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Racial Disparities in Sleep Disturbances among Patients with and without Coronary Artery Disease: The Role of Clinical and Socioeconomic Factors

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Abstract

Objective—To investigate differences in sleep quality by race in participants with and without a prior myocardial infarction (MI).

Design—Case-control study

Setting—Emory-affiliated hospitals in Atlanta, Georgia

Participants—273 individuals (190 Black) ≤ 60 years of age with a verified MI in the previous 8 months, and 100 community controls (44 Black) without a history of MI.

Measurements—Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). Psychological factors were assessed using standardized questionnaires and clinical risk factors through medical history and chart review.

Results—A significant interaction existed between race and MI status on sleep quality (p=0.01), such that Black individuals with a history of MI, but not controls, reported worse sleep quality than their non-Black counterparts. Among MI cases, being Black was independently associated

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with higher PSQI scores after adjusting for baseline demographics (B =2.17, 95% CI 1.17, 3.17, p=0.006). Clinical risk factors, psychological factors and socioeconomic status (household income and years of education) all contributed equally to explain race-related disparities in sleep among MI cases. After further adjustment for these factors, the association was attenuated and no longer significant (B=0.70, 95% CI= −0.10, 1.21, p=0.26).

Conclusion—Black post-MI patients, but not healthy controls, have significantly poorer sleep quality than non-Blacks. This difference is driven by a combination of factors, including clinical risk factors, psychological factors as well as adverse socioeconomic conditions among Black individuals with MI.

Keywords

sleep quality; Pittsburgh Sleep Quality Index; coronary artery disease; racial differences

Introduction

Well-established racial disparities between Black adults and other demographic groups exist in cardiovascular health outcomes across the United States.[1,2] National statistics indicate that Black adults have a higher burden of prevalent coronary artery disease (CAD), heart failure, and stroke.[3] Equally as concerning is the fact that Black adults have higher mortality rates from cardiovascular disease, and deaths occur at a younger age compared to other groups.[3,4] Potential mechanisms for these observed health disparities are likely multifactorial and multi-level, including societal, community and individual factors, and remain overall not well understood.[5]

Short sleep durations, poor sleep quality and sleep related disorders such as insomnia and obstructive sleep apnea are all recognized as independent risk factors for the development and progression of cardiovascular disease.[6–9] These factors also appear to differentially affect Black individuals compared to other groups, and race differences have been reported across multiple sleep domains.[10–15] Most prior literature has focused on racial differences in sleep duration and prevalence of sleep disorders, and data on racial disparities in broadly defined sleep quality are limited.[14–16] These questions have never been addressed among patients with CAD, a high-risk group among whom such disparities could be exacerbated. Understanding the magnitude of racial differences in sleep quality among patients with CAD could help mitigate racial disparities in the outcome of CAD.

One of the hypothesized explanations for racial differences in sleep quality is disparities in the socioeconomic status (SES) between racial groups.[17] However, few studies have directly examined the role of SES in explaining disparities in sleep quality by race, and these studies have produced conflicting results.[18–23] It is important to understand the factors explaining the disparities in sleep quality by race in order to develop successful interventions to improve sleep and reducing health inequalities.

In the present study, we investigated differences in sleep quality by race in participants with and without a prior MI, and further explored the role of SES in any differences found. We hypothesized that, among adults with a history of MI, poor sleep quality is more common

among Blacks than among other racial groups, and that these differences in sleep quality are partially mediated by SES.

Methods

Study population

The Myocardial Infarction and Mental Stress 2 study included patients with myocardial infarction (MI) and community controls without a history of CAD.[24] Cases and controls were recruited between June 2011 and March 2016. Cases with MI were recruited from a pool of patients admitted at Emory-affiliated hospitals in Atlanta, Georgia with a documented MI in the previous 8 months and who were 18–60 years of age at the time of screening. We first recruited the MI cases, and then recruited the population controls sampling them according to the cases' distribution of age and sex, as commonly done in case-control studies. The diagnosis of MI was verified by medical record review based on standard criteria of increases in troponin level together with symptoms of ischemia and ECG changes or other evidence of myocardial necrosis.[25] Control subjects were recruited from a community-based sample from the Atlanta area without established CAD, congestive heart failure, or stroke.[26] This community sample was selected using a two stage approach. The first stage included a random sample of Black and White residents of metropolitan Atlanta; the second stage included a subset who participated in an in-person study visit. Only community individuals whose age was in the same range as the MI cases were considered. The availability of this existing community sample for whom we already had information on demographic factors and past medical history allowed us to select controls without a previous history of CAD who could be matched to the cases. Cases and controls were frequency matched by age and sex, with a goal of achieving approximately 50% women and a similar mean age in both samples.[27] Data on sociodemographic and psychosocial variables were collected for all participants. All participants provided written informed consent and the protocol was approved by the Emory University Institutional Review Board.

Patient assessments

Both cases and controls were evaluated by study nurses and physicians during in-person clinical examination, with medical record review for cases. Baseline demographics (age, sex, race, years of education, and household income) were obtained using standardized self-reported questionnaires. Given that only 18 individuals (4.8%) were neither Black nor White, race was examined in 2 categories: Black and non-Black. Household income was classified into 2 groups (≤ $50,000, or > $50,000). Previous history of co-morbid conditions and risk factors (diabetes, hypertension, smoking, congestive heart failure, and obesity) was obtained by study nurses or physicians through medical history, clinical examination and by reviewing medical records. Angina symptoms were assessed with the Seattle Angina Questionnaire's angina-frequency subscale, which measures frequency of angina and use of nitroglycerin for chest pain over the previous 4 weeks, and with higher scores indicating less chest pain.[28]

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), which is a self-rated questionnaire. The scale includes 19 items that generate seven component scores:

subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medication, and daytime dysfunction. Each individual item is scored from 0 to 3, and a composite score is calculated as the sum of the seven components, ranging from 0 to 21, with a higher score denoting worse sleep quality. Poor sleep quality was defined as a PSQI > 5, as previously established.[29] In addition, the time to fall asleep in minutes, and total hours of sleep were measured using the raw numbers provided by the individual.

Several psychological factors were assessed using standardized questionnaires. Depressive symptoms were measured using the Beck Depression Inventory, a 21-item self-administered scale.[30] Posttraumatic Stress Disorder (PTSD) symptoms were assessed with the 17-item scale PTSD Symptom Checklist.[31] To avoid overlap with the PSQI, the sleep item in both the Beck Depression Inventory and PTSD Symptom Checklist were excluded. Trait anxiety was measured using with the State-Trait Anxiety Inventory scale.[32,33] For measurement of anger trait, we used the Spielberger's State-Trait Anger Expression Inventory was used,[33] and to measure general stress, the Perceived Stress Scale.[34] In order to assess overall psychological burden, a composite score of the above psychosocial scales was constructed as previously described.[35,36] First, for each psychological scale, a Z-score was constructed and the Z-scores were then summed to derive a composite psychological distress index. Since sleep disturbance is part of the Beck Depression Inventory and the PTSD Symptom Checklist, the composite scores were calculated after exclusion of the questions related to sleep disturbances from these two scales.

Statistical analysis

We compared non-Blacks and Blacks within each group (controls and MI cases) for demographic, behavioral, and clinical characteristics, and sleep quality indices using t tests for continuous variables and χ-squared tests for categorical variables. Linear regression analysis was used to determine the association between race and the PSQI score as the outcome variable. We constructed a series of sequential models. Model 1 adjusted for age and sex. Model 2 adjusted for all variables in Model 1 plus history of hypertension and obesity, and, among MI cases only, smoking, and congestive heart failure. Model 3 adjusted for all variables in Model 2 plus the composite psychological distress index. Model 4 adjusted for all variables in Model 3 plus angina frequency. Model 5 adjusted for all variables in Model 4 plus SES variables (years of education and household income). The interaction of race with case-control status was formally tested by entering the interaction term in the regression models. In order to characterize the contribution of each variable in the models predicting sleep quality, we performed relative importance analysis,[37] to represent the percentage of variance explained in the criterion that can be attributed to PSQI. [37] The STATA command DOMIN was used to generate general dominance weights and produce additive decompositions of R^2 indexes ascribing what can be interpreted as the "relative importance" of each variable in the prediction of PSQI as the outcome.

We also performed formal parallel mediation analysis with bootstrapping (1,000 bootstrap samples and a 95% confidence interval) to test the hypothesis that SES (household income and years of education) mediates the relationship between race and sleep disturbances using

the method by Preacher and Hayes.[38] All analyses were conducted using Stata 14 (StataCorp, College Station, Texas). A P-value of < 0.05 was considered statistically significant.

Results

Table 1 describes demographic, behavioral, and clinical characteristics, and sleep quality measures between non-Blacks and Black adults in each group (controls and MI cases). Among both controls and MI cases, Blacks had lower education and a lower household income (Table 1). Blacks with previous MI had a more adverse psychosocial profile (more depression, more PTSD symptoms, and more perceived stress) compared with non-Blacks with previous MI. Differences in psychosocial factors were less pronounced among controls. The rates of cardiovascular risk factors (diabetes, hypertension, and congestive heart failure), and angina frequency were also higher in Blacks with MI in comparison to non-Blacks; several risk factors were also disproportionately present in Blacks among controls (Table 1).

As shown in Table 2, among those with prior MI, Blacks exhibited higher PSQI scores as well as higher scores in multiple dimensions of the PSQI, denoting worse sleep compared to non-Black participants. Differences by race in the hours of sleep were modest among controls. Among both controls and MI cases, however, Blacks reported fewer hours of sleep (P for all < 0.01). There was a significant interaction between race and case-control status on PSQI scores (p=0.01), such that the association between PSQI and race was larger among those with previous MI (Table 2). Similarly, racial differences in poor sleep quality and time to fall asleep were larger among those with MI (Table 2).

Multiple regression analysis

In a multiple regression model adjusting for demographics, clinical and psychological factors, both race (B= 1.2, 95% CI= 0.4–1.9, for Blacks vs. non-Blacks) and case-control status (B= 1.1, 95% CI= 0.1–2.1, for MI vs. controls) were independently associated with higher PSQI scores. In addition, both lower income (B= −0.85, 95% CI= −1.57, −0.12) and lower levels of education (B= −0.18, 95% CI= −0.06, −0.007) were independently associated with higher PSQI scores.

After stratification by MI status (Table 3), Black race remained significantly associated with higher PSQI scores among MI cases after adjusting for baseline demographics (Model 1). Adjustment for clinical risk factors (Model 2) attenuated the association by 30.8%. This association was further attenuated after adjusting for the composite psychological score (Model 3), and angina (Model 4) by 26.0% and 9.0%, respectively, but it remained statistically significant. After further adjusting for SES (income and education), the association between higher PSQI and Black race was further attenuated by 30.7% and no longer significant (Model 5). Exclusion of those who did not identify themselves as White/Black did not change the associations (Supplemental Table 1).

Further analysis of the individual sleep quality component scores revealed that among those with previous MI, Black race was associated with worse scores in most of the components, including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, and

sleep disturbances, even after adjusting for clinical and psychological factors (Table 4). When further adjusting for SES, these associations were attenuated and only remained significant for sleep duration. Exclusion of those who did not identify themselves as White/Black did not change the associations (Supplemental Table 2).

Relative importance analysis was performed to characterize the contribution of each variable to sleep quality in the models with PSQI as outcome among individuals with prior MI (Supplemental Table 3). As shown in Figure 1 and Supplemental Table 3, the composite psychological distress index was the most important contributor to sleep quality (47.4%), followed by the frequency of angina symptoms (14.1%), and household income (12.2%), respectively. With these factors in the model, race was only a minor contributor to differences in sleep quality.

Mediation Analysis

We performed mediation analysis to examine the hypothesized pathway that among those with a prior MI, markers of lower SES mediate the relationship between black race and worse sleep quality. As shown in Figure 2, among cases with prior MI, Black race had direct relationships with both household income and education, as well as PSQI levels ($P<0.01$), after adjusting for other risk factors. Household incomes and years of education, when added to the model, mediated the relationship between Black race and PSQI by 34%, and 10% respectively.

Discussion

In the present study, we found that the racial differences in PSQI was more than 2 times higher among cases with prior MI compared to controls. Among those with prior MI, worse sleep quality was explained by the more adverse cardiovascular risk profile, worse psychosocial profile and fewer years of education and lower household income in Black patients compared to their non-Black counterparts. However, among controls, these factors were overall similar by race, which could help explain why the differences in sleep quality were not as pronounced among Blacks compared to non-Blacks. Consistent with our initial hypothesis, we found that SES was a major explanatory factor for race differences in sleep. However, we also found that, in addition to SES, differences in traditional risk factors and psychological factors contributed to the race-related differences in sleep quality among MI patients.

Poor sleep quality was shown to increase the incidence of cardiovascular risk factors, such as obesity,[39] hypertension,[40] and diabetes,[41] and is also linked to a higher risk of cardiovascular events including MI[42] and total mortality. Following a first-time acute MI, sleep impairment and insomnia have shown to increase the risk of future MI, stroke, heart failure and death.[43,44] Both the quantity and quality of sleep have been shown to influence the pathophysiology of cardiovascular disease via inflammatory and autonomic pathways, among others.[45] The higher degree of systemic inflammation and autonomic dysregulation associated with poor sleep quality could make the post-MI population especially vulnerable to the long-term adverse effects of poor sleep.

In our sample, Black adults had more risk factors and comorbidities than non-Blacks in both MI and control groups, but, as expected, the prevalence of these conditions was much higher in the MI cases. Black adults in the MI group had higher rates of hypertension, diabetes and congestive heart disease compared to non-Blacks. All these factors have shown to be associated with poor sleep quality.[46–48] Therefore, the fact that in our sample there were mostly no significant differences in the PSQI scores between Black and non-Black participants without CAD could be related to the lower risk profile of the control group. In addition, the disparity in SES indicators between Black and non-Black adults was less pronounced among controls compared to individuals with MI, which could also explain why these individuals had a more similar sleep quality. However, Blacks in the control group did sleep fewer hours than their non-Black counterparts. This difference could be partly attributed to higher prevalence of obesity in Blacks compared to non-Blacks in the control group, which could increase the rate of obstructive sleep apnea in the former group.

Among MI cases, clinical risk factors including diabetes, hypertension and congestive heart failure explained a considerable portion of the sleep disparity by race. These data suggest that an improvement in the clinical risk factors of Blacks with MI could potentially improve their quality of sleep. Nonetheless, even after accounting for clinical and psychosocial differences, Black patients with previous MI still had worse sleep quality than non-Blacks. It was only after accounting for differences in socioeconomic status that most of the differences in sleep were explained.

Consistent with prior literature, we found that Black participants reported fewer hours of sleep compared to non-Blacks. These differences were seen among both those with and without MI, and were independent of medical and psychological factors. Our findings are in line with a previous meta-analysis of subjective and objective measures of sleep that showed that, on average, Black people sleep fewer hours than non-Blacks, and that these differences are at least partly moderated by biopsychosocial factors.[15] Prior observational studies have linked a shorter sleep duration to the development of obesity, diabetes, and their downstream effects on cardiovascular health.[45,49] In another meta-analysis of over 400,000 participants, the relative risk for developing or dying from CAD or stroke was increased by almost 50% among those who slept less than 6 hours per night compared to those who slept between 7 – 8 hours per night.[50] These findings highlight the importance of understanding the reasons for short sleep durations in Black individuals as it may directly influence their future risk of cardiovascular disease incidence and mortality. Our results suggest that social factors related to low socioeconomic resources are important determinants of observed disparities in sleep quality by race.

In our study, we demonstrated that markers of SES including low income and fewer years of education were among the most important predictors of worse sleep quality among participants with prior MI, and more important than clinical risk factors. A lower SES (household income and education) among Black individuals with MI explained approximately one third of the relationship between racial disparities and sleep disturbances after adjustment of other factors. Previous studies have documented a direct relationship between SES and sleep health.[51–54] Furthermore, income level is associated with behavioral factors known to influence sleep, such as smoking, excessive alcohol, overweight and lack

of exercise,[55] as well as with higher rates of anxiety and depressive symptoms.[56,57] Individuals with lower income are also more likely to live in crowded households, to work later shifts, to hold multiple jobs, and to work long hours, all of which can have profound implications for sleep quality and timing.[58,59] These findings suggest that income and education as markers of SES may play an important role in sleep health both directly and also through modulation of physical and mental health. Psychological disturbances were by far the most important predictor of poor sleep, and were more prevalent among Black study participants with MI than those of other race/ethnicity groups. Thus, it is not surprising that they did contribute to explain a substantial portion of the race differences in sleep quality among MI cases.

Our study has a number of limitations. First, the cross-sectional design precludes any conclusions on the causality of the associations found. Second, sleep quality was evaluated subjectively, and objective measures of sleep were not available. Third, the lack of significant associations between race and sleep disturbances among the controls could be attributed to the relatively small sample size of this group. However, except for sleep duration, the effect size for race was unequivocally smaller among controls compared to the MI cases, with significant interactions for many sleep domains. Fourth, while our relative importance analysis showed race to be a minor contributor to differences in sleep quality, this could also be attributed to the fact that proximal factors demonstrate a higher level of relative importance in this method of analysis. Fifth, data on sleep disordered-breathing or other sleep disorders were not collected in our sample, which could also have a role in the variance seen in sleep quality especially among MI patients. Sixth, the control group could be affected by self-selection of participants who agreed to a study visit. However, the MI sample was also invited for a study visit; thus, if there is a selection bias, it should be non-differential. Finally, data regarding clinical outcomes were not available. Our group has recently shown that both short and long sleep duration is associated with higher all-cause mortality in both Blacks and non-Blacks with CAD.[60] Future studies are needed to investigate whether higher rates of sleep disturbances among Black adults with MI translate into worse future outcomes. Strengths of this study include the comprehensive phenotyping of both cases and controls across multiple levels, including sociodemographic, medical and psychosocial data, which allowed us to investigate the role of all these factors on racial disparities in sleep. The design including both MI cases and controls without CAD represents another important strength of this study.

In conclusion, we showed that Black individuals who have suffered an MI are especially vulnerable to sleep disturbance. Clinical risk factors, psychological factors and adverse SES all play important roles in race-related disparities in sleep. This group may potentially benefit for targeted interventions to ameliorate sleep. Future prospective studies should address the impact of sleep quality and duration on racial disparities in MI recurrence and mortality, and test the usefulness of interventions aimed at improving sleep in this population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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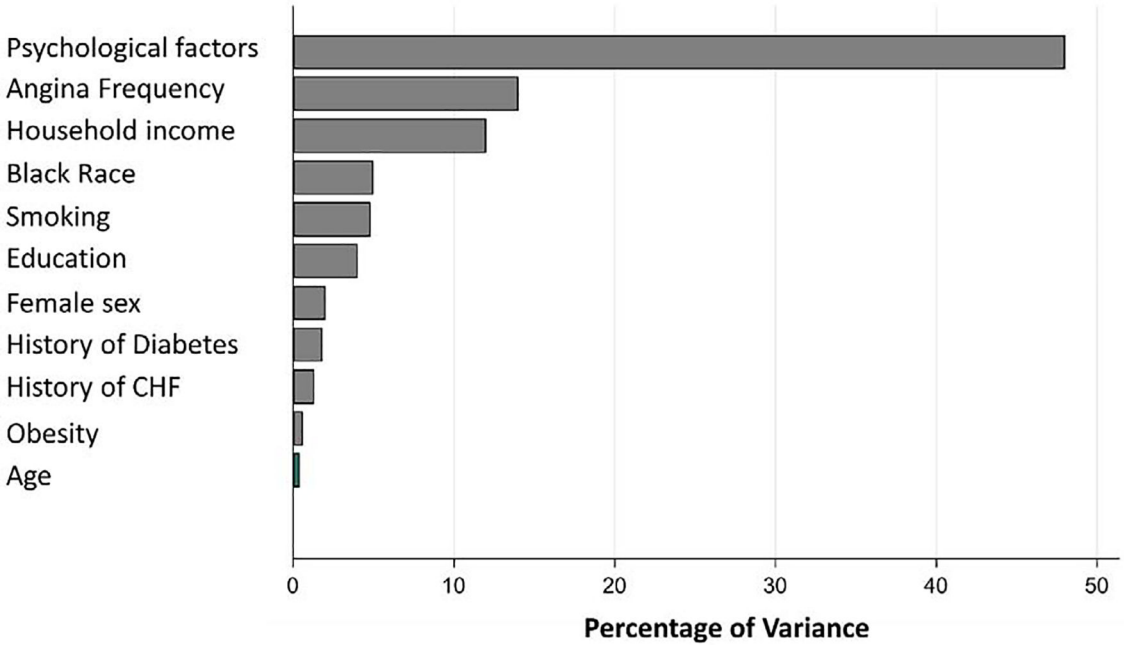

Figure 1.

Significant ($p<0.05$) predictors of PSQI among subjects with prior myocardial infarction. Variables displayed are the statistically significant predictors of PSQI subscales on univariate analysis.

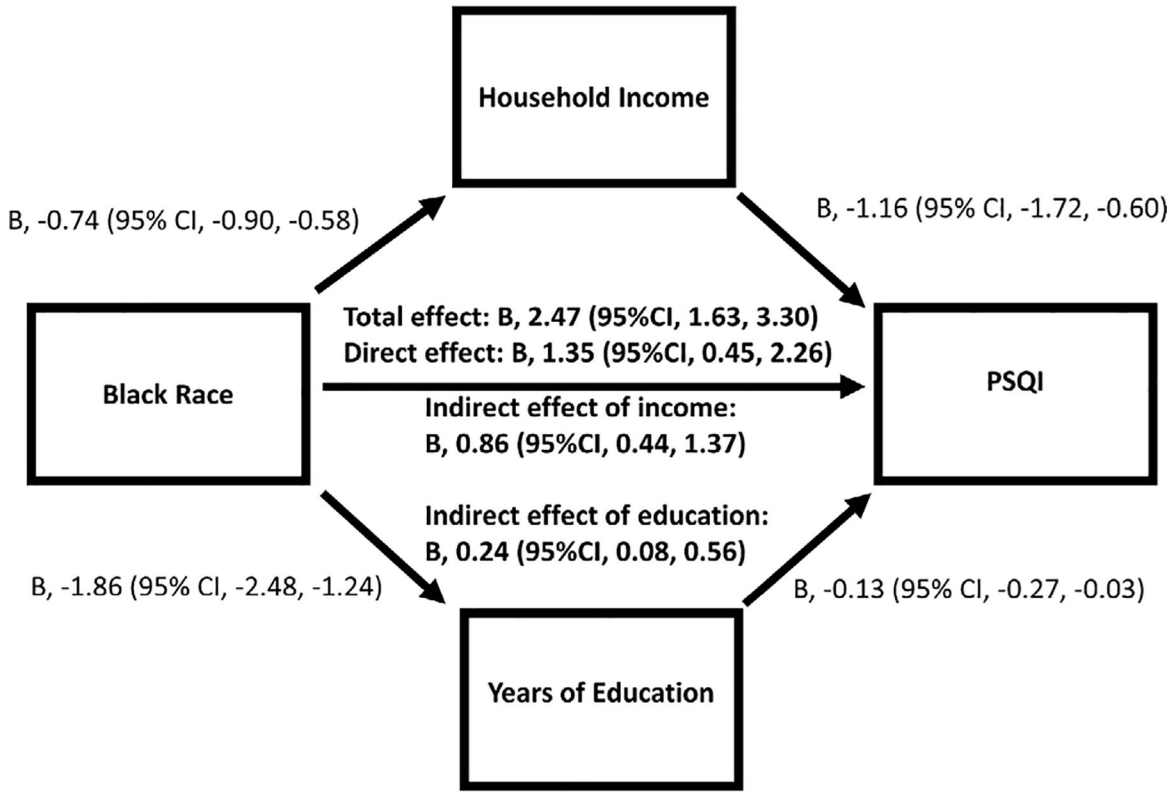

Figure 2.

Mediation analysis for socioeconomic status as mediator of the relationship between Black race and sleep disturbances among the MI cases (N=273)

Table 1.

Baseline characteristics by case-control status and race.

	Controls (N=100)		p-value	MI Cases (N=273)		p-value
	Non-Black (N=56)	Black (N=44)		Non-Black (N=83)	Black (N=190)	
Sociodemographics						
Age, Mean (SD)	48 (10)	50 (6)	0.33	52 (5)	50 (7)	0.07
Female, N (%)	27 (48.2)	25 (56.8)	0.39	32 (38.6)	106 (55.8)	**0.009**
Education, Years (SD)	17 (3)	15 (2)	**0.005**	14 (3)	13 (2)	**<0.001**
Household income, N (%)			0.62			<0.001
≤ $50,000	15 (24.6)	13 (28.9)		37 (36.3)	144 (81.8)	
> $50,000	46 (75.4)	32 (71.1)		65 (63.7)	32 (18.2)	
Psychological factors						
BDI score, Mean (SD)	6.1 (1.2)	6.3 (1.1)	0.86	9.6 (0.9)	13.4 (0.7)	**0.006**
PCL score, Mean (SD)	24.6 (111)	24.5 (10.2)	0.97	27.2 (1.2)	33.9 (1.1)	**<0.001**
STAI score, Mean (SD)	30.2 (10.1)	29.0 (12.2)	0.52	35.1 (13.1)	36.6 (13.0)	0.35
STAXI score, Mean (SD)	16.7 (5.6)	17.5 (6.7	0.45	17.8 (5.8)	18.7 (7.6)	0.28
PSS score, Mean (SD)	10.7 (6.7)	9.8 (5.82)	0.45	14.5 (6.2)	17.1 (8.4)	0.02
Summary score*, Mean (SD)	54.8 (25.3)	55.2 (23.7)	0.92	134.1 (71.0)	158.2 (71.1)	0.005
Comorbidities						
Diabetes, N (%)	2 (3.6)	5 (11.4)	0.13	16 (19.3)	69 (36.3)	**0.005**
Hypertension, N (%)	14 (25.0)	16 (36.4)	0.21	56 (67.5)	169 (88.9)	**<0.001**
Congestive heart failure, N (%)	0	0		3 (2.8)	29 (14.1)	**0.002**
Ejection Fraction, Mean (SD)	-	-		51.2 (12.0)	50.0 (11.4)	0.67
Obesity, N (%)	10 (17.9)	26 (59.1)	**<0.001**	39 (47)	107 (56.3)	0.23
Current Smoking, N (%)	3 (5.4)	2 (4.5)	0.85	16 (19.3)	50 (26.3)	0.21
Angina Frequency score, Mean (SD)	1.5 (6.6)	3.4 (8.3)	0.25	12.0 (17.3)	20.1 (21.6)	**0.002**

BDI: Beck Depression Inventory, PCL: Posttraumatic Stress Disorder Symptom Checklist, STAI: State-Trait Anxiety Inventory, STAXI: State-Trait Anger Expression Inventory, PSS: perceived stress scale, PSQI: Pittsburgh Sleep Quality Index

*Summary score aggregate of BDI, PCL, STAI, STAXI, and PSS scores

Table 2.

Sleep quality measures by case-control status and race.

	Controls (N=100)		Magnitude of difference (95% CI)	p-value	MI Cases (N=273)		Magnitude of difference (95% CI)	p-value	P for Interaction
	Non-Black (N=56)	Black (N=44)			Non-Black (N=83)	Black (N=190)			
PSQI Scale Scores									
PSQI total score, Mean (SD)	5.8 (0.5)	6.5 (0.6)	0.6 (−0.2, 0.1)	0.20	6.7 (0.3)	8.8 (0.3)	**2.2 (1.2, 3.2)**	**<0.001**	**0.01**
Poor quality sleep (PSQI > 5), N (%)	23 (41.1)	22 (50.0)	-	0.37	52 (61.9)	147 (75.0)	-	**0.02**	0.31
Subjective sleep quality	0.8 (0.6)	1.1 (0.8)	0.2 (−0.01, 0.5)	0.07	1.1 (0.7)	1.4 (0.9)	**0.38 (0.1, 0.6)**	**0.001**	0.12
PSQI Subscales									
Sleep latency	1.1 (1.0)	1.0 (1.0)	0.03 (−0.4, 0.3)	0.88	1.1 (0.8)	1.5 (1.0)	**0.43 (0.1, 0.7)**	**<0.001**	**0.009**
Sleep duration	0.3 (0.6)	0.9 (0.9)	**0.58 (0.2, 0.9)**	**<0.001**	0.6 (0.9)	1.2 (1.1)	**0.52 (0.2, 0.8)**	**<0.001**	0.74
Habitual sleep efficiency	0.3 (0.6)	0.5 (0.9)	0.25 (−0.06, 0.5)	0.11	0.5 (0.9)	1.0 (1.1)	**0.42 (0.1, 0.7)**	**0.002**	**0.02**
Sleep disturbance	1.31 (0.6)	1.35 (0.5)	0.04 (−0.2, 0.2)	0.72	1.5 (0.6)	1.8 (0.7)	**0.25 (0.08, 0.4)**	**0.005**	**0.01**
Use of sleeping medication	0.6 (1.1)	0.7 (1.2)	0.14 (−0.3, 0.6)	0.53	0.7 (1.1)	0.8 (1.2)	0.08 (−0.2, 0.4)	0.54	0.83
Day time sleep dysfunction	0.6 (0.6)	0.6 (0.8)	0.02 (−0.2, 0.3)	0.89	0.9 (0.7)	1.0 (0.8)	−0.09 (−0.1, 0.3)	0.34	0.61
Time to fall asleep (Minutes)	22.8 (23.4)	25.2 (22.8)	2.6 (−6.5, 11.8)	0.57	23.5 (21.6)	41.4 (43.8)	**18.6 (9.3, 27.8)**	**<0.001**	**0.03**
Hours of sleep	7.0 (1.0)	6.1 (1.1)	**−0.9 (−1.3, −0.5)**	**<0.001**	6.5 (1.5)	5.8 (1.8)	**−0.6 (−1.0, −0.2)**	**0.006**	0.41

A higher score in the PSQI subscales indicates worse sleep quality.

Table 3.

Multivariable regression investigating the association between PSQI (outcome) and race stratified by case-control status

	Controls (N=100) B (95% CI)		MI Cases (N=273) B (95% CI)		P for interaction	Percent of Attenuation among MI cases
	Non-Black (N=56)	Black (N=44)	Non-Black (N=83)	Black (N=190)		
Model 1	0 (Reference)	0.91 (−0.59, 2.41)	0 (Reference)	**2.17 (1.17, 3.17)**	**0.01**	
Model 2	0 (Reference)	0.54 (−1.04, 2.13)	0 (Reference)	**1.51 (0.47, 2.56)**	**0.02**	**30.8%**
Model 3	0 (Reference)	0.43 (−1.01, 1.88)	0 (Reference)	**1.11 (0.23, 1.99)**	**0.02**	**26.0%**
Model 4	0 (Reference)	0.78 (−0.87, 2.47)	0 (Reference)	**1.01 (0.10, 1.92)**	**0.02**	**9.0%**
Model 5	0 (Reference)	0.70 (−0.68, 2.08)	0 (Reference)	0.70 (−0.10, 1.21)	**0.03**	**30.7%**

The B coefficient expresses the increment in PSQI total score points (reflecting worse sleep quality) in Blacks compared with non-Blacks.

Model 1. Adjusted for age, and sex

Model 2. Adjusted for all variables in Model 2 plus history of hypertension and obesity, and, among MI cases only, also smoking and congestive heart failure.

Model 3. Adjusted for all variables in Model 3 plus the summary score aggregate of psychological factors

Model 4, Adjusted for all variables in Model 4 plus the angina frequency

Model 5. Adjusted for all variables in Model 5 plus years of education, and household income

Table 4.

Multivariable regression investigating the association of PSQI total score and individual PSQI component scores with race among patients with MI

	MI Cases (N=273) B (95% CI)		
	Non-Black(N=83)	**Black (N=190)**	**p- Value**
	Model 1		
PSQI total	0 (Reference)	**2.17 (1.17, 3.17)**	**0.005**
Subjective sleep quality	0 (Reference)	**0.027 (0.04, 0.50)**	**0.01**
Sleep latency	0 (Reference)	**0.25 (0.02, 0.48)**	**0.027**
Sleep duration	0 (Reference)	**0.38 (0.11, 0.64)**	**0.004**
Habitual sleep efficiency	0 (Reference)	**0.50 (0.22, 0.78)**	**<0.001**
Sleep disturbance	0 (Reference)	**0.36 (0.08, 0.65)**	**0.010**
Use of sleeping medication	0 (Reference)	0.04 (−0.25, 0.34)	0.76
Day time sleep dysfunction	0 (Reference)	0.10 (−0.10, 0.31)	0.31
Time to fall asleep	0 (Reference)	**17.5 (7.7, 27.2)**	**<0.001**
Hours of sleep	0 (Reference)	**−0.61 (−1.0, −0.17)**	**0.007**
	Model 2		
PSQI total	0 (Reference)	**1.51 (0.47, 2.56)**	**0.028**
Subjective sleep quality	0 (Reference)	**0.19 (0.03, 0.41)**	**0.01**
Sleep latency	0 (Reference)	**0.21 (0.05, 0.49)**	**0.009**
Sleep duration	0 (Reference)	**0.42 (0.12, 0.72)**	**0.005**
Habitual sleep efficiency	0 (Reference)	**0.34 (0.04, 0.64)**	**0.02**
Sleep disturbance	0 (Reference)	**0.20 (0.07, 0.29)**	**0.02**
Use of sleeping medication	0 (Reference)	−0.11 (−0.43, 0.19)	0.45
Day time sleep dysfunction	0 (Reference)	−0.03 (−0.24, 0.17)	0.72
Time to fall asleep	0 (Reference)	**13.4 (3.1, 23.7)**	**0.01**
Hours of sleep	0 (Reference)	**−0.55 (−1.03, −0.07)**	**0.02**
	Model 3		
PSQI total	0 (Reference)	**1.11 (0.23, 1.99)**	**0.015**
Subjective sleep quality	0 (Reference)	**0.18 (0.02, 0.39)**	**0.02**
Sleep latency	0 (Reference)	**0.20 (0.05, 0.46)**	**0.008**
Sleep duration	0 (Reference)	**0.41 (0.11, 0.70)**	**0.006**
Habitual sleep efficiency	0 (Reference)	**0.33 (0.03, 0.63)**	**0.02**
Sleep disturbance	0 (Reference)	**0.19 (0.04, 0.26)**	**0.02**
Use of sleeping medication	0 (Reference)	−0.12 (−0.43, 0.18)	0.43
Day time sleep dysfunction	0 (Reference)	−0.05 (−0.24, 0.13)	0.55
Time to fall asleep	0 (Reference)	**13.4 (3.1, 23.7)**	**0.01**
Hours of sleep	0 (Reference)	**−0.54 (−1.02, −0.06)**	**0.02**
	Model 4		
PSQI total	0 (Reference)	**1.01 (0.10, 1.92)**	**0.023**
Subjective sleep quality	0 (Reference)	**0.17 (0.01, 0.29)**	**0.021**

	MI Cases (N=273) B (95% CI)		
	Non-Black(N=83)	**Black (N=190)**	**p- Value**
Sleep latency	0 (Reference)	**0.17 (0.03, 0.35)**	**0.01**
Sleep duration	0 (Reference)	**0.40 (0.09, 0.66)**	**0.01**
Habitual sleep efficiency	0 (Reference)	**0.32 (0.02, 0.59)**	**0.03**
Sleep disturbance	0 (Reference)	**0.11 (0.02, 0.20)**	**0.03**
Use of sleeping medication	0 (Reference)	−0.10 (−0.40, 0.14)	0.53
Day time sleep dysfunction	0 (Reference)	−0.06 (−0.14, 0.09)	0.34
Time to fall asleep	0 (Reference)	**13.0 (2.8, 21.5)**	**0.02**
Hours of sleep	0 (Reference)	**−0.55 (−1.00, −0.03)**	**0.031**
	Model 5		
PSQI total	0 (Reference)	0.70 (−0.10, 1.21)	0.191
Subjective sleep quality	0 (Reference)	0.15 (−0.06, 0.38)	0.16
Sleep latency	0 (Reference)	0.12 (−0.15, 0.41)	0.37
Sleep duration	0 (Reference)	**0.39 (0.07, 0.72)**	**0.016**
Habitual sleep efficiency	0 (Reference)	0.30 (−0.02, 0.62)	0.09
Sleep disturbance	0 (Reference)	0.05 (−0.12, 0.24)	0.53
Use of sleeping medication	0 (Reference)	−0.09 (−0.44, 0.24)	0.57
Day time sleep dysfunction	0 (Reference)	−0.11 (−0.32, 0.08)	0.25
Time to fall asleep	0 (Reference)	**12.2 (1.1, 24.3)**	**0.03**
Hours of sleep	0 (Reference)	**−0.61 (−1.1, −0.11)**	**0.01**

The B coefficient expresses the increment in PSQI total score or subscore points (reflecting worse sleep quality) in Blacks compared with non-Blacks.

Model 1. Adjusted for age and sex

Model 2. Adjusted for all variables in Model 2 plus history of diabetes, hypertension, obesity, smoking and congestive heart failure

Model 3. Adjusted for all variables in Model 3 plus the summary score aggregate of psychological factors

Model 4. Adjusted for all variables in Model 4 plus angina frequency

Model 5. Adjusted for all variables in Model 5 plus years of education, and household income