Title

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SCIENTIFIC INVESTIGATIONS

African American race is associated with worse sleep quality in heavy smokers

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Study Objectives: To examine the association of self-identified race with sleep quality in heavy smokers.

Methods: We studied baseline data from 1965 non-Hispanic White and 462 African American participants from SPIROMICS with ≥ 20 pack-years smoking history. We first examined the Pittsburgh Sleep Quality Index's (PSQI) internal consistency and item-total correlation in a population with chronic obstructive pulmonary disease. We then used staged multivariable regression to investigate the association of race and sleep quality as measured by the PSQI) The first model included demographics, the second added measures of health status, and the third, indicators of socioeconomic status. We next explored the correlation between sleep quality with 6-minute walk distance and St. George's Respiratory Questionnaire score as chronic obstructive pulmonary disease-relevant outcomes. We tested for interactions between self-identified race and the most important determinants of sleep quality in our conceptual model.

Results: We found that the PSQI had good internal consistency and item-total correlation in our study population of heavy smokers with and without chronic obstructive pulmonary disease. African American race was associated with increased PSQI in univariable analysis and after adjustment for demographics, health status, and socioenvironmental exposures (P = .02; 0.44 95%CI: .06 to .83). Increased PSQI was associated with higher postbronchodilator forced expiratory volume in 1 second and lower household income, higher depressive symptoms, and female sex. We identified an interaction wherein depressive symptoms had a greater impact on PSQI score for non-Hispanic White than African American participants (P for interaction = .01).

Conclusions: In heavy smokers, self-reported African American race is independently associated with worse sleep quality.

Clinical Trial Registration: Registry: ClinicalTrials.gov; Name: Study of COPD Subgroups and Biomarkers (SPIROMICS); URL: https://clinicaltrials.gov/ct2/show/NCT01969344; Identifier: NCT01969344.

Keywords: sleep, socioeconomic status, SES, health disparities, COPD, validation, PSQI

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BRIEF SUMMARY

Current Knowledge/Study Rationale: African Americans in the general population have worse sleep quality. Lung disease changes sleep physiology with unknown impact on this disparity.

Study Impact: Among heavy smokers, worse sleep quality appeared to have unique drivers among African American participants and was associated with African American race even after adjustment for demographics, health status, and socio-environmental exposures. This study extends the psychometric characterization of the PSQI in the tobacco-exposed populations.

INTRODUCTION

Sleep disturbance is an important symptomatic dimension in the assessment of chronic obstructive pulmonary disease (COPD) severity.[1] Airway resistance, hypercapnia, dyspnea, and hypoxia are may all influence a patient's ability to sleep as one aspect of their perceived symptoms. Recent discoveries highlight that these problems are common among all smokers.[2] Similar to the disparities observed with smoking-related respiratory symptoms between non-Hispanic White and African American groups,[3,4] striking racial disparities are associated with sleep-related outcomes in the general population.[5] However, potential racial differences in sleep outcomes have not been well studied in this population. Given the importance of sleep assessment in treatment of smoking-related lung disease,[6,7] the presence or absence of any sleep disparities could contribute to our understanding of this disease's divergent outcomes.

The factors that contribute to disparities in sleep quality in patients with smoking-related respiratory symptomology also warrant further investigation. Sleep quality is profoundly influenced

by socioeconomic factors: in the general population, low household income, lower educational attainment, and high perceived stress are important predictors of poor sleep.[8–10] They are also associated with high symptom burden in COPD. Similarly, while long understood as important in sleep, mood symptoms have recently been identified as better predictors of patient-reported outcomes than even airflow obstruction.[11] Modeling the influence of sleep quality on COPD-related outcomes therefore requires consideration of these elements. In the present study we aim to (1) examine the association between race and poor sleep quality, (2) understand the relationship of poor sleep quality to mood and environment, and (3) explore the impact of poor sleep quality in heavy smokers. Some of the results of these studies have been previously reported in the form of an abstract.[12]

METHODS

This is a secondary analysis of the baseline data collected from the SubPopulations and Intermediate Outcome Measures in COPD Study (SPIROMICS), a multicenter prospective cohort studying symptomatic smokers and individuals with COPD. Its complete design, aims, and procedures are described elsewhere.[13] For the present study, we included all ever-smokers (≥ 20 pack-years) whom self-identified as either solely African American or non-Hispanic White race, as even those without formal diagnosis can carry a significant symptom burden and experience exacerbations.[2] Given this broad overlap, we take use evidence and outcomes defined in COPD as representative for both groups.

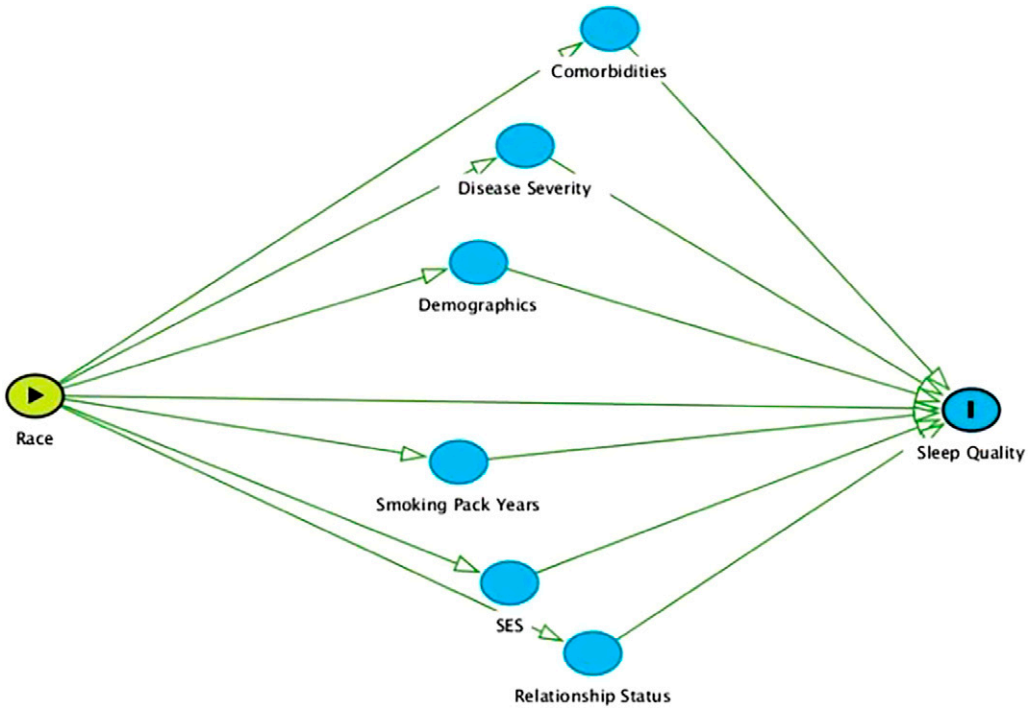
Pittsburgh Sleep Quality Index

In our primary analysis, the outcome of interest was sleep quality as assessed by the Pittsburgh Sleep Quality Index (PSQI).[14] The PSQI is a survey instrument with a total of 19 self-report items ranked on a Likert scale. These are used to generate seven "component" scores, each on a 0 to 3 scale, and summed into a global score, which has a range of 0 to 21, with greater scores indicating worse sleep quality.[14] In this secondary analysis, we examined associations between PSQI score and respiratory outcomes. To understand functional outcomes, we included 6-minute walk distance, measured as a percent of predicted total.[15] To measure quality of life, we used St. George's Respiratory Questionnaire (SGRQ), which has a consensus minimal clinically important difference of 4.[16] In these outcomes-oriented tests, we used the same covariates as the final model of our primary analysis.

Mood symptoms

Mood disorders have an important relationship to sleep. In this work, we modeled mental symptoms as primarily driving sleep disturbance, rather than the reverse causal relationship.[17,18] Supporting this approach, sleep disturbance is commonly recognized as the most common residual symptom of depression.[19,20] To measure depressive symptoms with minimal confounding for their underlying chronic illness, we used the Hospital Anxiety and Depression Scale, which was psychometrically designed for use in populations with chronic illeness.[21] Given their centrality to sleep and differentially distribution by race in the general public,[22] we tested an interaction between race and Hospital Depression Scale score.

Figure 1—Directed acyclic graph conceptualizing the relationship between self-identified race and subjective sleep quality as measured by the Pittsburgh Sleep Quality Index.



Socioenvironmental exposures

We used multiple metrics to assess the impact of socioenvironmental exposures. To capture the impact of neighborhood conditions, we used the Area Deprivation Index, a previously validated metric with correlation to worse sleep- and COPD-relevant outcomes.[23,24] To measure individual and historic exposures, we used the Adversity-Opportunity Index, a composite metric that equally weighted household income, educational attainment, history of occupation with significant respiratory exposure, history of *in utero* smoke exposure, and access to fresh healthy food.[25] Each category was scored 0 to 2, with higher scores representing greater privilege while intermediate or uncertain responses were scored as 1. Finally, marital status was included for its importance in both sleep and respiratory outcomes.[26,27]

Additional covariates

Informed by prior literature, we developed a directed acyclic graph to explore the relationship between self-identified race and sleep quality (**Figure 1**). All variables were treated as mediators of the relationship of interest. Reasoning that in a perfectly equitable system, the residual direct effect of race on sleep quality should be zero, we adjusted for all mediators to account for indirect pathways. We highlighted socioenvironmental exposures and mood symptoms as especially important to both COPD as a disease process and self-rated sleep quality as our primary outcome. These two mediators were tested for evidence of effect modification.

As recommended by current Global Initiative for Chronic Obstructive Lung Disease guidelines, we assessed COPD severity according to both volume of air expired after bronchodilation during the first second of a forced expiratory maneuver (forced expiratory volume in 1 second (FEV_1)) and self-reported symptom burden.[28] For the latter, we opted for the modified Medical Research Council dyspnea scale to avoid collinearity with the COPD Assessment Test's assessment of sleep quality.[1] We also included use of long-acting muscarinic or inhaled corticosteroid/beta-2 agonist medications to assess disease control. To understand the cumulative impact of medical comorbidity in COPD, we used the previously validated simple comorbidity count.[29] This includes multiple potentially important diagnoses including obstructive sleep apnea, gastro-esophageal reflux disease, and allergic rhinitis.[29] Finally, we assessed age, sex, and body mass index as demographic covariates.

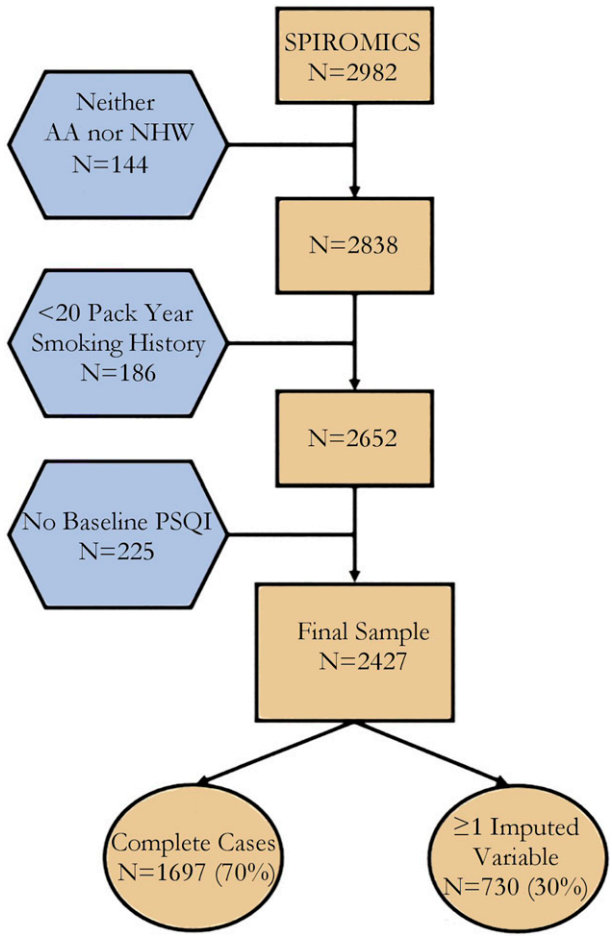
Analysis

First, we undertook psychometric characterization of the PSQI. It is widely used in COPD research, but its properties have never been formally explored in this population. We measured internal consistency by computing Cronbach's alpha from the subscales. We additionally calculated item-total correlations. We also sought to explore the underlying theoretical model for the PSQI. Factor analysis is a statistical technique that allows reducing conceptually interrelated measured variables to a smaller number of underlying "factors."[30] In our case, we used factor analysis to test Buysse's original hypothesis that the seven separate subscales of the PSQI actually describe a single underlying factor or phenomenon of sleep quality. We also employed dominance analysis to understand the relative importance the PSQI's subscores in our population of interest.[31]

To prevent introducing bias in our analyses due to missingness in the dataset, we performed imputation. We elected multiple imputation by chained equations as most appropriate given our judgment that missingness occurred at random. Consistent with prior literature, we generated 10 imputed datasets, with 10 cycles in each imputation. We imputed only covariate data. The results from using the imputed datasets were combined using the "mi estimate" package from Stata, which calculates point estimates and confidence intervals according to Rubin's Rules.[32]

In our primary analysis, we performed staged multiple linear regression to explore the association between PSQI score and self-identified race. Before beginning this analysis, we examined baseline differences by race using Student's *t* test for continuous variables and Pearson's Chi-square test for categorical variables. In Model 1, we added age, body mass index, and biological sex to the unadjusted association. In Model 2, we added metrics of COPD severity, comorbidity count, pack-years

Figure 2—Consolidated Standards for Reporting of Trials diagram showing how the final sample size of this study was derived from the total SPIROMICS cohort.



smoking history, and use of respiratory medications and used the Hospital Anxiety and Depression Scale to characterize symptoms of depression and anxiety. In our final model, we included Area Deprivation Index, Adversity-Opportunity Index, and marital status as metrics of socioenvironmental condition. We tested for an interaction between self-identified race and Global Initiative for Chronic Obstructive Lung Disease diagnosis of COPD as a sensitivity test. As a secondary analysis, we used multiple linear regression to see whether sleep quality was independently associated with either St. George's Respiratory Questionnaire as a patient-reported outcome or with 6-minute walk distance as a functional outcome in participants with a history of heavy smoking.

We performed all data analyses in Stata 16 SE using the Core Clinical Data version 6.0 release of the SPIROMICS dataset.[33] We performed all statistical tests as two-sided and calculated 95% confidence intervals. To account for the multiple tests performed in this work, we used the false discovery rate approach for identifying statistically significant results. Utilizing the Benjamini-Hochberg procedure and a preset false discovery rate of 5%, we calculated a critical value of $P = .03$ as our threshold for significance, as it was the largest P value smaller than its accompanying critical value.

RESULTS

We had complete data from 1697 SPIROMICS participants, constituting 70% of the eligible dataset (**Figure 2**). The largest single source of missingness was the composite comorbidity count variable, which was missing for 11.5% of the total dataset. The majority of participants with incomplete records were missing data for only a single variable, and most patterns of missingness constituted < 1% of the total dataset. In comparison to complete cases, those with incomplete records were not of significantly different age, FEV_1, PSQI, sex, or Adversity-Opportunity Index (**Table S1** in the supplemental material).

The estimated association between self-identified race and PSQI total score ($P < .001$) was unchanged when comparing all cases vs those with complete data only (**Table 1** and **Table S2**). However, among participants with incomplete records there were a greater proportion of African Americans ($P = .01$) and higher mean scores on the Hospital Anxiety ($P = .03$) and Depression ($P = .02$) Scale questionnaires (**Table S1**). In comparison with non-Hispanic White participants, African Americans in our analysis were on average younger, had a higher PSQI, were more likely single, less likely male, and of lower privilege, from worse neighborhoods, but with higher FEV_1 (**Table 2**; **Figure 3**). A similar pattern was observed when comparing participants according to Buysse's recommended threshold of PSQ1 > 5 for "poor" sleep (**Table S3**).

Psychometric analysis of the PSQI among heavy smokers

Internal consistency was measured by Cronbach's alpha at 0.73, above the standard cut-off of 0.70 (**Table 3**). We used Spearman's rank-order correlation to compare results by individual subscales to the total PSQI score. We found that correlation coefficients varied from 0.51 to 0.73 (**Table 3**). Contrary to Buysse's hypothesis,[14] the PSQI did not load well onto a single factor in this population (**Figure 4**). Factor-loading varied between races; sleep efficiency in particular was moderately less important among African Americans as compared to non-Hispanic White participants (**Figure 4**). In dominance analysis, sleep duration was the most important subscale for African American participants but was fifth most important for non-Hispanic White participants as a driver of the total PSQI score (**Figure 4**).

Contributors to PSQI for heavy smokers

In the unadjusted model, self-reported African American race was significantly associated with higher PSQI score (mean difference 1.14, 95%CI: 0.74 to 1.53, $P < .001$). This relationship was attenuated but persistent in our final model after adjustment for demographics, COPD severity and control, comorbid disease, and environmental exposures (0.44, 95%CI: 0.06 to 0.83, $P = .02$). In our final model, female sex, increasing postbronchodilator FEV_1, modified Medical Research Council dyspnea score, Hospital Anxiety and Depression Scales scores, and comorbidity count were associated with higher PSQI score, while nonsingle relationship status was associated with lower PSQI (**Table 1**). Results from complete case analysis were similar (**Table S2**).

We identified an interaction between self-reported African American race and Hospital Depression Scale score (−0.13, 95%CI: −0.23 to −0.03, P for interaction = .01). This revealed that while depressive symptoms had a strong association with PSQI score in both races, among African Americans this made a much smaller contribution to PSQI score than was seen in non-Hispanic Whites. An exploratory, racially stratified analysis suggests there are several determinants of PSQI for which this may be true (**Table 4**). We did not identify evidence for effect modification when testing hypotheses about the relationship between self-identified race and diagnosis of obstructive sleep apnea ($P = .57$), Adversity-Opportunity Index ($P = .28$), or Area Deprivation Index ($P = .85$). Sensitivity testing also suggested that the observed relationship between self-identified race and PSQI score was similar regardless of the absence or presence of formal Global Initiative for Chronic Obstructive Lung Disease diagnosis of COPD ($P = .11$).

Impact of PSQI score on COPD outcomes

In univariable linear regression models, the PSQI was associated with both SGRQ score and percent of predicted 6-minute walk distance (Ps < .001). After adjustment using the multivariable model of regression, the SGRQ score remained significantly associated (0.81, 95%CI: 0.67 to 0.95) but performance on the 6-minute walk test was not (0.05%, 95%CI: −0.21% to −0.31%). Scores on the PSQI would have to change by 5 points to cross the threshold of minimal clinically important difference for the SGRQ.

DISCUSSION

Using SPIROMICS, a longitudinal multicenter cohort of heavy smokers with and without COPD, we explored the association

Table 1—Mean contribution to Pittsburgh Sleep Quality Index among ever-smoker SPIROMICS participants by imputation.

	Unadjusted	*P*	Model 1	*P*	Model 2	*P*	Model 3	*P*	Model 3b	*P*
African American race	**1.14 (0.74 to 1.54)**	**<.001**	**0.47 (0.06 to 0.87)**	**.025**	**0.68 (0.31 to 1.04)**	**<.001**	**0.45 (0.06 to 0.83)**	**.02**	**1.02 (0.43 to 1.61)**	**.001**
Age	–		**−.79 (−0.95 to −0.63)**	**<.001**	**−0.39 (−0.54 to −0.24)**	**<.001**	**−0.33 (−0.49 to −0.18**	**<.001**	**−0.32 (−0.47 to −0.16)**	**<.001**
Female sex	–		**0.92 (0.62 to 1.23)**	**<.001**	**0.47 (0.19 to 0.75)**	**.001**	**0.44 (0.16 to 0.72)**	**.002**	**0.44 (0.16 to 0.72)**	**.002**
BMI	–		0.13 (−.03 to 0.28)	.11	−0.10 (−0.26 to 0.05)	0.21	−0.09 (−0.05 to 0.27)	0.26	−0.02 (−0.05 to 0.02)	0.25
FEV_1 percent predicted	–		–		**.27 (0.09 to 0.44)**	**.003**	**0.27 (0.09 to 0.45)**	**.003**	**0.27 (0.09 to 0.45)**	.003
Comorbidity count	–		–		**0.32 (0.15 to 0.48)**	**<.001**	**0.31 (0.09 to 0.31)**	**<.001**	**0.31 (0.14 to 0.47)**	**<.001**
Smoking pack years	–		–		0.02 (−0.10 to 0.14)	0.72	0.01 (−0.12 to 0.13)	0.90	0 (−0.12 to 0.13)	0.96
Asthma	–		–							
No	–		–		Ref		Ref		Ref	
Yes	–		–		0.21 (−0.14 to 0.57)	0.24	0.21 (−0.14 to 0.57)	0.22	0.20 (−0.16 to 0.56)	0.17
Uncertain	–		–		0.39 (−0.35 to 1.14)	0.31	0.37 (−0.37 to 1.10)	0.33	0.39 (0.25 to 1.12)	0.31
Medical Research Council dyspnea score	–		–							
0	–		–		Ref		Ref		Ref	
1	–		–		**0.69 (0.35 to 1.03)**	**<.001**	**0.67 (0.33 to 1.01)**	**<.001**	**0.67 (0.33 to 1.01)**	**<.001**
2	–		–		**1.16 (0.67 to 1.64)**	**<.001**	**1.11 (0.62 to 1.60)**	**<.001**	**1.10 (0.61 to 1.59)**	**<.001**
3	–		–		**1.09 (0.49 to 1.68)**	**<.001**	**1.01 (0.42 to 1.61)**	**.001**	**0.98 (0.39 to 1.57)**	**.001**
4	–		–		**1.33 (0.33 to 2.32)**	**.009**	**1.26 (0.27 to 2.26)**	**.01**	**1.35 (0.36 to 2.35)**	**.01**
Hospital Depression Scale	–		–		**0.26 (0.21 to 0.31)**	**<.001**	**0.25 (0.19 to 0.30)**	**<.001**	**0.27 (0.22 to 0.31)**	**<.001**
Hospital Anxiety Scale	–		–		**0.26 (0.22 to 0.31)**	<.001	**0.26 (0.22 to 0.31)**	**<.001**	**0.26 (0.22 to 0.31)**	<.001
LAMA	–		–		0.15 (−0.20 to 0.50)	0.40	0.17 (0.19 to 0.53)	0.36	0.17 (0.19 to 0.53)	0.40
LABA ICS	–		–		−0.04 (−0.38 to 0.30)	0.81	−0.05 (−.40 to 0.29)	0.80	−0.04 (−0.39 to 0.30)	0.83
Nonsingle relationship status	–		–		–		**−0.34 (−0.63 to –0.05)**	**.02**	**−0.33 (−0.62 to −0.04)**	**.02**
Adversity-Opportunity Index	–		–		–		−0.07 (−0.15 to 0.0)	.048	−0.07 (−0.14 to 0.01)	.05
Area Deprivation Index	–		–		–		0.12 (−0.03 to 0.27)	0.12	0.12 (−0.03 to 0.027)	0.12
Race × depression interaction	–		–		–		–		**−0.13 (−0.23 to −0.02)**	**.01**

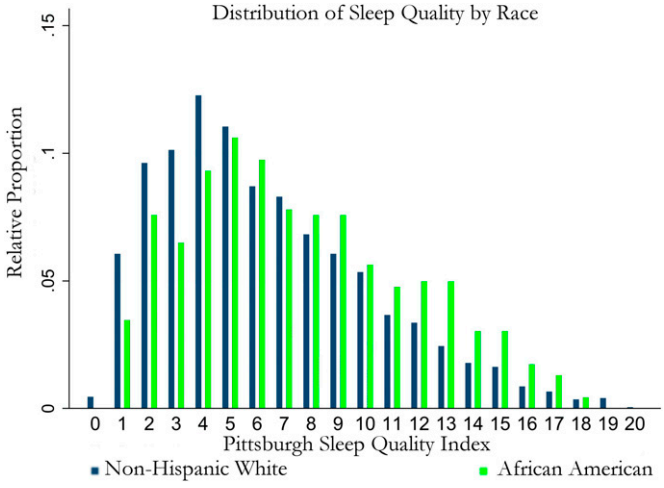
Self-report survey instruments reported per 1-point change; all other continuous variables per 1 standard deviation. Bolded values significant at threshold from Benjamini-Hochberg procedure. Model 1: demographics. Model 2: model 1 + COPD, severity, treatment, and comorbidities. Model 3: model 2+ social and socioenvironmental exposures. Model 3b: model 3 + race × depression interaction. BMI = body mass index, FEV_1 = forced expiratory volume in 1 second, LABA-ICS = Long-acting beta-2 agonist with inhaled corticosteroid, LAMA = Long-acting muscarinic antagonist.

Table 2—Baseline characteristic of SPIROMICS participants by race.

		Non-Hispanic White	African American	*P*
n (%)		1,965 (81%)	462 (19%)	
Age		**65.0 ± 8.4**	**58.1 ± 8.7**	**<.001**
Male		**1,083 (55.1%)**	**227 (49.1%)**	**.02**
Obstructive sleep apnea		**345 (18.7%)**	**61 (14.1%)**	**.02**
Hospital Anxiety Score		5.5 ± 3.8	5.8 ± 4.0	.66
Hospital Depression Score		4.4 ± 3.5	4.4 ± 3.4	.18
Postbronchodilator percent predicted FEV_1		**72.2 ± 26.1**	**76.5 ± 27.2**	**.002**
Modified Medical Research Council dyspnea score	0	281 (23.6%)	39 (18.9%)	.08
	1	538 (45.1%)	91 (44.1%)	
	2	224 (18.8%)	40 (19.4%)	
	3	117 (9.8%)	24 (11.6%)	
	4	32 (2.7%)	12 (5.8%)	
Asthma diagnosis	Yes	**327 (17.0%)**	**145 (32.0%)**	**<.001**
	No	**1,526 (79.2%)**	**293 (64.7%)**	
	Unsure	**73 (3.8%)**	**15 (3.3%)**	
LAMA usage		539 (28.1%)	125 (27.7%)	.87
LABA-ICS usage		606 (31.6%)	151 (33.6%)	.43
In relationship		**1,068 (54.7%)**	**103 (23.7%)**	**<.001**
Comorbidity count		**2.4 ± 1.6**	**2.0 ± 1.5**	**<.001**
Adversity-Opportunity Index		**5.5 ± 2.0**	**4.6 ± 1.8**	**<.001**
Area Deprivation Index		**4.4 ± 2.8**	**7.1 ± 2.8**	**<.001**
Pittsburgh Sleep Quality Index		**6.3 ± 3.9**	**7.5 ± 4.1**	**<.001**

All values in n (%) or mean ± SD. Self-report survey instruments reported per 1-point change; all other continuous variables per 1 standard deviation. Bolded values significant at threshold from Benjamini-Hochberg procedure. FEV_1 = forced expiratory volume in 1 second, LABA-ICS = Long-acting beta-2 agonist with inhaled corticosteroid, LAMA = Long-acting muscarinic antagonist.

Figure 3—Histogram of Pittsburgh Sleep Quality Index scores in our study sample of SPIROMICS participants, divided by self-identified race.



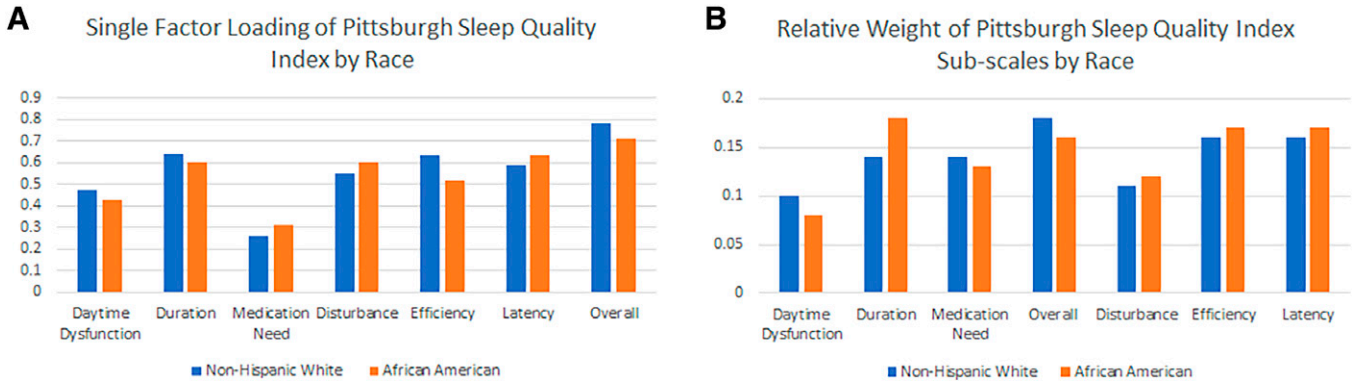
Higher markings on the y-axis represent greater frequency of that score.

Table 3—Item-total correlation and internal consistency of the Pittsburgh Sleep Quality Index among participants with spirometrically confirmed COPD in SPIROMICS.

Subscale	Spearman Correlation Coefficient Rho
Sleep duration	0.60
Sleep disturbance	0.60
Sleep latency	0.69
Daytime dysfunction	0.52
Sleep efficiency	0.63
Overall sleep quality	0.73
Medications to sleep	0.52
Total Score	Cronbach's alpha: 0.73

COPD = chronic obstructive pulmonary disease.

Figure 4—Subscales of Pittsburgh Sleep Quality Index.



(A) The subscale loading in a one-factor model of the Pittsburgh Sleep Quality Index is presented in bar graph form, stratified by race. **(B)** The relative dominance of the seven subscales of the Pittsburgh Sleep Quality Index are presented in bar graph form, stratified by race.

between self-reported race and sleep quality as measured by the Pittsburgh Sleep Quality Index. We found that African American race was positively associated with PSQI when controlling for demographics, disease severity and medical comorbidities, and environmental exposures. We identified an interaction wherein depressive symptoms were more impactful on PSQI score for non-Hispanic White than African American participants. In a limited psychometric evaluation of the PSQI, we identified good item-total correlation and internal consistency.

Table 4—Mean contribution to Pittsburgh Sleep Quality Index among ever-smoker SPIROMICS participants, by race.

	Non-Hispanic White	*P*	African American	*P*
Age	**−0.04 (−0.06 to −0.02)**	**<.001**	−0.02 (−0.06 to 0.03)	.50
Female sex	**0.36 (0.05 to 0.66)**	**.02**	**0.83 (0.09 to 1.56)**	**.03**
BMI	−0.02 (−0.05 to 0.01)	.22	0 (−0.07 to 0.07)	.99
FEV_1 percent predicted	0.02 (0.01 to 0.03)	.05	**0.02 (0.01 to 0.04)**	**.01**
Comorbidity count	**0.18 (0.06 to 0.29)**	**.002**	0.22 (−0.07 to 0.51)	.14
Smoking pack years	0 (−0.01 to 0.00)	.99	0 (−0.02 to 0.02)	.72
Asthma				
No	Ref		Ref	
Yes	0.40 (0.0 to 0.81)	.049	−0.23 (−1.01 to 0.55)	.56
Uncertain	0.28 (−0.51 to 1.07)	.49	0.56 (−1.44 to 2.56)	.59
Medical Research Council dyspnea score				
0	Ref		Ref	
1	**0.67 (0.31 to 1.04)**	**<.001**	0.57 (−0.30 to 1.45)	.20
2	**1.08 (0.55 to 1.62)**	**<.001**	1.01 (−0.16 to 2.18)	.09
3	0.70 (0.03 to 1.37)	.04	**1.82 (0.47 to 3.17)**	**.01**
4	**1.80 (0.65 to 2.94)**	**.002**	0.14 (−1.94 to 2.22)	.89
Hospital Depression Scale	**0.28 (0.22 to 0.33)**	**<.001**	**0.15 (0.03 to 0.28)**	**.02**
Hospital Anxiety Scale	**0.26 (0.21 to 0.31)**	**<.001**	**0.28 (0.18 to 0.39)**	**<.001**
LAMA	−0.03 (−0.40 to 0.35)	.89	0.96 (0.03 to 1.89)	.04
LABA-ICS	0.04 (−0.33 to 0.41)	.83	−0.39 (−1.26 to 0.48)	.37
Relationship status	−0.32 (−0.63 to −0.02)	.04	−0.41 (−1.23 to 0.40)	.32
Adversity-Opportunity Index	−0.04 (−0.12 to 0.03)	.27	**−0.23 (−0.43 to −0.03)**	**.02**
Area Deprivation Index	0.04 (−0.01 to 0.09)	.15	0.04 (−0.08 to 0.18)	.49

Self-report survey instruments reported per 1-point change; all other continuous variables per 1 standard deviation. Bolded values significant at threshold from Benjamini-Hochberg procedure. BMI = body mass index, FEV_1 = forced expiratory volume in 1 second, LABA-ICS = Long-acting beta-2 agonist with inhaled corticosteroid, LAMA = Long-acting muscarinic antagonist.

We found that the association between African American race and PSQI was attenuated when also adjusting for individual and neighborhood socioeconomic factors. These findings are consistent with the reported importance of socioeconomic factors in sleep quality in the general population. Using a community sample, Patel identified that among poor African Americans but not poor Non-Hispanic Whites, adjustment for educational attainment and employment significantly diminished previously significant associations to poor sleep quality.[8] Similarly, the Study of Woman's Health Across the Nation found that the combination of socioeconomic status and health condition mediated 79% of the differences in perceived sleep quality between African American and non-Hispanic White participants in that study.[32] By contrast, neither in CARDIA nor in a later Chicago-area analysis by Carnethon did these considerations appear to significantly attenuate the disparity.[34,35] To our view, the underlying implications of these varying effect sizes for socioeconomic status in racial sleep differences are unlikely different. Existing social disparities are not randomly distributed but heavily influenced by discriminatory policymaking, the effects of which are likely difficult to account for comprehensively.[36] Although related, measures of socioeconomic inequity and racial discrimination are not monotonically correlated in all communities, leading to variable estimates of importance when measured across multiple studies.

Our finding of lesser consequences among African American participants despite a similar level of depressive symptomology echoes a known paradox in psychiatric literature.[37] One theory about this phenomenon states that it is simply the result of sampling error because it is hard to access many populations that are both high risk for depression and disproportionately African American. However, even studies whose methodologies account for this shortcoming have a persistent paradoxical finding.[38] Other authors have instead suggested that African Americans may manifest their depression through different symptoms or may be interpreted differently by mental health professionals. For instance, it was once proposed that they expressed relatively more somatic complaints like appetite changes as opposed to complaints about their thought content.[39] However, beyond the incompleteness of the aforementioned explanations there is also direct, meaningful evidence that this phenomenon might represent substantive differences between the two populations. One model of geriatric depression noted greater resilience was associated with both chronic disease and non-White race.[40] Our work similarly tested for a concept that is important in understanding depression and likewise found racial differences that could support an actual underlying paradox. One reason for these results could be different coping mechanisms preferred by each group.[41] Previous findings have suggested that, relative to non-Hispanic Whites, African Americans may rely to a greater extent on kinship networks[42] or formal social structures like religious organizations.[43] As an active area of inquiry, the most salient takeaway is the extent to which lived conditions vary by race and inform disease presentation.

We appreciated a similar pattern of racially specific characteristics in our analysis of the PSQI. The range of observed item-total correlation was similar to those originally reported by Buysse in his depressed cohort.[14] This suggests a certain consistency for this instrument when applied to individuals with diseases known to worsen sleep quality. Similarly, support for multiple over single factor structures for the PSQI has been extensively reported.[44] Previous reports from the SPIROMICS cohort reported a favorable interclass correlation coefficient of 0.85 on repeat testing with the PSQI.[45] Overall qualities thus appear generally favorable insofar as they have been tested. Our unique findings relate to the extent to which both subscales, their relative weights, and determinants of PSQI score varied between races. This is a concrete demonstration of previous suggestions about the extent to which nonbiological differences may affect sleep quality. Consistent with the social determinants of health framework, consequences of lived experience were not isolated but were instead associated with health consequences worthy of clinical attention.

Our finding of a positive association between PSQI score and quality of life as measured by the SGRQ highlights the importance of this metric in COPD. This confirms previous reports from this cohort.[46] We did not find an association with 6-minute walk distance, at variance with a prior study.[47] Previous work has reported higher PSQI scores as a risk factor for future exacerbation.[7] Considering a broader spectrum of clinical ailments, worse sleep quality has also been associated with fall risk and progression to diabetes mellitus or end-stage kidney disease.[48–50] One Taiwanese study noted an association with mortality that was attenuated after adjustment for depressive symptoms and other variables, again highlighting the important interactions of these two problems.[51]

The limitations of this work should be appreciated. There were no objective measures of sleep quality. Cross-sectional analysis precluded any assessment of causality. We did not measure disease control or severity except in COPD, but several conditions considered are known to vary by race and could have influenced results. The data we collected on depressive symptoms was limited to a single self-report instrument, while the highest quality assessment would have been through structured psychiatric interviews. Recall bias may also have affected some measures, especially components of our Adversity-Opportunity Index like in utero smoke exposure. Future studies should explore further consequences of poor sleep quality, as well as interventions to address it. COPD research would also benefit from continued exploration of the PSQI's construct validity and confirm the best threshold for good vs poor sleep in this particular population.

CONCLUSIONS

Among 1,965 non-Hispanic White and 465 African American current or former heavy smokers, we found that African American race was independently associated with worse PSQI score after controlling for demographics, socioeconomic status, and health status. When compared by race, we found that both the determinants and subscales of the PSQI had differing relative importance. We identified the PSQI had favorable psychometric characteristics in the limited analysis performed and that higher scores were associated with worse quality of life. These findings attest to the importance of social determinants in

clinical outcomes. Future research should assess interventions to address these inequities. Providers should attend to sleep quality as a potential cause for poor outcomes among their patients.

ABBREVIATIONS

COPD, chronic obstructive pulmonary disease
FEV_1, forced expiratory volume in 1 second
PSQI, Pittsburgh Sleep Quality Index
SGRQ, St. George's Respiratory Questionnaire
SPIROMICS, Subpopulations and Intermediate Outcome Measures in COPD Study

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