Self-reported Sleep Quality is Associated With the Metabolic Syndrome

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Study Objectives: To determine whether a simple, structured self-report of overall sleep quality is associated with the presence of the metabolic syndrome and its component risk factors.

Design: An observational, cross-sectional study comparing global scores on the Pittsburgh Sleep Quality Index with concurrently collected measures of the components of the metabolic syndrome and presence or absence of the syndrome. The metabolic syndrome criterion of the American Heart Association/National Heart, Blood, and Lung Institute was adopted.

Setting: University laboratory.

Patients/Participants: Two hundred ten volunteers with a mean age of 46 years (57% men) screened for the presence of serious illness and related medications.

Interventions: N/A.

Measurements and Results: All analyses were adjusted for sex and age. Logistic regression showed that poor global sleep-quality scores on the Pittsburgh Sleep Quality Index were related significantly to the presence of the metabolic syndrome—an increase of the global sleep score of 2.6 points (approximately 1 SD) was associated with an odds of having the metabolic syndrome of 1.44 (p = .04, confidence interval = 1.01-2.06). Linear-regression results showed that the Pittsburgh Sleep Quality Index global sleep-quality score was related significantly to waist circumference, body mass index, percentage of body fat, serum levels of insulin and glucose, and estimated insulin resistance.

Conclusions: Self-reported global sleep quality is significantly related to the metabolic syndrome and several of its core components.

Keywords: Self-reported global sleep quality, metabolic syndrome, obesity, sleep quality, insulin resistance, Pittsburgh sleep quality index

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INTRODUCTION

OBESITY IS A GROWING EPIDEMIC IN MUCH OF THE WORLD.1,2 AS RECOGNIZED BY REAven AND COLLEAGUES,3 OBESITY TENDS TO CLUSTER WITH OTHER factors to form the “metabolic syndrome” that has been found to predict Type 2 diabetes mellitus, and relate as well to morbidity and mortality from cardiovascular disease, cancer, and arthritis.2,4-6 The metabolic syndrome is defined by a coexistence of several diagnostic factors.7,9 These factors include obesity (particularly abdominal adipose tissue), blood pressure elevation, high fasting serum concentrations of triglycerides and glucose, and low serum high-density lipoprotein cholesterol. Increased insulin levels and insulin resistance are defined by some as part of the metabolic syndrome but are considered by others as an underlying cause for the syndrome.1,3,10,11 When initially identified, the clustering of these risk factors was thought to be induced by dysregulation of insulin.3 However, the pathophysiology basis of the metabolic syndrome remains in doubt;12,13 indeed, some have questioned the clinical value of identifying the syndrome despite the associations between the syndrome and disease incidence.11

Short sleep duration is associated with, and may contribute to, obesity and the metabolic syndrome.14-19 For example, in a large heterogeneous sample of clinic patients, Vorona and colleagues20 found an inverse relationship between body mass index and reported hours of sleep. Experimental evidence suggests that short sleep duration plays a causal role in obesity. In Spiegel and Van Cauter’s pioneering studies,16,21-23 sleep restriction to 4 hours per night for only a single week in young, normal-weight men both increased body weight and induced endocrine and metabolic changes consistent with presence of the metabolic syndrome. Relatedly, patients with obstructive sleep apnea very often have the metabolic syndrome, an observation that may or may not be mediated by obesity or reduced sleep quality.24,25 Despite these associations, not all studies have found sleep disruption and metabolic disease to be related. For example, one recent report failed to find a relationship between sleep disturbance in midlife in women and subsequent incidence of diabetes.26

Given the public-health significance of the metabolic syndrome and the evidence of its relationship with sleep, it is important to know whether normative variation in reported sleep quality is associated with the metabolic syndrome. The Pittsburgh Sleep Quality Index (PSQI) is a widely used measure of sleep quality that is well validated, reliable, and readily completed by patients with obstructive sleep apnea very often have the metabolic syndrome, an observation that may or may not be mediated by obesity or reduced sleep quality.24,25 Despite these associations, not all studies have found sleep disruption and metabolic disease to be related. For example, one recent report failed to find a relationship between sleep disturbance in midlife in women and subsequent incidence of diabetes.26

Given the public-health significance of the metabolic syndrome and the evidence of its relationship with sleep, it is important to know whether normative variation in reported sleep quality is associated with the metabolic syndrome. The Pittsburgh Sleep Quality Index (PSQI) is a widely used measure of sleep quality that is well validated, reliable, and readily completed by most individuals.27,28 The PSQI provides an overall index of sleep quality, includes indexes for a number of components of sleep quality, and is suitable for large-scale epidemiologic investigations. Here we report available data on 210 individuals from a sample of community volunteers who completed the PSQI as part of an investigation of cardiovascular risk12 and its sources.

METHODS

Subjects

Subjects were participants in the University of Pittsburgh’s Adult and Human Behavior Project and were recruited from Allegheny County, Pennsylvania, via mailed brochures. All were community volunteers, 30 to 54 years of age. Exclusion criteria included clinical history of atherosclerotic disease, cancer diag-
nosis or treatment within the past year, chronic liver or kidney disease, as well as use of insulin, weight-loss, or psychotropic medications. The University of Pittsburgh Institutional Review Board approved the protocol, and subjects gave informed consent.

A total of 231 participants completed the PSQI. Fifteen were excluded because of current use of statin medications (which precluded determination of metabolic syndrome criterion). An additional 6 individuals were excluded because of missing fasting blood-sample results. This left data from 210 participants for analysis.

Risk-Factor Assessments

Subjects arrived at the project office between 7:30 am and 10:30 am after a 12-hour overnight fast. After subjects rested in the seated position for at least 10 minutes, a trained staff member obtained 2 blood pressure measurements from the right arm using a mercury sphygmomanometer and a regular, large, or large extra adult cuff, according to the subject’s arm circumference. The 2 readings were averaged. Then, phlebotomy was performed along with measurement of height, weight, waist circumference at the umbilicus, and lean body mass (bioelectrical impedance body composition analyzer, Tanita Corp. of America, Inc., Arlington Heights, IL).

Determinations of standard serum lipids, glucose, and insulin were performed by the Heinz Nutrition Laboratory, University of Pittsburgh Graduate School of Public Health, as previously described. Insulin resistance was estimated from the homeostasis model assessment (HOMA-IR) = [serum insulin (μIU/mL) x fasting blood glucose (mmol/L)]/22.5.30

The metabolic syndrome was defined by the criteria of the American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. The metabolic syndrome is considered to be present in any individual meeting at least 3 or more of the following 5 criteria: blood pressure ≥ 130 mm Hg systolic/85 mm Hg diastolic or taking antihypertensive medication, fasting glucose ≥ 100 mg/dL or taking diabetic medication, waist circumference > 102 cm for men or 88 cm for women, triglycerides ≥ 150 mg/dL or use of dyslipidemic medication, or high-density lipoprotein cholesterol < 40 mg/dL in men and 50 mg/dL in women or use of dyslipidemic medication.

Sleep-Quality Assessment

After consuming a snack, subjects completed several psychometric instruments that included the PSQI. The PSQI has been widely used and has good psychometric properties. The instrument, its reliability, and its validity are presented in Buysse et al.27,28 Nineteen items generate a global sleep-quality score, as well as scores on 7 components of sleep quality: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. A global sleep-quality score of greater than 5 is recommended as defining “poor sleepers.”

Analysis

Because glucose, triglycerides, insulin, and HOMA data were positively skewed, logarithmic transformations were conducted. Preliminary analyses revealed that the PSQI subscales all showed a substantial correlation with the global sleep-quality score (r values = .37-.73, p < .001). For this reason and to protect against experiment-wise error rate inflation, we focused solely on the global sleep-quality score. A logistic-regression analysis was performed using Statistica (6.1, StatSoft, Tulsa, OK), with presence or absence of the metabolic syndrome as the dependent variable and the PSQI global sleep-quality score as the predictor variable, controlling for sex and age. The same predictors were then used in a multiple-regression analysis relating global sleep quality to each of the metabolic-syndrome component risk factors, insulin levels, the HOMA index of insulin resistance, and fat as a percentage of body weight. Follow-up analyses assessed the robustness of the association of the PSQI global score and the metabolic syndrome when controlling for depression, smoking history, alcohol use, years of education, and use of hypertensive medication.

RESULTS

Table 1 describes the characteristics of the sample. The average global sleep-quality score indicated slightly poorer sleep quality than the average of the healthy controls used in developing the PSQI, and 30% of the current sample scored in the poor-sleeper range (global sleep-quality index > 5). Blood pressure and serum indexes were reasonably typical for healthy individuals in this age range. Forty-one (20%) of the participants met the criterion for the metabolic syndrome.

The PSQI global sleep-quality score was associated significantly with the presence of the metabolic syndrome (Table 2). After adjusting for the independent effects of sex and age on the metabolic syndrome, for every increase of 2.6 points on the PSQI, an individual was 1.44 times more likely to meet the criterion for the metabolic syndrome. The strength of association between the PSQI and the metabolic syndrome (p = .02) was essentially the same in a more complex model that added smoking history, alcohol use, and years of education into the logistic model. Due

Table 1—Description of the 210 Subjects Comprising the Sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age, y</th>
<th>Men</th>
<th>Caucasian</th>
<th>PSQI, global score</th>
<th>Blood Pressure, mm Hg</th>
<th>Waist Circumference, cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>45.8 ± 6.0</td>
<td>119 (57)</td>
<td>200 (95)</td>
<td>4.6 ± 2.6</td>
<td>114.3 ± 12.4</td>
<td>90.6 ± 16.0</td>
</tr>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>97.9 ± 22.8</td>
<td></td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>119.8 ± 86.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High density lipoprotein, mg/dL</td>
<td>56.1 ± 16.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.6 ± 5.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat, % of total body weight</td>
<td>27.6 ± 8.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin, μIU/mL</td>
<td>13.2 ± 6.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HOMA</td>
<td>3.3 ± 2.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beck Depression Inventory, score</td>
<td>3.1 ± 3.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presence of metabolic syndrome, no.</td>
<td>41 (20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taking medications, no.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antihypertensive</td>
<td>13 (6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslipidemic</td>
<td>2 (1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral hypoglycemic</td>
<td>4 (2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD or number (%). HOMA refers to homeostasis model assessment, a measure of insulin resistance.
to the possibility that depressed affect impacted reports of sleep quality, a separate model was run that included scores on the Beck Depression Inventory, as well as age and sex as covariates. In this analysis, depression did not contribute significantly to odds for meeting the criterion for the metabolic syndrome, but the statistical strength of the association between the PSQI global sleep-quality score and the metabolic syndrome was decreased to \( p = .12 \).

The relationship between the PSQI global score and individual features of the metabolic syndrome was examined with multiple regressions relating each factor to the PSQI global sleep-quality score, controlling for age and sex. As shown in Table 3, global sleep quality was related significantly to the 3 measures of obesity (body mass index, waist circumference, and fat percentage), and the 3 measures of insulin resistance (fasting insulin concentration, fasting glucose concentration, and estimated insulin resistance). Global sleep quality was unrelated to blood pressure or lipid measures.

**DISCUSSION**

In our analyses of data from middle-aged, generally healthy, Caucasian adults, poor global sleep quality, as measured by the PSQI, was related significantly to the metabolic syndrome. In this sample, the prevalence of the metabolic syndrome was 20%, whereas Ford found a prevalence of 34.5% for the metabolic syndrome among the 3501 men and women older than 20 years of age in the National Health and Nutrition Examination Survey (1999-2002). Therefore, the sample of community volunteers in the current study had somewhat more-favorable average values than national estimates of adult body composition and metabolic indexes. PSQI scores were well within a standard deviation of the control sample used to develop the scale but with a slightly greater mean score for global sleep quality. Thus, the descriptive results suggest that our sample is reasonably typical and that the association between PSQI reports and the metabolic syndrome may generalize to middle-aged, reasonably healthy, Caucasian individuals. Our sample of African Americans was too small to justify generalization to this group and too small to separately analyze. Notably, virtually all reported results were marginally stronger when these participants were excluded from the analyses.

The current report complements other reports on sleep and facets of the metabolic syndrome. Prior epidemiologic reports have related amount of sleep time to obesity, hypertension, and to incident diabetes (in women). Obstructive sleep apnea has also been related to the metabolic syndrome. In the current report, we examined the relationship of sleep quality with the risk factors comprising the metabolic syndrome and also insulin, estimated insulin resistance, body mass index, and percentage of body fat. The PSQI global sleep-quality score correlated positively with those metabolic syndrome components related to adiposity—waist circumference, body mass index, and percentage of body fat. This suggests that sleep quality may influence the metabolic syndrome through its relationship with degree of obesity. Significant relationships between sleep quality and glucose, fasting insulin levels, and insulin resistance were also identified.

The initial conceptualization of the metabolic syndrome postulated insulin regulation as a key underlying factor. Although it is now known that not all individuals with the metabolic syndrome are also insulin resistant, our results relate interestingly to a hypothesis relating sleep restriction to insulin resistance. The human experimental work on sleep restriction and metabolic factors has been interpreted to suggest that inadequate sleep time relates to increased appetite and subsequently to insulin resistance. These experiments were in normal-weight volunteers and, therefore, suggest that sleep time may be an initiating factor influencing body weight and the metabolic syndrome. If our current nonclinical sample suffers from mild disruptions of sleep similar to those induced in the experimental volunteers, then the current results may be consistent with this hypothetical mechanism. Note, however, that the PSQI is not directed at the subjective effects of experimental sleep restriction and that our participants were not all normal weight but, instead, show a substantial range of body mass indexes. Our results may be readily interpreted as suggesting that increased body mass alters both sleep quality and metabolic function.

Our results are limited by a several factors. As noted above, our sample was largely Caucasian and extension to racially diverse groups would be required to establish generalizability to the overall population. The sample is cross-sectional, so we cannot judge the direction of influence between our variables, eg, sleep quality and the metabolic syndrome or body mass and sleep qual-

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**Table 2—Results of Logistic Regression Relating the Metabolic Syndrome to the PSQI Global Sleep Quality Index and Control Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio*</th>
<th>95% CI</th>
<th>Significance*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>1.13</td>
<td>1.0 to 1.22</td>
<td>.002</td>
</tr>
<tr>
<td>Sex</td>
<td>2.19</td>
<td>1.48 to 3.36</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PSQI global sleep quality</td>
<td>1.44</td>
<td>1.01 to 2.06</td>
<td>.04</td>
</tr>
</tbody>
</table>

CI refers to confidence interval.

*Odds ratio is the difference in odds of meeting the criterion for metabolic syndrome as associated with a variation of 1 SD in the Pittsburgh Sleep Quality Index (PSQI) (SD = 2.6) or with 1 year of age and male sex more likely to exhibit the metabolic syndrome.

*Wald statistic.

**Table 3—Multiple Regression Results’ Relating Aspects of the Metabolic Syndrome to the Global Pittsburgh Sleep-Quality Index in 210 Subjects**

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>( \beta )</th>
<th>SE</th>
<th>t</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>.04</td>
<td>.06</td>
<td>.67</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>.07</td>
<td>.06</td>
<td>1.08</td>
<td>NS</td>
</tr>
<tr>
<td>Glucose</td>
<td>.14</td>
<td>.07</td>
<td>2.13</td>
<td>.03</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>.04</td>
<td>.07</td>
<td>.66</td>
<td>NS</td>
</tr>
<tr>
<td>HDL</td>
<td>-.09</td>
<td>.06</td>
<td>-1.48</td>
<td>NS</td>
</tr>
<tr>
<td>BMI</td>
<td>.19</td>
<td>.07</td>
<td>2.88</td>
<td>.004</td>
</tr>
<tr>
<td>Fat percentage</td>
<td>.19</td>
<td>.06</td>
<td>3.24</td>
<td>.001</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>.12</td>
<td>.06</td>
<td>2.11</td>
<td>.04</td>
</tr>
<tr>
<td>Insulin</td>
<td>.14</td>
<td>.07</td>
<td>2.07</td>
<td>.04</td>
</tr>
<tr>
<td>Insulin resistanceb</td>
<td>.17</td>
<td>.07</td>
<td>2.54</td>
<td>.01</td>
</tr>
</tbody>
</table>

PSQI refers to Pittsburgh Sleep Quality Index; HDL, high-density lipoprotein; BMI, body mass index. See Table 1 for measurement units.

*Controlled for sex and age.

bEstimated from the homeostasis model of assessment, a measure of insulin resistance.
Self-reports of sleep are valuable but do not provide the direct measures of sleep that electroencephalographic studies provide. Self-reports have value as a measure of sleep in usual circumstances and as an overall index rather than an index solely for particular nights using polysomnography. Self-reports are, however, subject to recall biases and do not provide measures of sleep architecture. We cannot determine the precise basis for the reports of poor sleep quality without physiologic studies.

For example, obstructive sleep apnea is associated with the metabolic syndrome. An important issue is whether the current result predominantly reflects a relationship between sleep apnea and the metabolic syndrome. Three observations question this interpretation. First, based on available statistics from the general population in the appropriate age range, 6% of the participants could be expected to be diagnosed with obstructive sleep apnea. This prevalence compares to a prevalence of 20% for the metabolic syndrome in our sample. Thus, in the unlikely case that all participants with sleep apnea exhibited the metabolic syndrome, sleep apnea would not predict all cases of the metabolic syndrome. Second, Coughlin et al found, in regression analyses, that obstructive sleep apnea is significantly or marginally related to the following components of the metabolic syndrome: systolic and diastolic blood pressure, insulin level, HOMA, and triglyceride and high-density lipoprotein cholesterol levels. The PSQI results shared with sleep apnea only an association with the insulin and HOMA values. In contrast to the relationship between sleep apnea and components of the metabolic syndrome, PSQI global sleep quality also showed significant relationships with body mass index, waist circumference, fat percentage, and glucose. Finally, the PSQI global sleep-quality index is not closely associated with obstructive sleep apnea. In a polygraphic study of 435 patients, 58% of whom had sleep apnea, the PSQI global sleep index failed to significantly distinguish between those with and without apnea.

In a smaller sample of survivors of sexual assault (n = 151) clinically diagnosed sleep-disordered breathing was correlated with PSQI global sleep quality. In short, the relative prevalence of obstructive sleep apnea, the pattern of its relationship to the metabolic syndrome, and the weak relationships between sleep apnea and the PSQI in previous reports suggest that obstructive sleep apnea is only likely to explain a small portion of the relationship that we have observed between global sleep quality and the metabolic syndrome. Nonetheless, polysomnography studies that identify sleep apnea and relate it to both self-reported sleep and the metabolic syndrome are necessary to evaluate our inferences from the literature and gain further insight into the relationship of physiologically assessed sleep and the metabolic syndrome.

In summary, the current investigation found that poor overall sleep quality is associated with increasing prevalence of the metabolic syndrome and several of its component risk factors. The practical significance of our finding is that a simple self-report instrument on sleep quality can alert individuals and their physicians to additional possible risks for the metabolic syndrome or, more generally, a set of risk factors for cardiovascular disease and diabetes. Future studies should characterize general-population samples with respect to specific types of sleep disturbances (particularly, obstructive sleep apnea) and the relationships of each to the metabolic syndrome and each to self-reports of sleep quality.

ACKNOWLEDGMENTS

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