converging evidence suggests that sleep restriction leads to weight gain via alterations in systemic and cellular metabolism as well as behaviors associated with craving and food choices [48–50]. Release of IL-6 from adipocytes, in turn, stimulates CRP release by the liver. Sleep restriction has also been associated with increased autonomic arousal which, similarly, increases circulating CRP levels [51–54]. Psychological pathways including increased interpersonal conflict, psychological stress, and emotional lability are, similarly, plausible pathways through which sleep duration may influence inflammation. Short sleep duration and experimental sleep deprivation have been associated with decreased emotion regulation and increased reactivity to stress [55–57]. In a separate literature, experimental manipulation of stress has been associated with increased circulating CRP levels [58]. While these "pathway" studies have been conducted in adults, these mechanisms are equally plausible for adolescent samples. Certainly, additional research is needed to probe the biological, behavioral and psychological pathways through which short sleep duration may influence

inflammation, including CRP in order to identify plausible targets for intervention in adolescents.

Interpretation of the present data should take into consideration the following limitations. First, the cross-sectional nature of our data precludes causal attributions. Although several studies have reported increased circulating CRP levels in response to experimental sleep restriction and deprivation in healthy adults, results have not always been consistent [13–16]. Moreover, marked differences in sleep profiles in adolescents and adults suggest that associations among sleep duration and inflammation may differ as a function of age. Second, we did not assess sleep apnea, which has been associated with inflammation, including increased CRP [59;60]. Adjustment for BMI in our statistical analyses may have attenuated the influence of sleep apnea in our models as overweight/obesity is a leading risk factor for sleep apnea in adolescents [61]. Although we evaluated daytime sleepiness as a proxy for sleep apnea, it was unrelated to CRP in our sample. Third, data cannot be generalized to adolescents with different sleep duration and inflammation profiles as a function of race/ethnicity or existing medical/psychiatric co-morbidities. Notable study strengths include behavioral assessment of sleep duration via wrist actigraphy, examination of the independent associations among weeknight and weekend sleep duration with CRP, evaluation of sleep debt, statistical adjustment for possible confounders including BMI, and the absence of health confounds such as diabetes, hypertension and kidney disease. Fourth, although formal tests of interactions were not significant, analyses stratified by race suggest that race and sex may modify associations among weekday sleep duration and CRP risk. Larger studies will be needed to more fully evaluate these relationships.

These data extend the limited literature on sleep duration and CRP in adolescents by evaluating weekday and weekend nights separately. Sleep duration differences across weekday and weekend nights are especially relevant in adolescents who operate under conditions of chronic sleep restriction during the school year due to the constraints imposed by biological and social drives for later sleep start times despite societally-mandated early school start times [17;18]. Prospective studies are needed to evaluate the impact of sleep restriction and sleep debt in otherwise healthy adolescents to chronic inflammation and to the pathogenesis and clinical course of cardiovascular disease. Identification of important effect modifiers and the modifiable biological, behavioral and psychological pathways through which short sleep duration in adolescence influences inflammation and its downstream consequences to cardiovascular risk will be critical to risk stratification and intervention. That these relationships may be observed prior to the onset of clinical or even subclinical disease suggests that adolescence may provide opportunities for disease prevention.

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Highlights

- Short sleep duration on school nights was associated with CRP values > 3 mg/L.
- Sleep debt was also associated CRP values > 3 mg/L.
- Associations among sleep and CRP in adolescence may differ as by sex and race.
- Findings were independent of known risk factors for elevated CRP.

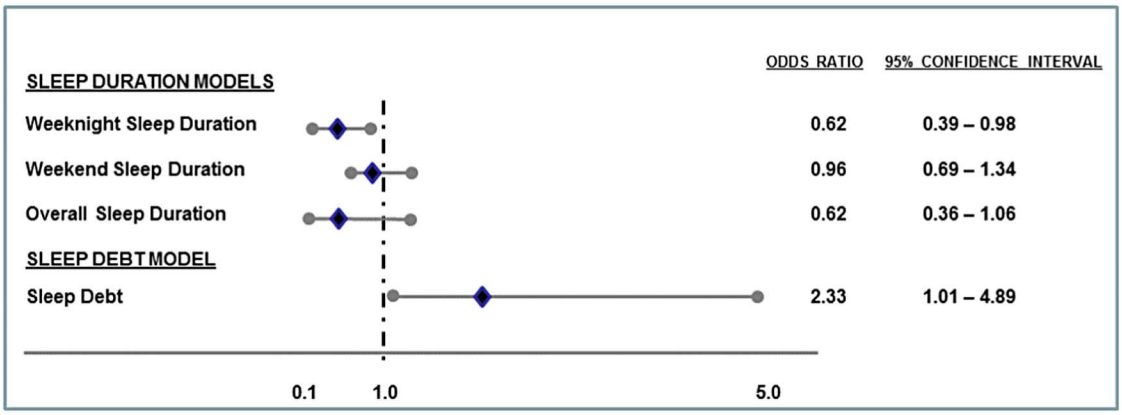

Figure 1.

Associations among sleep duration and sleep debt with CRP Risk Groups. Diamonds depict adjusted odds ratios for the High Risk CRP group and circles represent lower and upper confidence limits. Odds ratios are adjusted for sex, race, highest parental education and BMI.

Table 1

Sample characteristics

		Sex		Race	
	All (n=244)	**Female (n=128)**	**Male (n=116)**	**White (n=107)**	**Black (n=137)**
Age, years					
mean (SD)	15.71 (1.31)	15.69 (1.29)	15.74 (1.33)	15.65 (1.30)	15.76 (1.31)
range	14 – 19	14 – 19	14 – 19	14 – 19	14 – 19
BMI					
Mean (SD)	26.08 (5.86)	26.11 (6.12)	26.05 (5.60)	26.05 (6.34)	26.11 (5.48)
range	16.44–46.89	16.44–43.48	16.67–46.89	16.44–46.89	17.75–43.48
BMI Percentile (sex & age)					
mean (SD)	78.64 (22.64)	77.41 (23.41)	79.98 (21.79)	76.09 (25.93)	80.62 (19.57)
range	27 – 100	3 – 100	6 – 100	3 – 100	10 – 100
Parental Education[1]					
No. (%) ≤ high school	142 (58,2%)	82 (64.1%)	6 (51.7%)	73 (68.2%)	69 (50.4%)
No. (%) ≥ some college	102 (41.2%)	46 (35.9%)	56 (48.3%)	34 (31.8%)	68 (49.6%)
hsCRP Levels (mg/L)[2]					
Mean (SD)	1.71 (3.16)	1.98 (3.63)	1.42 (2.53)	1.87 (3.21)	1.59 (3.13)
Range	0.03 – 23.10	0.03 – 23.10	0.05 – 15.70	0.03 – 20.20	0.03 – 23.10
Median	0.64	0.65	0.58	0.75	0.50
Interquartile Range	0.24 – 1.67	0.25 – 2.26	0.23 – 1.47	0.32 – 1.64	0.22 – 1.72
High Risk CRP Group					
No. (%) No	211 (86.5%)	107 (83.6%)	104 (89.7%)	88 (82.2%)	123 (89.8%)
No. (%) Yes	33 (13.5%)	21 (16.4%)	12 (10.3%)	19 (17.8%)	14 (10.2%)
Sleep Duration (hours)	5.94 (.89)	6.07 (.91)	5.80 (.84)		
Mean (SD) Weekday[3]	7.42 (1.23)	7.55 (1.29)	7.27 (1.14)	6.18 (.94)	5.76 (.80)
Mean (SD) Weekend[4]	6.44 (.79)	6.57 (.82)	6.30 (.74)	7.62 (1.25)	7.26 (1.19)
Mean (SD) Full Week[5]				6.70 (.84)	6.24 (.70)
Sleep Debt					
No. (%) No	159 (65.2%)	79 (61.7%)	80 (69.0%)	71 (66.4%)	88 (64.2%)
No. (%) Yes	85 (34.8%)	49 (38.3%)	36 (31.0%)	36 (33.6%)	49 (35.8%)

		Sex		Race	
	All (n=244)	**Female (n=128)**	**Male (n=116)**	**White (n=107)**	**Black (n=137)**
Daytime Sleepiness					
Mean (SD)	16.0 (3.67)	16.33 (3.51)	15.63 (3.83)	15.83 (3.56)	16.13 (3.77)
range	10 – 29	10 – 28	10 – 29	10 – 26	10 – 29

[1] p<.05 for sex comparison and p<.01 for race comparison;

[2] Raw values are shown in table; hsCRP levels were natural log transformed prior to analyses;

[3] p<.05 for sex and p<.001 for race comparisons;

[4] p<.05 for race comparison;

[5] p<.01 for sex and p<.001 for race comparisons

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Table 2

Associations among sleep duration and sleep debt with hsCRP levels[1]

	Beta	P value
Weeknight Sleep Duration	−0.04	0.51
Weekend Sleep Duration	−0.01	0.86
Average Sleep Duration	−0.04	0.54
Sleep Debt	0.03	0.62

[1] hsCRP level was natural log transformed prior to analyses