Self-Reported Sleep Impairment and the Metabolic Syndrome among African Americans

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Objectives: African Americans (AA) experience a high mortality from cardiovascular disease (CVD), even without an increase in the prevalence of the metabolic syndrome (MetS). The potential role of sleep impairment in this phenomenon has not been studied. The current study examined the relationship between self-reported sleep and MetS components among AAs. Sleep variables included total sleep quality and specific symptoms: loud snoring, difficulty breathing, and sleep duration.

Design: Anthropometric (BMI, BP, waist circumference, body fat percent) and biologic (fasting glucose, triglycerides, total cholesterol, and HDL) measures were obtained from 248 community-recruited AA (63% female; mean age 44 years). The Pittsburgh Sleep Quality Index (PSQI), a 19-item scale with a total sleep quality score and 7 subscales, was used to assess self-reported sleep quality. Analyses were controlled for age and sex.

Results: PSQI total sleep quality predicted neither presence of MetS (Beta=.04, P=.29) nor individual CVD variables. However, symptomatic snoring corresponded with MetS (Beta=.38, SE=.12, P<.001; OR: 2.57), as well as with fasting glucose, BMI, body fat percentage, and waist circumference.

Conclusions: Among AA, overall sleep quality as self-reported may not contribute to MetS, but symptomatic snoring appears to be important. Further work in this area should focus on sleep at the symptomatic level, and include racial and sex variables, as well as physiologic and etiologic mechanisms. (*Ethn Dis*. 2012; 22[4]:410–415)

Key Words: African Americans, Metabolic Syndrome, Sleep, Snoring, PSQI

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Introduction

Rising rates of obesity have led researchers to identify factors that place individuals at risk for cardiovascular disease (CVD). The most widely researched risk factors are included in the criteria for metabolic syndrome (MetS), which is a constellation of health risks, covering abdominal obesity, insulin resistance, hypertension, and hyperlipidemia.[1] An individual with MetS is at higher risk for diabetes and other cardiovascular-related diseases.[1] MetS is controversial because its components have varying etiologies and are manifested differently by race and sex. In particular, African Americans (AA) have a higher prevalence and mortality from cardiovascular diseases (CVD) than Caucasians, and yet have comparable, if not lower, rates of MetS,[2–5] at least in part due to MetS criteria which are based on Caucasian samples.[3,6]

Sleep research may be one important frontier in understanding MetS, as aspects of sleep can affect daily health behaviors.[7] The two most common sleep disorders are insomnia and obstructive sleep apnea (OSA).[8–10] Insomnia consists of non-restorative sleep due to trouble falling or staying asleep. When poor sleep is caused by nighttime breathing problems, it is classified as a breathing-related sleep disorder, the most common of which is OSA. These disorders have overlapping symptoms, although insomnia is more frequently characterized by difficulty getting to sleep, whereas OSA is characterized by excessive daytime sleepiness.[9] Many individuals also exhibit abnormal nighttime breathing, which is not accompanied by any impairment.[9,10]

MetS is weakly or, at best, moderately more common among people with insomnia[11]; correlations with OSA are stronger and more consistent than insomnia.[9,10,12–18] Overall sleep quality tends to be weakly related to MetS, whereas specific sleep symptoms – particularly snoring and sleep-disordered breathing – are stronger markers of MetS than sleep quality. For instance, one cross-sectional study of 210, mostly Caucasian, community-recruited participants[19] used a standardized questionnaire of self-reported sleep quality, The Pittsburgh Sleep Quality Index, PSQI,[20] and found a weak relationship between overall sleep quality and MetS. Furthermore, sleep quality related to some components of MetS (glucose, central obesity, body fat, insulin resistance), but not others (diastolic and systolic blood pressure, triglycerides, HDL). The authors noted that correlations between sleep quality and MetS increased after the 10 AA participants were excluded from the analyses. Subsequent longitudinal research, using more racially diverse samples,[21,22] found that symptomatic snoring, but not overall sleep, predicted the development of MetS. Taken together, these studies underscore the importance of looking at both overall sleep and sleep symptoms in relation to MetS, as well as focusing on specific racial groups.

A key similarity between sleep disorders and MetS is their differential manifestations in different populations.[23–25] A recent meta-analysis[24] examined studies comparing rate and severity of sleep disorders between Caucasians and AA. Overall, Caucasians were more likely to report symptoms of insomnia, while AAs were more likely to report OSA and sleep-disordered breathing. Some studies have identified other racial differences with regard to sleep patterns and overall, AA have

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shorter sleep duration (both self-reported and objective) and exhibit poorer sleep quality compared to Caucasians.[26,27] Results from the 2005 National Health Interview Survey of 29,818 Americans[27] found both short- and long-sleepers to be at greater risk for type 2 diabetes, with larger effect sizes among AA than Caucasians. In sum, AA experience different, and possibly more severe, sleep problems than Caucasians; it is likely these differences are associated with CVD risk.

The current study sought to replicate the findings of Jennings et al[19] in a larger AA sample by using the same self-reported sleep measure (PSQI). Another objective was to explore the relationship between sleep and MetS by focusing on specific aspects of sleep identified in the literature, which may be equal or more important than overall sleep quality. Sleep variables of interest include self-reported loud snoring, difficulty breathing, and sleep duration.

METHODS

Participants included self-identified AA men and women aged 18–60 years old. They were recruited through newspaper ads and community bulletins for a larger experiment addressing health disparities. Participants who were pregnant or taking steroid medications were excluded for reasons related to the experiment (which is not reported here). Aside from these two criteria, participants were not screened based on health status or medical conditions. The study was approved by the institutional review board of the Uniformed Services University of the Health Sciences, and written informed consent was obtained from all participants.

Measures

Prior to visiting the lab, participants were asked to 1) fast for 9 hours, 2) avoid caffeine and alcohol for 9 hours, and 3) avoid non-essential medications for 24 hours. They arrived at the laboratory between 7:00 and 9:00 AM, underwent a thorough CVD-risk assessment (detailed below), and were then offered time to eat a snack (if hungry) before completing a series of questionnaires and undergoing other measures.

Participants had MetS if they met at least three of the following five criteria[1]:

- Blood pressure≥130/85 mm Hg
- Fasting glucose≥6.1 mmol/L
- Waist size>88.9 cm for males, or 101.6 cm for females
- HDL≥2.2 mmol/L for males, or 2.8 mmol/L for females
- Triglycerides≥8.3 mmol/L

Body weight was measured with a calibrated balance beam metric scale to the nearest .1 kg, and height was measured to the nearest .1 cm, while the participant was wearing light clothing and no shoes. BMI was calculated from height and weight. Percentage body fat was estimated by bioelectric impedance with the portable RJL body composition analyzer (RJL Systems; Clinton Township, Mich.). Waist and hip circumferences were measured with a non-elastic tape (Gulick II Measuring Tape, by Country Technology, Gays Mills, Wisc.) around the waist and hip by using standard techniques. Fasting blood samples were collected to measure glucose (participants had fasted for at least nine hours), triglycerides, total cholesterol, and high density lipoprotein cholesterol.

Participants filled out questionnaires covering demographics, medical history, sleep, and other factors relevant to the larger study. Self-reported sleep was assessed using the PSQI,[20] a 19-item scale that assesses sleep during the past month using seven heterogeneous subscales: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, sleep medication, and daytime dysfunction. The subscales are summed to calculate a total sleep quality score. Three additional variables comprised individual PSQI items measuring sleep duration (''How many hours of actual sleep did you get at night?'') in hours, and difficulty breathing (''How often have you had trouble sleeping because you cannot breathe comfortably?'') and loud snoring (''How often have you had trouble sleeping because you cough or snore loudly?'') along a 4-point Likert scale (''not during the past month,'' ''less than once a week,'' ''once or twice a week,'' ''three or more times a week''). The PSQI has been widely used, and responses are reliable, valid,[20,28] and consistent over time.[29] Internal consistency for global sleep quality is calculated using the seven subscale scores. Buysse[20] originally reported an internal consistency of .83 among a group of clinic patients and healthy controls; internal consistency for our sample was somewhat lower (.72), which is likely due to differences between our sample and Buysse's.

Statistical Analysis

Frequency tables and descriptive statistics were reviewed to remove outliers and confirm assumptions for parametric tests. Logistic regression and analysis of covariance were used to test the relationships between sleep factors and MetS. Independent variables were categorical, and included global sleep quality (dichotomized, with PSQI > 5 indicating disordered sleeping),[20] symptomatic snoring and breathing (both symptoms dichotomized by frequency

Table 1. Sample characteristics, *n*=248

Variable	Mean ± SD
Age, y	44 ± 11.5
Women, *n* (%)	151 (63%)
PSQI global score	6.6 ± 3.8
PSQI, disturbing snoring[a]	1.8 ± 1.2
PSQI, disturbing breathing[a]	1.5 ± .9
PSQI, sleep duration, hours	6.5 ± 1.5
Blood pressure, mm Hg	
Systolic	133 ± 16.5
Diastolic	83 ± 12.9
Mean arterial pressure, mm Hg	174.3 ± 21.7
BMI	30.4 ± 8.7
Waist circumference, cm	96.3 ± 18.7
Fat percentage	34.6 ± 10.7
Total cholesterol, mmol/L	4.1 ± 1.0
Triglycerides, mmol/L	1.2 ± .9
HDL, mmol/L	1.3 ± .4
Fasting glucose, mmol/L	6.2 ± 1.9

PSQI, Pittsburgh Sleep Quality Index.

[a] Item answered along of 1–4 Likert scale, with higher numbers indicating more frequent symptom occurrence.

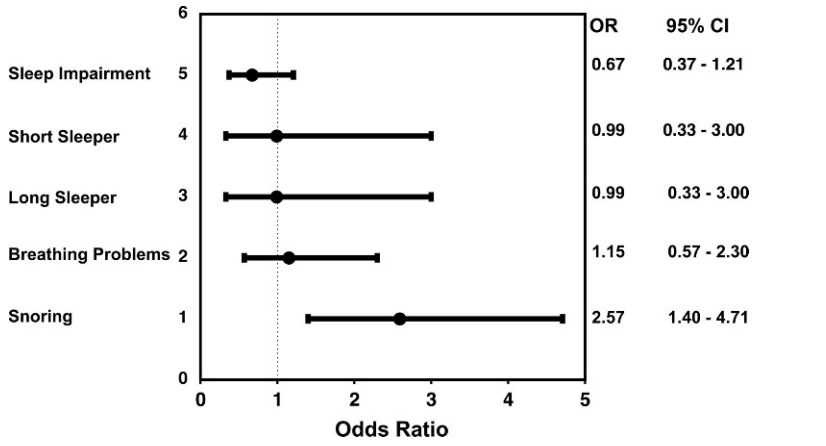

Fig 1. Sleep impairment risk factors for MetS. Odds that groups of participants with different sleep impairments have MetS. Odds ratios are adjusted for age and sex. All categories are dichotomous. Sleep impairment is defined by total PSQI score of 6 or greater; normal sleepers (6–9 hours per night) were used as the reference group for short and long sleepers. Breathing problems and snoring were dichotomized by minimal (less than once per week) vs moderate frequency (once or more per week) of the symptom

of once or more/less than once per week), and sleep duration (trichotomized, due to possible U-shaped effect,[27,30] coded as: short, ≤6 hours; normal/reference, 6–9; and long, ≥9).

Replicating Jennings et al,[19] separate multiple regression models were used to test the relationships between global sleep quality and CVD risk factors (BP, fasting glucose, triglycerides, HDL, waist circumference, BMI, and body fat percentage).

Lastly, exploratory analyses were conducted to further examine the relationship between the sleep factors that were associated with MetS and specific CVD-risk factors. All tests controlled for age and sex.

RESULTS

Demographics

Characteristics of the sample are presented in Table 1. Thirty-nine percent of participants met the criteria for MetS and, as expected, females were more likely than males to have MetS ($P<.005$).

Sleep Quality

Forty-nine percent of the sample was classified as reporting disturbed global sleep (PSQI>5). Average sleep duration was 6.5 ± 1.5 hours, and more than half of the participants were classified as short-sleepers (≤6 hours, 54%), followed by regular- (6–9 hours, 38%) and long-sleepers (≥9 hours, 8%). Most participants reported minimal (<once per week) symptomatic snoring (74%) or breathing (81%); 16% reported frequent snoring problems (≥three times per week) and 7% frequent breathing problems. None of these variables (global sleep quality, sleep duration, symptomatic breathing, snoring) differed by sex.

Sleep and Presence of MetS

Logistic regression was used to test the relationship between sleep factors (PSQI global score, snoring, symptomatic breathing, sleep duration) and presence of MetS, controlling for age and sex. Figure 1 depicts the odds ratios of having MetS by different sleep groupings. PSQI sleep quality, symptomatic nighttime breathing, and sleep duration were not associated with MetS. Snoring was the only sleep factor that was associated with MetS ($P<.005$, OR = 2.57, 95% CI: 1.40–4.71). Persons who snored were 2.6 times more likely to have MetS as compared to non-snorers.

Sleep and MetS Components

Global Sleep Quality

Separate multiple regression models (controlling for age and sex) were examined by using the PSQI global score to predict continuous MetS-related variables. The PSQI global score was not associated with any of the MetS variables, which included systolic blood pressure, diastolic blood pressure, mean arterial pressure, glucose, triglycerides, HDL, waist circumference, BMI, and body fat percentage.

Snoring

Group differences by snoring were assessed by analyses of covariance (controlling for age and sex). In order to test for a linear relationship between snoring and MetS, participants were categorized into three groups (no snoring/less than once per week; once to twice per week;

Table 2. Mean values for cardiovascular disease risk factors by snoring group[a]

Variable	Snoring Group		
	Minimal (*n*=182)	Moderate (*n*=22)	High (*n*=41)
Blood pressure (mm Hg)			
Systolic	132 ± 1.2	130 ± 3.3	136 ± 2.5
Diastolic	82 ± 0.9	81 ± 2.6	84 ± 1.9
Mean arterial pressure	173 ± 1.5	170 ± 4.4	178 ± 3.3
Body composition			
Body Mass Index (kg/m^2)[b]	28.9 ± .6	32.8 ± 1.6	33.9 ± 1.2
Body fat (%)[b]	33.3 ± .6	38.0 ± 1.6	37.4 ± 1.2
Blood lipids (mmol/L)			
HDL cholesterol[c]	1.29 ± .03	1.44 ± .08	1.17 ± .06
Triglycerides	1.23 ± .07	0.98 ± .20	1.45 ± .15
Total cholesterol	4.08 ± .07	4.09 ± .20	3.90 ± .15

[a] Mean values adjusted for age and sex. Minimal snoring = none, or less than once per week; moderate = once to twice per week; high = three or more times per week.
[b] $P<.001$.
[c] $P<.05$.

three times or more per week), and significant outcome variables were assessed with polynomial contrasts for linear differences. The results for significance of group differences by snoring were body fat percentage ($P<.001$), BMI ($P<.001$), HDL ($P<.05$; Table 2), fasting glucose ($P<.05$), waist size ($P<.001$; Figure 2); snoring groups did not differ by systolic blood pressure, diastolic blood pressure, mean arterial pressure, total cholesterol ($P=.64$), and triglycerides ($P=.16$). Post-hoc polynomial contrasts revealed that, with the exception of HDL, all of the significant group differences were linear: the means increased with snoring severity. Two of the largest snoring-group differences were found for waist size and fasting glucose; the dose-response-like effects for these two variables are depicted in Figure 2.

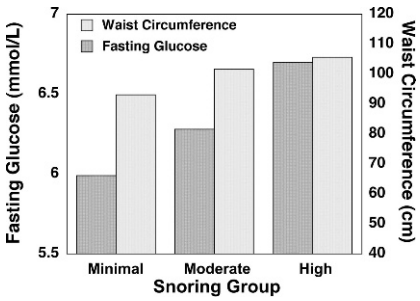

Fig 2. Snoring by fasting glucose and waist circumference. Average fasting glucose and waist circumference by frequency of snoring. Snoring groups consisted of minimal (snores < 1 time/week), moderate (snores 1–2 times/week), and high (snores ≥ 3 times/week)

DISCUSSION

The major findings in our study were that, in a group of AA participants, overall sleep quality, as measured by the PSQI, was not associated with the presence of MetS, or with individual MetS criteria. In contrast, the presence of snoring was associated with selected MetS criteria. These results are based on a large African American community sample.

Several factors may explain the discrepancies between our results among an AA sample and those of Jennings et al[19] with a Caucasian sample. In addition to the racial differences, the sample used by Jennings et al[19] was more restrictive than our sample with regard to age (30–54) and medical history (exclusion criteria were presence of atherosclerosis, cancer, chronic liver/kidney disease; or use medications to treat insulin resistance, weight-loss, or mental disorders). Mean global PSQI sleep quality differed as well, with our mean score (6.6 ± 3.8) being much higher than the mean (4.6 ± 2.6) reported by Jennings et al.[19] In fact, the global PSQI score in our study classified almost half the participants as experiencing poor sleep, which may be indicative of racial differences in sleep quality. Despite these differences, snoring, in our study, and sleep quality, in Jennings,[19] correlated with the same MetS components (fasting glucose, BMI, body fat percent, waist size), with the exception of HDL, which is generally lower among AA than Caucasians.[31] These predictors had analogous roles in their respective studies, and it may be that different aspects of sleep, across different populations, have a similar relationship with MetS.

With regard to sleep symptoms, our results are consistent with two other studies,[21,22] both with multi-racial samples, reporting that snoring, but not overall sleep, correlated with CVD outcomes. A cross-sectional study of 935 women, who were 80% Caucasian, aged 43 to 69 years, and had type 2 diabetes, found that frequent snoring (3–5 times per week), but not sleep duration, was related to poor lipid profiles and abnormal levels of the hormones leptin and adiponectin.[22]

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Additionally, a longitudinal study of 812 participants, 36% AA and without MetS at baseline, reported that snoring, but not sleep quality, predicted whether participants would develop MetS three years post-baseline.[21]

OSA is clearly related to MetS,[11,12,14,16,18] but this alone does not explain the pattern observed in our study, in which sleep quality and disturbed breathing were not related to MetS. Some researchers have recognized that abnormal nighttime breathing, even in the absence of notable impairment, may be a CVD risk factor in addition to OSA.[15,25] Silverberg and Oksenberg,[25] for instance, document that both OSA and sleep-disordered breathing can cause acute and chronic hypertension, and possibly lead to other CVD risk factors. Surveys have likewise demonstrated that snoring is related to BMI,[22,32,33] and a tenuous interpretation of our results is that the relationship between snoring and MetS is mediated through BMI. Both MetS and BMI were derived to represent the health risks of obesity, but BMI is an indirect surrogate of CVD risk, whereas the MetS criteria are actual risk factors.[1] As a measure of obesity, BMI lags behind other measures, such as percent body fat, and its meaning can be ambiguous for non-Caucasians.[6,34,35] Moreover, many studies use BMI because it is easy and can be derived from self-reported height and weight. However, when measures of obesity are available (eg, waist circumference, percent body fat), they should be used.

Additional interpretations are that snoring is a benign correlate of MetS, or that a third variable is related to both snoring and MetS. For instance, anatomical features that obstruct breathing, eg, tonsil size and tongue position, are well-known to cause snoring and OSA, and are more prevalent among AA than Caucasisns.[32] We were unable to identify any research that investigated whether these anatomical features affect CVD health independently of sleep, although such a finding would help clarify the current ambiguity regarding the causal relation between OSA and MetS,[11] along with potentially explaining CVD-related health disparities.

The main limitation of this study is its reliance on self-reported data. Cultural norms with regard to sleep and reporting sleep symptoms may differ by race,[17,32] which would make it difficult to compare studies across race/ethnicity. Although the items on the PSQI are worded objectively, different participants may be more or less likely to recall different behaviors.

The results of our study are applicable to a few different research fields. Epidemiological surveys of health behaviors often only ask about sleep duration or quality in order to conserve space; a snoring question may be more valuable. Physiological research into CVD and health disparities might include Friedman's techniques for measuring tonsil size and tongue position,[32,36] and The Berlin Questionnaire[37] for OSA risk. Sleep studies investigating CVD risk might benefit by focusing more on OSA and breathing, and less on insomnia and sleep deprivation. CVD risk and sleep impairment are often seen as broad and increasing problems. These problems may be connected through a few key pathways, but further research would be required to confirm this.

In summary, MetS is a growing public health issue, particularly among AA. Well-known risk factors for MetS include physical inactivity and a high-calorie diet, and our data suggest that snoring may be a risk factor or possibly a marker for MetS. Future work should investigate anatomic and physiologic correlates of snoring, their relation to MetS, and pertinent differences by race/ethnicity or other demographics.

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Statistical expertise: Kazman
Acquisition of funding: Poth, Deuster
Administrative: Abraham
Supervision: Zeno