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Associations of Chronic Burden, Sleep Characteristics, and Metabolic Syndrome in the Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Abstract

Objective: Chronic exposure to stress is associated with metabolic syndrome (MetS), but the mechanism is unclear. We investigated associations between chronic burden, sleep and MetS in the CARDIA Study.

Methods: Chronic burden was self-reported (2000-2001) according to experiences with stressors for longer than 6 months. Wrist actigraphy-measured sleep duration and sleep efficiency were collected for 6-days; sleep duration, sleep quality and daytime sleepiness were self-reported (2003-2004). MetS was measured during the clinic visit, 2005-2006. Multivariable logistic and

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Conflicts of interest: None

Cox proportional hazard models were fit to determine the associations of interest. Mediation by sleep was assessed using the product of coefficients approach.

Results: Among participants (N=606), the average age was 40 years (standard deviation=3.6), 58% were female, and 43% were Black. The prevalence of chronic burden, short sleep (≤6 hours), and MetS was 35%, 43% and 20.5%, respectively. High vs. low chronic burden was associated with shorter self-reported sleep duration and higher daytime sleepiness. Chronic burden was associated with 1.85 higher odds (95% confidence interval:1.11, 3.09) of MetS. Sleep characteristics were not associated with MetS. There was no evidence that sleep mediated the chronic burden-MetS relation.

Conclusion: Burden of chronic stress may be an emerging novel risk factor for both poor sleep and MetS.

Keywords

Chronic Stress; Sleep; Actigraphy; Metabolic Syndrome

Introduction

Metabolic syndrome (MetS) is highly prevalent (20-25% of adults) and is a risk factor for cardiovascular morbidity and mortality [1-3]. A cluster of cardiovascular risk factors including abdominal adiposity, insulin resistance/hyperinsulinemia, hypertension, and dyslipidemia define MetS [1]. Given the high prevalence of these factors, it is critical to identify contributors to the development of MetS.

Emerging evidence suggests that psychosocial stress is a risk factor for MetS [4, 5]. Researchers have consistently found an association between MetS and various specific dimensions of psychosocial stress including marital stress, global perceived stress, work stress and psychological distress [4]. However, these prior studies were limited in studying a specific psychosocial stressor as opposed to assessing the impact or burden of several psychosocial stressors, which may have more of a physiological effect. Moreover, the mechanism linking psychosocial stress and MetS remains unclear. One plausible mediating mechanism through which psychosocial stress may contribute to MetS is sleep [6].

Multiple dimensions of psychosocial stressors are associated with sleep disturbances, particularly short sleep duration and poor sleep quality [7-10]. In addition, extreme sleep durations (e.g. short or long) and insomnia are associated with incident and prevalent MetS [11, 12]. However, the prior literature is limited to mainly assessing self-reported sleep, as opposed to objectively measured sleep. Self-reported sleep duration and quality and actigraphy-based sleep only moderately correlate, and are influenced by different biases, but yet may provide complementary information. Furthermore, prior study samples have been homogeneous in regard to race or sex. Thus, there is a clear need to investigate both objective and subjective sleep measures in relation to MetS in a diverse population and determine the potential mechanism between burden of psychosocial stress, sleep and MetS.

Among participants of the Coronary Artery Risk Development in Young Adults Study (CARDIA), we aimed to determine (1) the association of burden of psychosocial stress,

hence forward referred to as chronic burden, with sleep characteristics and MetS, and (2) whether objectively and subjectively measured sleep duration and quality mediate the association between chronic burden and MetS.

Methods

CARDIA is a large multi-center cohort study to assess development and progression of cardiovascular disease (CVD) risk factors in a U.S. sample of White and Black adults. Details of the CARDIA recruitment and study protocol have been previously published [13]. Briefly, study enrollment occurred from 1985-1986 across four U.S. regions (Birmingham, Alabama; Minneapolis, Minnesota; Chicago, Illinois; and Oakland, California), with inclusion of a total of 5,115 adult men and women between the ages of 18 and 30. Those with a previous history of symptomatic/clinical CVD were excluded during initial study enrollment (1985-1986). A balanced racial/ethnic composition was achieved with inclusion of 48.5% White participants and 51.5% of Black participants. Participants have been followed across a 30-year time period, with a total of eight follow-up examinations to date. The current study includes data from the CARDIA sleep ancillary study (described below), 669 participants. However, individuals who did not attend Year 20 exam (N=46) and those with missing covariates from Year 15 exam (N=18) were excluded from the current analysis. The present main analysis was restricted to 606 participants from the Chicago field center who underwent actigraphy; covariates were measured at the Year 15 and Year 20 follow-up examinations that took place in 2000-01 and 2005-06. In secondary analyses, we examined associations with incident MetS through Year 30 (2016). A timeline for the data collection of the study variables is shown in Supplemental Figure 1 (Supplemental Digital Content).

Metabolic Syndrome

Participants of the Year 20 and 30 exams had a clinic visit which included a fasting blood draw, and measures of obesity and resting blood pressure. MetS was classified according to the presence of at least three of the following: 1. Waist girth>=102 cm for men or >=88 cm for women; 2. Triglycerides>=150 mg/dL; 3. High-density lipoproteins (HDL) <40 mg/dL for men or <50 mg/DL for women; 4. Systolic blood pressure (SBP) ≥130 mmHg or diastolic blood pressure (DBP) ≥85 mmHg or self-reported use of hypertensive medications; 5. Fasting glucose ≥100 mg/dL or diabetes control medications.

Chronic Burden

Participants indicated whether they have experienced certain strains for longer than 6 months during the Year 15 examination. The strains included serious ongoing health problem (yourself or someone close to you), ongoing difficulties with your job or ability to work, ongoing financial strain, and ongoing difficulties in a relationship with someone close to you [14]. The response scale included no; yes, but not very stressful; yes, moderately stressful; and yes, very stressful. To define the burden of stress, the number of strains that were moderately or very stressful were summed to produce an overall score (range from 0 to 5). Few participants reported 3, 4, or 5 strains (11.2%, 5.6%, 1.2%, respectively), therefore

chronic burden was analyzed as categories of low (0 strains), medium (1 strain), and high (2+ strains).

Sleep Characteristics

Actigraphy-measured (objective) and self-reported sleep were collected 2003-2004. Actigraphy-measured sleep were obtained as part of an ancillary to the original CARDIA Study (n=669). The sleep ancillary study included participants from Chicago, one of four CARDIA sites. Non-pregnant participants in the CARDIA Year 15 clinical examination were invited to participate in the sleep study. Details regarding the ancillary study have been previously published [15]. In brief, participants wore a wrist activity monitor (Actiwatch-16; Mini-Mitter Inc, Bend, Oregon) for three consecutive days on two occasions approximately 1 year apart between 2003-2005. Actigraphy-based sleep duration and sleep efficiency were assessed. Sleep duration was defined as the total amount of time spent asleep in the sleep period. Sleep duration was analyzed continuously and categorically (≤6 hours for short sleep vs. >6), which was determined after review of the sleep duration distribution. Sleep efficiency was defined as the proportion of time spent asleep while in bed, i.e., sleep time divided by time in bed. Sleep efficiency was analyzed continuously and dichotomized at <85% to indicate poor sleep efficiency.

Self-reported sleep measures included sleep duration, sleep quality (Pittsburgh Sleep Quality Index, PSQI), and daytime sleepiness (Epworth Sleepiness Scale, ESS). Sleep duration was measured using the following question: “During the past month, how many hours of *actual sleep* did you get at night? (This may be different than the number of hours you spend in bed.). Self-reported short sleep duration was defined as ≤6 hours. The PSQI was included to assess sleep quality according to the following components: sleep duration, sleep disturbances, sleep latency, daytime dysfunction due to sleepiness, sleep efficiency, overall sleep quality, and sleep medications. The seven components were each scored 0 (no difficulty) to 3 (severe difficulty) and summed to produce an a global score (range from 0-better to 21-worse) with >5 indicating poor sleep quality [16]. The Epworth Sleepiness Scale assessed daytime sleepiness by asking participants to rate their likelihood of falling asleep in eight scenarios, scored on a scale from 0 (not likely) to 3 (highly likely), with the score ranging from 0 to 24 (17).

Covariates

The following sociodemographic characteristics from Year 15 were included as covariates: age, sex, race, education, income, and marital status. Age and sex were self-reported. Participants indicated their race as Black or White. Education was reported as the highest grade (or year) of regular school completed and further categorized as less than high school, high school or GED, or college graduate or higher. Income was self-reported as total combined family income for the past 12 months, and was analyzed as an ordinal variable ranging 0 to 6 (i.e., <$16,000, $16,000 to $24,999, $25,000 to $34,999, $35,000 to $49,999, $50,000 to $74,999, $75,000 to $99,999, and $100,000 or more) with higher scores representing higher income. Marital status was classified as three categories: 1) married or living with someone in a marriage-like relationship; 2) divorced, widowed, separated; 3) or never married. For physical activity, participants were asked about participation in

13 specific moderate- and vigorous-intensity activities over the previous year, including exercise, sports, home maintenance, and occupational activities. Each activity was assigned an intensity score (range: 3-8 metabolic equivalents) and a duration threshold (ranging: from 2-5 hours/week). These scores were summed across activities to define the usual level of activity over the previous year.[18] Smoking was classified as current smoker vs. never or former smoker. Alcohol consumption was reported as the number of beverages in the past week and was analyzed as the total ml/day-a value of 0 was recorded for those that do not consume alcohol. Depressive symptoms were assessed according to the Center for Epidemiologic Studies Depression Scale (CES-D) and was analyzed continuously.

Statistical Analysis

Descriptive analyses were executed to examine the distribution of key variables across categories (low, medium, high) of chronic burden. We conducted preliminary analyses to test for linearity for the continuous variables. A series of linear and logistic models were fit to test the associations between chronic burden and sleep characteristics. For our primary analyses, separate multivariable logistic regression models were fit to test the association between chronic burden and MetS. Models were adjusted for age, sex, race, education, income, insurance status, physical activity, marital status, smoking, alcohol, and depressive symptoms. Three additional models were fit in which actigraphy-measured sleep duration (Model 2), actigraphy-measured sleep efficiency (Model 3) and self-reported sleep duration, daytime sleepiness and sleep quality (PSQI) were added as predictors of MetS (Model 4). In exploratory analyses, we restricted the sample by race and tested the associations between chronic burden, sleep, and MetS. Our subgroup analyses were based on the literature demonstrating racial differences in stress, sleep and CVD[19], as opposed to statistical significance. We also examined chronic burden and sleep measures separately with the components of MetS.

In secondary analyses, we expanded the analysis to the entire CARDIA sample and conducted a prospective analysis of incident MetS by year 30, excluding those with MetS from years 0 to 15. Cox proportional hazard models were fit with the same model adjustment as above. This analysis includes only self-reported sleep measures that were collected across the exams, self-reported sleep duration and daytime sleepiness.

Mediation Analysis:

A formal causal mediation analysis was conducted using the Valeri-VanderWeele SAS macro adapted for mediation analysis when the independent variable has more than two categories [20]. The direct effect of chronic burden and the indirect effect through sleep measures was estimated specifying 10,000 bootstrap samples for the 95% confidence limits.

Results

Selected sample characteristics by the total sample and categories of chronic burden are displayed in Table 1. The total sample had an average age of 40.1 (standard deviation (SD)= 3.6) years, 57.9% were female, 56.9% were White and 43.1% Black, and 52.7% had a college degree or higher. Participants with a high chronic burden relative to low or

medium, were less likely to be insured or married, and more likely to be a smoker and report depressive symptoms, $p<0.05$. Self-reported sleep characteristics such as daytime sleepiness, poor sleep quality and self-reported short sleep duration were more common among those with high vs. low or medium chronic burden, $p<0.01$. Whereas, their profile of actigraphy-measured sleep characteristics was similar across categories of chronic burden.

Chronic Burden and Sleep Characteristics

Associations between chronic burden and sleep characteristics are shown in Table 2. High compared to low chronic burden was associated with 2.06 higher odds (95% confidence interval: 1.32, 3.22) of self-reported short sleep duration and 1.63 higher odds (1.01, 2.65) of daytime sleepiness. There was a suggestive association between high chronic burden compared to low and worse sleep quality. In examining chronic burden with continuous self-reported sleep duration, high chronic burden was associated with sleeping 30 minutes less on average compared to low chronic burden. There were no associations between chronic burden and actigraphy-measured sleep characteristics.

Similar to the overall findings, there was no association observed between chronic burden and actigraphy-measured sleep duration or sleep efficiency among Black or White participants (Supplemental Table 1). For self-reported sleep duration, Black participants had a 2.31 (1.19, 4.52) greater odds of self-reported short sleep duration. Contrary to the overall findings, there was no significant association between chronic burden and daytime sleepiness, although the separate odds ratios (OR) for Black and White adults were similar in magnitude to the OR for the whole sample. However, Black participants with medium and high chronic burden compared to low, had greater odds of poor sleep quality OR: 2.31 (1.09, 4.88) and OR: 2.33 (1.08, 5.00), respectively; no association was observed in the overall sample. There were no observed associations between chronic burden and sleep among White adults.

Chronic Burden, Sleep Characteristics and Metabolic Syndrome

The odds of MetS with chronic burden and sleep characteristics are shown in Table 3. After adjusting for sociodemographic characteristics, high chronic burden was associated with 85% higher odds [OR: 1.85 (1.11, 3.09)] of MetS relative to low chronic burden. Actigraphy-measured sleep duration, sleep efficiency, and self-reported sleep characteristics were added separately as predictors of MetS in Models 2, 3, and 4 respectively. Neither actigraphy nor self-reported sleep measures were associated with MetS. The association between chronic burden and MetS did not substantially change with the adjustment for sleep characteristics. There was no evidence that any of the explored sleep measures mediated the association between chronic burden and MetS (Supplemental Table 2).

High chronic burden was associated with incident MetS (hazard ratio [HR]: 1.36, 95% CI: 1.12, 1.66). This association persisted with adjustment of self-reported sleep duration and daytime sleepiness (Table 4).

In general, the associations between chronic burden and MetS were attenuated for both Black and White adults (Supplemental Table 3). However, in the fully adjusted model with actigraphy-measured sleep efficiency (Model 3), the significance of the association between

chronic burden and MetS persisted for Black adults only, although the magnitude for White adults was similar to the magnitude for Black adults. There were no observed associations between chronic burden and incident MetS for Black adults, but the association persisted for White adults (Supplemental Table 4).

Supplemental Analyses

To further understand the results of these analyses, we explored chronic burden and sleep characteristics with the components of MetS. High chronic burden was associated with higher odds of low HDL-C, OR: 1.95 (1.22, 3.09); this association was independent of sleep measures. There were no other associations between chronic burden and other components (e.g., waist, triglycerides, blood pressure, fasting glucose) of MetS (Supplemental Table 5). Longer actigraphy-measured sleep duration and greater sleep efficiency was associated with lower odds of elevated blood pressure, OR: 0.81 (0.66, 1.01) and OR: 0.97 (0.95, 0.99), respectively. Worse sleep quality measured by the PSQI was associated with a high waist girth (i.e., >=102 cm for men or >=88 cm for women), OR: 1.14 (1.06, 1.24). Remaining sleep characteristics (actigraphy and self-reported) were not associated with the other components of MetS (Supplemental Table 6).

We examined the components of chronic burden (i.e., own health problems, health problems of others, job or ability to work, relationships, finances) with sleep characteristics and MetS (Supplemental Table 7-9). Financial stress was associated with higher odds of self-reported short sleep duration, OR: 1.56 (1.01, 2.42). Remaining individual components of chronic burden were not associated with other sleep characteristics or prevalent MetS. Job or ability to work and finances were associated with incident MetS, HR: 1.29 (1.06, 1.57) and HR: 1.24 (1.02, 1.51), respectively. There was no association between the remaining components and incident MetS.

Discussion

In a sample of Black and White adults in Chicago IL, we found that (1) chronic burden was associated with shorter self-reported sleep duration and more daytime sleepiness in the overall sample; (2) associations between chronic burden and self-reported sleep duration and sleep quality were particularly pronounced among Black participants; and (3) chronic burden but not sleep characteristics were associated with MetS. There was no evidence that sleep mediated the association between chronic burden and MetS. We found the burden of experiencing chronic stress had more utility than the individual components, thus highlighting the importance of burden to health outcomes. Our results indicate that the burden of chronic stress may influence self-reported sleep duration in diverse samples, particularly among Black adults. These results have significant implications, given self-reported sleep duration is a risk factor for cardiovascular disease [21], which is disproportionately prevalent among Black adults [22].

MetS is associated with increased risk for diabetes, heart disease, stroke and cardiovascular disease [23]. These chronic conditions lead to mortality and have important implications for quality of life [24], thus underscoring the importance of reducing the burden of MetS. Traditional approaches to preventing MetS include pharmacological strategies and lifestyle

changes (e.g., diet and exercise). These approaches largely ignore stress reduction. Our findings suggest that the burden from experiencing chronic stress increases the odds of MetS by 85%. This is consistent with prior research that has demonstrated that psychosocial stress is associated with incident MetS [4]. It is plausible that exposure to chronic stress may lead to MetS through stress-induced changes in the hypothalamic-pituitary-adrenal (HPA) axis or through engaging in unhealthy behaviors such as smoking, consumption of unhealthy foods, physical inactivity or heavy drinking [25]. Other lifestyle factors such as sleep, can also be affected by stress and lead to MetS. In examining chronic burden and components of MetS, we found that high chronic burden was associated with higher odds of low HDL, even after adjustment for physical activity, which is typically protective. Our result is consistent with the literature. For example, studies have found that psychological stress is a risk factor low HDL [26, 27]. In fact, a review article reported that 6 of 8 studies reported higher rates of depression, anxiety, suicide attempts, and violent behaviors in participants with low HDL [28]. Studies have shown that having more control over strains, such as work is associated with higher HDL [29]. Our results suggest that HDL may be the driver of the chronic burden – MetS relation; future research should further examine this association. Researchers have suggested that MetS may have limited practical utility as a screening or management tool [30]. Thus, it may be more informative to investigate the components of MetS to provide more insight on metabolic pathways underlying CVD development and progression.

Although prior studies have shown an association between sleep and MetS [11, 12], there was no observed association in the current study. The null association may be attributable to MetS as a cluster of several conditions. Mozaffarian and colleagues found that examining MetS criteria individually was more predictive of mortality than MetS as one measure [24]. In examining sleep characteristics with the individual components of MetS, longer actigraphy-measured sleep duration and greater sleep efficiency were associated with lower odds of elevated blood pressure, and the PSQI (higher scores relate to worse sleep quality) was associated with larger waist girth; there were no other observed associations between sleep and components of MetS. It is important to note that prior papers among CARDIA participants have reported a cross-sectional association between sleep and cardiovascular risk factors including body mass index, blood pressure, and lipids [31-33]. More specifically, long sleep duration was associated with lower blood pressure and BMI as well as higher low-density lipoproteins [31-33]. The observed differences in the effect estimates are likely attributable to the modeling of the outcome variables. In the current study, we dichotomized each component according to the MetS definition, whereas in prior studies it was analyzed continuously. The 3-day measurement for actigraphy may have been inadequate to fully capture the relevant sleep metrics, including sleep timing variability, which was recently found to be associated with MetS in a multi-ethnic sample [34]. Also, our lack of a sleep – MetS relation may be due to the inclusion of sleep characteristics as opposed to sleep disorders. The pathways by which sleep may be associated with MetS likely involve sleep apnea, which is associated with obesity and subsequently with insulin resistance and CVD [35]. Obstructive sleep apnea may lead to intermittent hypoxia and/or sleep fragmentation, which can lead to MetS through inflammation [36]. Polysomnography was not conducted in the study, thus, there was no objective measure of sleep apnea available. Additionally, assessing sleep apnea was outside the scope of the current paper. Our primary

interest was disentangling the relationship of psychosocial stress, sleep characteristics (e.g., sleep duration and quality) and MetS. As a result of the null association between sleep characteristics and MetS, there was no evidence of the measured sleep characteristics mediating the chronic burden and MetS relation.

Despite the lack of an association between sleep and MetS, we found that chronic burden was associated with self-reported sleep characteristics. This finding is consistent with the stress and sleep literature [7, 8, 37, 38], but adds a component-burden of stress. Of note, we found a strong association between chronic burden and self-reported sleep duration. In fact, participants with high chronic burden reported sleeping 24 minutes less on average relative to those with a low chronic burden. Similarly, we observed that chronic burden was also related to daytime sleepiness, which may be shorter sleep duration or poorer quality of sleep or less restorative sleep associated with a high stress burden. Among the Black participants, we found stronger associations between chronic burden and self-reported short sleep duration and sleep quality. It is also important to note that there were no associations between chronic burden and actigraphy-measured sleep. These results suggest that a self-reported measure of chronic burden may relate more to perceptions of sleep (self-reported). While self-reported sleep is limited by various reporting biases, it represents a patient-reported outcome that is important to patients and a predictor of important health endpoints [21]. Also, self-reported data may provide information over longer time frames than 3 days of objective monitoring from actigraphy, and thus may be more representative. On the other hand, actigraphy provides the ability to quantify sleep continuity and aspects of sleep timing. Our findings underscore the value in collecting self-reported as well as objective data.

In contrast to our findings with self-reported sleep duration and daytime sleepiness, there was no association between chronic burden and sleep quality in the overall sample. This finding is not consistent with the literature [8, 39, 40]. The lack of association may be due to our measure of chronic burden which assesses the burden of chronic stress over time, whereas prior studies have mainly focused on perceived stress and not the assessment of the impact of the stress. However, this may vary by race. Among Black participants, chronic burden was associated with poor sleep quality. This finding is consistent with the literature that has found that psychosocial stress is associated with sleep quality among Black adults [8, 41]. Consistent across research studies, Black adults report poorer sleep quality and have objectively worse sleep continuity (i.e., lower sleep efficiency, more wakefulness after sleep onset) compared to White adults [41-43]. Research suggests that discrimination or race-related stress may explain the racial difference in sleep quality [43, 44]. Our results indicate that chronic burden may be a target for intervention to improve sleep quality among Black adults.

There are many strengths to our study. We expanded the assessment of stress to include assessing the influence of the burden of chronic stress on health, which is less frequently studied but has important implications for health. More specifically, the inclusion of chronic burden, as a psychosocial factor extends the literature to assessing the potential effects of prolonged activation of neurobiological pathways, such as the HPA axis, which can result in elevated inflammatory response [45]. Future research should measure the context

of the stress exposure in order to determine the response to stress and whether it will have a cumulative effect beyond a single stressor exposure [46]. Sleep was measured both subjectively as well as objectively with actigraphy, however, there are limitations to self-reported sleep, as an overestimate, which we attempted to address by defining short sleep duration as ≤6 hours. Close to half of our sample identified as Black, which expands the diversity of the literature, which typically has limited minority representation. The longitudinal design of CARDIA allowed the assessment of the development of MetS in relation to chronic burden. However, the sleep measures were collected around the same time as the MetS data, therefore the temporal association was unknown. Also, there was no baseline measure of sleep, therefore we were unable to test whether chronic burden led to shorter sleep duration or poorer sleep quality. The time difference between the assessment of chronic burden (2000-01) and the sleep measures (2003-04) present a limitation, particularly because experiences of stress and/or burden of stress may change over time. Therefore, the chronic burden score may not be an accurate representation of the current experience during the sleep ancillary study. Other limitations include a lack of generalizability due to the inclusion of a single site analysis. The participants in the current study are likely not representative of the US due to the limited racial composition of the sample.

Conclusion

Our results suggest that chronic burden is an emerging novel risk factor for short sleep duration and MetS. These findings also demonstrate associations with sleep are dependent on measurement (self-reported vs. objective). Mitigating chronic burden may be a promising intervention target for the reduction of poor sleep and MetS. Future studies should explore the pathway by which the burden of psychosocial stress is associated with MetS.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

MetS	metabolic syndrome
CARDIA	Coronary Artery Risk Development in Young Adults Study
CVD	cardiovascular disease
HDL	high-density lipoproteins
SBP	systolic blood pressure

DBP	diastolic blood pressure
PSQI	Pittsburgh Sleep Quality Index
ESS	Epworth Sleepiness Score
GED	general educational development
CES-D	Center for Epidemiologic Studies Depression Scale
HPA	hypothalamic-pituitary-adrenal

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Table 1.

Study Sample Characteristics (2000-01 or 2003-04*) According to Categories of Chronic Burden (N=606), 2000-2001)

		Chronic Burden		
	Total sample Mean (SD) or %	**Low N=216 Mean (SD) or %**	**Medium N=175 Mean (SD) or %**	**High N=215 Mean (SD) or %**
Age	40.1 (3.6)	40.0 (3.4)	40.2 (3.9)	40.2 (3.5)
Sex				
Female	57.9	56.5	53.7	62.8
Male	42.1	43.5	46.3	37.2
Race				
White	56.9	54.6	62.3	54.9
Black	43.1	45.4	37.7	45.1
Education				
Less than high school	4.6	6.0	3.4	4.2
High school or GED	15.8	16.2	12.6	18.1
Some college	26.9	27.3	28.6	25.1
College degree	25.3	26.4	22.9	26.1
College degree +	27.4	24.1	32.6	26.5
Family income				
<$50,000	32.7	33.3	24.0	39.1
≥$50,000	67.3	66.7	76.0	60.9
Insured	87.3	87.5	92.0	83.3
Marital Status				
Married	61.9	62.0	70.3	54.9
Widow, separated, divorced	16.7	16.7	11.4	20.9
Never married	21.4	21.3	18.3	24.2
Physical activity	366.7 (298.6)	379.7 (311.5)	358.6 (287.8)	360.4 (294.9)
Alcohol consumption, ml/day	10.5 (20.1)	9.4 (19.3)	9.8 (14.3)	12.3 (24.4)
Current smoker	19.3	16.7	14.9	25.6
Depressive symptoms	9.1 (7.8)	6.9 (6.7)	7.4 (6.3)	12.6 (8.7)
Actigraphy-measured sleep duration, hours *	6.1 (1.0)	6.1 (1.1)	6.2 (1.0)	6.1 (1.1)
Actigraphy-measured sleep duration ≤6 hours *	42.7	42.3	41.7	43.9
Actigraphy-measured sleep efficiency *	81.1 (10.0)	80.2 (12.0)	82.3 (9.2)	81.1 (8.5)
Actigraphy-measured sleep efficiency <85% *	58.5	59.4	51.2	63.6
Daytime sleepiness *	7.4 (4.0)	6.7 (3.5)	7.2 (4.1)	8.4 (4.2)
PSQI *	5.8 (2.8)	5.2 (2.6)	5.6 (2.8)	6.5 (3.0)
Self-reported sleep duration, hours *	6.6 (1.2)	6.8 (1.1)	6.6 (1.1)	6.4 (1.3)
Self-reported sleep ≤6 hours *	37.1	28.8	37.2	45.5

PSQI=Pittsburgh Sleep Quality Index

*Measure from (2003-04) ancillary study

Table 2.

Association Between Categories of Chronic Burden (2000-01) and Categorical Sleep Outcomes (2003-04), CARDIA N=604

	Actigraphy-measured sleep duration ≤ 6 hours		Actigraphy-measured sleep efficiency < 85%		Self-reported sleep duration ≤ 6 hours		Daytime sleepiness > 10		PSQI ≥ 5	
	Odds ratio	**95% CI**	**Odds ratio**	**95% CI**	**Odds ratio**	**95% CI**	**Odds ratio**	**95% CI**	**Odds ratio**	**95% CI**
Chronic burden										
Low	REF	REF	REF	REF	REF	REF	REF	REF	REF	REF
Medium	1.15	0.72, 1.81	0.77	0.49, 1.20	1.42	0.92, 2.21	1.07	0.64, 1.79	1.30	0.84, 2.00
High	0.89	0.56, 1.41	1.07	0.68, 1.68	2.06[a]	1.32, 3.22	1.63[b]	1.01, 2.65	1.48[c]	0.94, 2.33

[a] $P \leq 0.01$

[b] $P \leq 0.05$

[c] $P \leq 0.10$ from Wald Chi-Square test; Logistic Regression Model adjustment: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, and depressive symptoms.

Table 3.

Logistic Regression Models for Association between Categories of Chronic Burden (2000-01) and Sleep Measures (2003-04) with Odds of Metabolic Syndrome (2005-06), CARDIA N=606

	Model 1		Model 2		Model 3		Model 4	
	Odds ratio	**95% CI**	**Odds ratio**	**95% CI**	**Odds ratio**	**95% CI**	**Odds ratio**	**95% CI**
Chronic burden								
Low	REF	REF	REF	REF	REF	REF	REF	REF
Medium	1.00	0.58, 1.73	1.03	0.60, 1.79	1.03	0.59, 1.79	0.94	0.54, 1.65
High	1.85[b]	1.11, 3.09	1.94[a]	1.16, 3.25	1.97[a]	1.17, 3.31	1.84[b]	1.09, 3.10
Actigraphy-measured sleep duration			1.01	0.81, 1.27				
Actigraphy-measured sleep efficiency					0.99	0.96, 1.01		
Self-reported sleep duration							1.00	0.83, 1.19
Daytime sleepiness							1.00	0.95, 1.06
PSQI							1.03	0.95, 1.12

[a] $P \leq 0.01$

[b] $P \leq 0.05$, from Wald Chi-Square test

Model 1: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, and depressive symptoms

Model 2: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, depressive symptoms, and actigraphy-measured sleep duration

Model 3: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, depressive symptoms, and actigraphy-measured efficiency

Model 4: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, depressive symptoms, self-reported sleep duration, daytime sleepiness, and sleep quality

Table 4.

Cox Regression Models for Association between Categories of Chronic Burden (2000-01) and Sleep Measures (2000-01) with Incident Metabolic Syndrome, CARDIA, N=2601

	Model 1		Model 2		Model 3	
	HR	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**
Chronic burden						
Low (reference)						
Medium	0.93	0.75, 1.16	0.92	0.75, 1.15	0.93	0.75, 1.16
High	1.36[a]	1.12, 1.66	1.36[a]	1.12, 1.65	1.36[a]	1.12, 1.66
Self-reported sleep duration			1.00	0.93, 1.07		
Daytime sleepiness					1.00	0.83, 1.21

[a] $P \leq 0.01$, from Wald Chi-Square test

Model 1: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, and depressive symptoms

Model 2: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, depressive symptoms, and self-reported sleep duration

Model 3: age, sex, race, education, income, insurance, marital status, physical activity, alcohol, smoker, depressive symptoms, and daytime sleepiness