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Anxiety Sensitivity and Racial Differences in Sleep Duration: Results from a National Survey of Adults with Cardiovascular Disease

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Abstract

Although Blacks sleep between 37–75 minutes less per night than non-Hispanic Whites, research into what drives racial differences in sleep duration is limited. We examined the association of anxiety sensitivity, a cognitive vulnerability, and race (Blacks vs. White) with short sleep duration (<7 hours of sleep/night), and whether anxiety sensitivity mediated race differences in sleep duration in a nationally representative sample of adults with cardiovascular disease. Overall, 1289 adults (115 Black, 1174 White) with a self-reported physician/health professional diagnosis of 1 myocardial infarction completed an online survey. Weighted multivariable logistic regressions and mediation analyses with bootstrapping and case resampling were conducted. Anxiety sensitivity and Black vs. White race were associated with 4% – 84% increased odds, respectively, of short sleep duration. Anxiety sensitivity mediated Black – White differences in sleep duration. Each anxiety sensitivity subscale was also a significant mediator. Implications for future intervention science to address sleep disparities are discussed.

Keywords

anxiety sensitivity; cardiovascular disease; sleep; race; health disparities; mechanism

Racial disparities in sleep duration are well documented (Chen et al., 2015; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Rutter, Decoster, Jacobs, & Lichstein, 2011). For example, prior research using objective measures of sleep such as wrist actigraphy has found that Black men sleep on average up to 75 minutes less per night than White men, while Black women sleep up to 45 minutes less per night than White women (Chen et al., 2015). Short sleep duration, often classified as sleeping less than 7 hours per night, is also more

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common among Blacks than Whites (Jackson, Redline, & Emmons, 2015; Krueger & Friedman, 2009; Stamatakis, Kaplan, & Roberts, 2007). Indeed, several studies find that Black Americans are more than twice as likely to report short sleep duration than White Americans, and this pattern has been observed in healthy adults as well as those with cardiovascular disease (CVD) (Alcantara, Peacock, Davidson, Hiti, & Edmondson, 2014; Hale & Do, 2007; Jackson, Redline, Kawachi, Williams, & Hu, 2013). While racial differences in sleep duration are well characterized, potential modifiable factors driving racial differences in sleep duration are understudied in the general adult population broadly, and in those with cardiovascular disease, specifically. Importantly, short sleep duration is becoming increasingly recognized as a risk factor for cardiovascular disease and related health risk behaviors, as well as a marker of excess prognostic risks in those with CVD (St-Onge et al., 2016). Thus, knowledge into factors that drive racial differences in sleep patterns, particularly in adults with CVD, may inform future intervention efforts to improve sleep, reduce CVD health disparities, and in turn potentially advance health equity for all.

Prior research with healthy adults and patients with CVD suggests that anxious pathology and poor sleep often co-occur (Alvaro, Roberts, & Harris, 2013; Babson, Trainor, Feldner, & Blumenthal, 2010; Jackson et al., 2015). For example, in an experimental study, adults who were exposed to either anxious cognitive arousal (i.e., speech threat task) or physiological arousal (i.e., caffeine capsule) immediately before bedtime exhibited longer sleep onset latency and shorter total sleep time (Tang & Harvey, 2004). Observational longitudinal research has also found evidence of an adverse effect of anxiety on sleep. In a large sample of Finnish adults, a general predisposition toward anxious arousal, or —anxious liability, was associated with poor sleep quality in both men and women (Vahtera et al., 2007). People who endorsed high symptoms of sympathetic nervous system hyperactivity exhibited a higher vulnerability to report poor sleep quality, and this relationship was stronger in men compared to women, particularly under increasing severity of stressful life events (Vahtera et al., 2007). This finding demonstrates that subthreshold anxiety levels also affect self-reported sleep quality. Further, in adults with CVD, anxiety and sleep deficits have been shown to co-occur. At least one study finds that sleep latency and short slow wave sleep are associated with cognitive anxiety (Edell-Gustaffson, 2002). Importantly, far less research has focused on identifying underlying and modifiable cognitive vulnerabilities, such as anxiety sensitivity, and their associations with poor sleep.

Anxiety sensitivity, often referred to as —fear of fear, is defined as the fear of the negative social, physical, or cognitive consequences of anxiety-related sensations (Olatunji & Wolitzky-Taylor, 2009; S. Reiss & R. McNally, 1985; Reiss, Peterson, Gursky, & McNally, 1986). Anxiety sensitivity is conceptualized as a multi-dimensional and trait-like cognitive vulnerability that intensifies pre-existing levels of anxiety (Olatunji & Wolitzky-Taylor, 2009; S. Reiss & R. McNally, 1985; Reiss et al., 1986). Three lower-order and interrelated factors that pertain to fear of anxiety have been identified, and include: social concerns (e.g., belief that observable anxiety symptoms will lead to social rejection), physical concerns (e.g., belief that palpitations result in cardiac arrest/heart attack), and cognitive concerns (e.g., belief that concentration difficulties result in mental incapacitation) (Taylor, Rabian, & Fedoroff, 1999; Taylor et al., 2007). Individuals with high anxiety sensitivity are more likely to exhibit increased attention to signals and cues of anxious arousal, and to make negative

and catastrophic interpretations about anxiety-related sensations, and in turn experience increased anxiety, creating a positive and escalating feedback loop (Reiss & McNally, 1985).

A growing body of empirical evidence suggests that anxiety sensitivity is adversely related to optimal sleep health. High anxiety sensitivity has been linked with poor sleep quality, difficulty initiating sleep, and longer sleep onset latency in anxiety disorder clinical patient samples, even after accounting for important confounders such as depressive symptoms, worry, and traumatic life events (Babson, Boden, Woodward, Alvarez, & Bonn-Miller, 2013; Babson, Trainor, Bunaciu, & Feldner, 2008; Hoge et al., 2011; Raines et al., 2015). Related research with veteran populations also found that anxiety sensitivity was positively associated with worsening subjective sleep quality (Babson et al., 2013).

There is also recent preliminary evidence to suggest that specific subscales of anxiety sensitivity may be particularly related to sleep in the general adult population. For example, concerns about mental incapacitation or cognitive concerns related to anxious arousal were associated with sleep-related impairment (Vincent & Walker, 2001) and sleep dysfunction (Calkins, Hearon, Capozzoli, & Otto, 2013). Similarly, other research has found that cognitive concerns related to anxiety sensitivity (and not physical concerns or social concerns) partially mediated the effect of unacceptable thoughts on insomnia symptoms in adults with obsessive compulsive disorder (Raines et al., 2015). In contrast, other research has found that only the physical concerns subscale of anxiety sensitivity moderated the relationship between sleep anticipatory physical concerns and sleep onset latency (Babson et al., 2008). Specifically, sleep anticipatory physical concerns were associated with longer sleep onset latency only in those with high vs. low anxiety sensitivity (Babson et al., 2008).

Although there is mounting cross-sectional and longitudinal observational evidence of a relationship between anxiety sensitivity and poor sleep, most of this research was conducted with psychiatric samples and therefore not in cardiovascular disease samples, and without attention to racial/ethnic differences. Yet, the prevalence of anxious pathology, cardiovascular morbidity, and poor sleep differ across races/ethnicities (Chen et al., 2015; Evangelista, Ter-Galstanyan, Moughrabi, & Moser, 2009; Himle, Baser, Taylor, Campbell, & Jackson, 2009). Indeed, Blacks compared to Whites exhibit greater risk of adverse cardiovascular health behaviors, including poor sleep, and greater risk of CVD incidence and morbidity compared to their White counterparts (Jackson, Redline, & Emmons, 2015; Williams, Mohammed, Leavell, & Collins, 2010). Further, both poor sleep and anxiety sensitivity are established independent risk factors for CVD and cardiovascular health risk behaviors such as medication nonadherence, exercise, and smoking (Alcantara, Edmondson, et al., 2014; Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; Frasure-Smith et al., 2012; Smits, Tart, Presnell, Rosenfield, & Otto, 2010).

We used data from a nationally representative sample of adults with CVD in the United States (US) to: (1) examine the association of anxiety sensitivity and race (Black vs. White) with short sleep duration (< 7 hours of sleep/night), (2) test whether anxiety sensitivity mediates the relationship between race and sleep duration, and (3) explore whether specific subscales of anxiety sensitivity are consistent mediators of the effect of race on sleep duration. We hypothesized that anxiety sensitivity and Black race relative to White race

would be independently associated with increased odds of short sleep duration (< 7 hours of sleep per night). We also hypothesized that anxiety sensitivity would mediate the relationship between race and sleep duration. Our working conceptual model was that increasing levels of anxiety sensitivity would correspond with increased attention to and vigilance for any cues or signals that indicate anxious arousal. This increased attention would promote perseveration about anxiety sensations and their consequences, which in turn would reduce total sleep time because of time spent in bed perseverating (worrying or ruminating) about anxious arousal, and time not spent sleeping. We hypothesized that this type of vigilance and attention to anxious arousal would partly explain Black White differences in sleep duration.

Method

Participants

Participants included 1333 self-identified Black or non-Hispanic White US adults with a self-reported history of a physician or a health professional diagnosis of at least 1 myocardial infarction (MI). We restricted our sample to those who self-identified as Black or non-Hispanic White, reported 5 or less lifetime MIs, and average sleep duration of less than 15 hours per night. Our final analytic sample included 1289 participants (787 men, 502 women) with complete data for the predictor, outcome, covariate, and mediator variables. Participants had a mean age of 64.9 years ($SE=0.36$).

Procedures

We conducted a nationally representative online survey of US adults who reported that a doctor or health professional informed them that they had been diagnosed with a MI. YouGov, a nonpartisan research firm, administered the survey in English and Spanish from May 2013 to July 2013 to 1500 participants recruited from an opt-in survey panel. Participants received incentives through a YouGov loyalty program for their participation in the survey. YouGov panel members earn points for completing online surveys; the points can then be redeemed for rewards. Sampling targets and sampling weights were set based on gender, age, and race distribution of MI according to the 2010 National Health Interview Survey. Response rates were 45.8% for the English language survey and 16.2% for the Spanish survey. The Institutional Review Board of Columbia University approved this study.

Measures

Anxiety sensitivity—The Anxiety Sensitivity Index (ASI), a 16-item self-report questionnaire about fear of anxiety-related symptoms was used to measure anxiety sensitivity (Peterson & Reiss, 1992). Each item is scored using a 0 to 4 point Likert scale, where 0 refers to —very little and 4 refers to —very much. Sample questions include —It scares me when I am nervous and —It is important for me to stay in control of my emotions. Responses are summed to compute a total ASI score. Higher scores reflect higher levels of anxiety sensitivity. The internal consistency of this scale was excellent in this sample (Cronbach's $\alpha = 0.94$). The total ASI score was used to represent the global anxiety sensitivity factor. We also computed total scores for the three subscales: physical concerns

(Cronbach's $\alpha = 0.92$), social concerns (Cronbach's $\alpha = 0.73$), and cognitive concerns (Cronbach's $\alpha = 0.87$).

Race—Participants' self-reported their race as Black or non-Hispanic White.

Sleep Duration—Participants completed an abbreviated Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), a widely used self-report measure of sleep during the past month. Specifically, participants completed 6 out of the 24 items (questions 1–4, 6–7). Responses to the item, —During the past month, how many hours of actual sleep did you get at night? were used to evaluate self-reported sleep duration as a categorical and continuous variable. Subjectively-measured sleep duration was dichotomized *a priori* as less than 7 hours sleep/night (short sleep duration) or 7 hours or more of sleep/night (average sleep duration) based on previous research on sleep in cardiovascular disease samples that has found increased cardiovascular risks associated with this cut-off (Cappuccio et al., 2011). Responses to the item During the month, how would you rate your sleep quality overall were used to create a dichotomous poor sleep quality variable, where 0 referred to —fairly good/very good sleep quality and 1 referred to —fairly bad/very bad sleep quality. Participant responses to usual bedtime and wake time, and sleep duration were used to create a dichotomous poor sleep efficiency variable, where 1 referred to poor sleep efficiency defined as sleep efficiency <85%, and 0 referred to average/good sleep efficiency defined as $\geq 85\%$.

Covariates—Participants' age, gender (male, female), educational attainment (high school or less, some college, 4 years or more of college), number of lifetime MIs, self-reported lifetime anxiety disorder, and region of the country (Northeast, Midwest, South, West) were all self-reported. Participants' responses to the question —Has a nurse or doctor ever told you, you have anxiety was used as a proxy measure for lifetime anxiety disorder. The Patient Health Questionnaire (PHQ-8) (Kroenke et al., 2009) was used to measure depressive symptoms.

Analysis Plan

All analyses incorporated the complex survey design, specifically sampling weights using the *svy*—package in STATA (v.14). We computed descriptive statistics for the overall sample and by race and report unweighted N s and weighted percentages. Chi-square tests or Adjusted Wald tests were computed to examine potential racial differences across all predictors, outcomes, and covariates. Weighted multivariable logistic regression models were conducted to examine the minimally adjusted and fully adjusted associations of anxiety sensitivity and race with short sleep duration (<7 hours sleep/night). Model 1 adjusted for age, gender, and education level, Model 2 further adjusted for number of lifetime MIs, Model 3 included a final adjustment for depressive symptoms, and Model 4a and Model 4b additionally adjusted for either self-reported lifetime anxiety disorder or region of the country. Similarly, weighted multivariable linear regression models were also conducted to examine the minimally adjusted and fully adjusted associations of anxiety sensitivity and race with sleep duration across the four models. Mediation analyses using the *bootstrap sgmediation* command in STATA, which runs bootstrapping with case resampling (1000

replications) to calculate bias-corrected confidence intervals of the indirect effect, and *svy* prefix to account for the complex survey design were conducted to assess whether anxiety sensitivity (as a continuous variable) mediates the relationship between race (as a binary variable: White =0, and Black =1) and sleep duration (as a continuous variable). Standard errors of the indirect effect were bootstrapped. In sensitivity analyses, the single mediation models were rerun with: (1) an adjustment for important covariates, namely age, gender, educational attainment level, and number of lifetime MIs, (2) an adjustment for depressive symptoms, (3) an adjustment for lifetime anxiety disorder, (4) an adjustment for region of the country (Northeast, Midwest, South, and West); and (5) with each anxiety sensitivity subscale entered separately in unadjusted models. We also conducted three supplemental analyses with three sleep-related outcomes to evaluate the consistency of the findings. The additional outcomes included: (1) poor sleep quality; (2) poor sleep efficiency; and (3) short sleep duration operationalized as <6 hours of sleep.

Results

There were 1289 participants, 115 who self-identified as Black and 1174 as non-Hispanic White race. There were significant differences in sociodemographic, sleep duration, and psychosocial characteristics by race (Table 1). Black participants were younger, more likely to be female, and less likely to have completed 4 years or more of college than non-Hispanic White participants. Black participants had higher anxiety sensitivity scores ($M=29.60$, $SE=1.53$) and depressive symptom scores ($M=8.6$, $SE=0.62$) compared to non-Hispanic White participants (anxiety sensitivity: $M=15.23$, $SE=0.37$; depressive symptoms: $M=5.51$, $SE=0.17$). Black participants also slept fewer hours per night ($M=5.76$; $SE=0.18$) than non-Hispanic Whites ($M=6.65$, $SE=0.05$). There were no significant differences in number of lifetime MIs by race.

A one-unit increase in the total anxiety sensitivity symptom score was associated with a 4% increase in the odds of short sleep (< 7 hours sleep/night) in minimally adjusted and fully adjusted models (adjusted OR=1.04; 95% CI: 1.03, 1.05) (Table 2). Similarly, Black race vs. non-Hispanic White race was associated with 84% increased odds of short sleep (adjusted OR=1.84; 95% CI: 1.13, 3.00). These associations remained statistically significant after further adjustment for depressive symptoms, lifetime anxiety disorder, and region of the country (Table 2). These findings were largely consistent when a continuous measure of sleep duration was used (Table 3). Black race vs. non-Hispanic White race ($b=-0.47$, $SE=0.18$) and anxiety sensitivity ($b=-0.03$, $SE=0.00$) were each negatively associated with sleep duration (in hours) (Table 3, Models 1–2). In other words, Black race was associated with sleeping 37.2 minutes less than White race, and a one unit increase in anxiety sensitivity was associated with a 1.8 minute reduction in total sleep time while holding sociodemographics and number of heart attacks constant. However, the estimates for sleep duration associated with anxiety sensitivity were attenuated with further adjustment for depressive symptoms, though the estimates remained in the expected direction.

Results from the mediation analyses supported the direct and indirect effect of anxiety sensitivity on racial differences in average nightly sleep duration (Figure). The total effect for the model with race was significant, $b=-.88$, $SE=.19$, $p<.001$, as was the direct effect of

race on sleep duration accounting for anxiety sensitivity, $b = -.62$, $SE = .17$, $p < .001$. The indirect effect of race on sleep duration through anxiety sensitivity was also significant, $b = -.26$, $p < .001$, 95%BCa CI $[-.41 \text{ } -.15]$. Results of the complex sample adjusted Sobel-Goodman (Aroaian) test $z' = -3.95$, $p < .001$, suggest significant mediation (Figure). Anxiety sensitivity explained 29.6% of the total race effect on sleep duration. In supplemental mediation models that adjusted for covariates, including age, gender, education, and number of lifetime MIs, there was continuing evidence of significant attenuation of the direct effect ($z' = -2.32$, $p < .05$), and anxiety sensitivity mediated 17.5% of the race effect on sleep duration. In mediation models that adjusted for depressive symptoms alone, anxiety sensitivity mediated 9.5% of the effect of race on sleep duration ($z' = -2.04$, $p < .05$). Each of the anxiety sensitivity subscales were consistent significant mediators of the effect of race on sleep duration. The physical concerns subscale mediated 22.5% of the effect of race on sleep duration ($b = -.20$, 95%BCa CI $[-.39 \text{ } -.08]$, $z' = -3.49$, $p < .001$), whereas the social concerns subscale mediated 27.3% of the effect of race on sleep duration ($b = -.24$, 95%BCa CI $[-.37 \text{ } -.12]$, $z' = -3.95$, $p < .0001$), and the cognitive concerns subscale mediated 25.7% of the effect of race on sleep duration ($b = -.23$, 95%BCa CI $[-.35 \text{ } -.10]$, $z' = -3.54$, $p < .001$). In supplemental mediation models that separately adjusted for lifetime anxiety disorder and region of the country, anxiety sensitivity mediated 29.1% of the effect of race on sleep duration ($b = -.26$, 95%BCa CI $[-.38 \text{ } -.15]$, $z' = -4.21$, $p < .0001$), and 29.7% of the effect of race on sleep duration ($b = -.26$, 95%BCa CI $[-.43 \text{ } -.15]$, $z' = -3.92$, $p < .0001$), respectively.

Supplemental Analyses

Neither anxiety sensitivity nor Black race vs. non-Hispanic White race were statistically associated with poor sleep quality (Supplemental Table). In contrast, only anxiety sensitivity (adjusted OR=1.02; 95% CI: 1.00, 1.03), and not race (adjusted OR=0.93; 95%CI: 0.55, 1.55), was associated with poor sleep efficiency. Anxiety sensitivity was statistically associated with short sleep duration when defined as <6 hours sleep/night (adjusted OR=1.02; 95%CI: 1.00, 1.03). Although Black race vs. non-Hispanic White race was not statistically associated with short sleep duration when operationalized as <6 hours, the estimate was in the expected direction (adjusted OR=1.70; 95%CI: 1.00, 2.90) (Supplemental Table).

Discussion

Although Black White differences in sleep duration are well documented, the mechanisms driving this association are understudied in the general adult population broadly, and in adults with cardiovascular disease, specifically. Sleep is increasingly recognized as a risk factor for incident CVD and poor cardiovascular health as evidenced in the 2016 Scientific Statement of the American Heart Association on the importance of sleep duration for cardiometabolic health (St-Onge et al., 2016). Using cross-sectional data from a nationally representative sample of adults with CVD, we found that Black adults have an 84% increased odds of reporting short sleep (< 7 hours of sleep) compared to non-Hispanic White adults; these estimates are comparable to those observed in non-CVD samples (Ruiter et al., 2011; Stamatakis et al., 2007). Consistent with previous research with psychiatric samples, we found that an incremental increase in anxiety sensitivity had an independent and

significant positive association with short sleep duration. These results were largely consistent when sleep duration was evaluated as a continuous outcome, and when alternative cut-offs for short sleep duration were used. Further, the independent associations of anxiety sensitivity and race were specific to the sleep duration outcome; these associations were not observed when other sleep outcomes such as sleep quality and sleep efficiency were considered. Results from the mediation analyses provide initial empirical evidence that anxiety sensitivity partially mediated Black White differences in sleep duration. Specifically, anxiety sensitivity mediated 29.6% of the total race effect on sleep duration, and mediated between 9.5–17.5% of the total race effect on sleep duration after adjustment for important covariates such as sociodemographic factors, medical history, depressive symptoms, and when self-reported lifetime anxiety disorder and region of the country were also included as covariates. Our exploratory mediation analyses suggest a significant indirect effect through each anxiety sensitivity subscale on sleep duration.

Anxiety sensitivity or —fear of fear may mediate a proportion of the relationship between Black race and sleep duration in adults with CVD through physiological, cognitive, and psychological pathways that impede optimal sleep. For example, anxiety sensitivity, a trait-like cognitive vulnerability, may predispose individuals to be more vigilant for any cues or signals that indicate anxious arousal, which may promote perseveration about their symptoms, which in turn may lead to shorter sleep duration because of time spent perseverating (either worrying or ruminating) about their anxious arousal, and time not spent sleeping. Our findings indicate that this pernicious cycle of vigilance, anxious cognitive arousal, and potential perseveration may indeed contribute to the shorter average nightly sleep duration observed in Black adults with CVD compared to non-Hispanic White adults with CVD. Relatedly, mean anxiety sensitivity scores in this sample were markedly higher among Blacks than Whites, and met clinical thresholds for high anxiety sensitivity (e.g., scores greater than or equal to 24). Qualitative and quantitative research to understand the socio-structural and cultural factors (e.g., discrimination, neighborhood structural characteristics) that contribute to the differential distribution of anxiety sensitivity by race in adults with and without cardiovascular disease would advance scholarship on racial differences in cognitive vulnerabilities affecting mental health and sleep.

Importantly, anxiety sensitivity was only a partial mediator of racial differences in sleep duration, which indicates that other mechanisms are contributing to this relationship as well. For example, factors related to the social environment such as neighborhood structural characteristics (e.g., neighborhood noise level, neighborhood crime levels), and differential exposure to specific types of stressors such as discrimination, unfair treatment, or barriers to appropriate medical care after a MI, which are often directly related to race, may account for some of the remaining proportion of the effect of race on sleep duration (Slopen, Lewis, & Williams, 2016; Tomfohr, Pung, Edwards, & Dimsdale, 2012). Particularly relevant to the current work, previous research found that racism-related vigilance (a construct that shares key features such as vigilance and perseveration with anxiety sensitivity) was associated with poor sleep quality in a multi-ethnic sample, and that racism-related vigilance accounted for some of the observed Black White differences in sleep quality (Hicken, Lee, Ailshire, Burgard, & Williams, 2013). Although anxiety sensitivity and race were not associated with sleep quality in this sample of adults with cardiovascular disease, it is possible that racism-

related vigilance may further mediate racial differences in sleep duration. Future research on Black-White differences in sleep patterns should test the simultaneous contribution of socio-structural factors, discrimination, and other psychological factors such as racism-related vigilance as well as anxiety sensitivity.

Although our study is one of the first to identify a potential cognitive mediator of Black – White differences in sleep duration, there are several limitations. First, this was a cross-sectional study and as such we are unable to assess causality or directionality. Future research should use prospective designs to replicate these results and assess temporal ordering using more contemporary measures of the anxiety sensitivity construct such as the ASI-3, which was developed to reliably assess the three domains of anxiety sensitivity. Second, our assessment of sleep duration was based entirely on self-report and assessed at one time point. We also used an abbreviated version of the PSQI, and therefore could not compute a PSQI global score. Future research should use multi-method assessments of sleep, including the administration of the complete PSQI measure, and the inclusion of objective measures of sleep duration such as wrist actigraphy to assess habitual sleep and its relationship to anxiety sensitivity. Third, the sample was composed entirely of people who self-reported a CVD history and who were members of an online panel. Thus, cardiovascular events and medical history were not adjudicated and thus subject to recall bias. The selected sample also poses a threat to the generalizability of the findings. Relatedly, African Americans composed only 9% of the sample, which although comparable to the national prevalence of CHD in African American adults at the time the study were conducted (Go et al., 2013), may have resulted in small cells. Fourth, we did not collect information on current health behaviors such as alcohol use or other drug use such as opiate or cannabis use, all of which are known to impair sleep.

Conclusion

Racial differences in sleep duration are well documented in adults with and without cardiovascular disease, with Blacks obtaining on average between 37–75 minutes of less sleep per night than their non-Hispanic White counterparts (Chen et al., 2015; Lauderdale et al., 2008; Ruiter et al., 2011). However, mechanistic research to understand what accounts for racial differences in sleep duration, which in turn can be used to identify potential intervention targets, is sparse. To our knowledge, this is one of the first empirical studies to provide preliminary evidence that anxiety sensitivity, a cognitive vulnerability, mediates Black-White differences in sleep duration (through an indirect effect), accounting for 29.6% of the total direct effect of race on sleep duration in a sample of adults with cardiovascular disease. If replicated, these findings can be used to further intervention science to reduce racial disparities in sleep outcomes and in cardiovascular health. For example, while studies to extend sleep duration are in their infancy, these findings suggest that anxiety sensitivity, a modifiable or malleable cognitive vulnerability for which there are known and established treatments, may be a reasonable target for intervention with promise for use in cardiovascular disease samples (Keough & Schmidt, 2012; Schmidt et al., 2007; Short, Allan, Raines, & Schmidt, 2015). Future research is needed that moves beyond the documentation of racial disparities in sleep duration to potential behavioral interventions to improve sleep health and cardiometabolic health for all.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Anxiety sensitivity was associated with increased odds of short sleep duration.
- Black vs. White race was associated with increased odds of short sleep duration.
- Blacks had higher mean anxiety sensitivity scores than non-Hispanic Whites.
- Anxiety sensitivity partially mediated Black-White differences in sleep duration.

Figure.
Anxiety sensitivity as a mediator of the relationship between race and sleep duration.

Table 1

Weighted Demographic, Covariate, Exposure, and Outcome Variables Overall and by Race

Variables	Overall (N=1,289)			Black (n=115)			White (n=1174)			X ² or F	p-value
	M/N	SE/%		M/N	SE/%		M/N	SE/%			
Age, y	64.9	(0.36)		58.9	(1.03)		65.6	(0.39)		40.36	<0.001
Female	502	(38.3%)		68	(53.9%)		434	(36.6%)		15.19	<0.001
Male	787	(61.7%)		47	(46.1%)		740	(63.4%)			
High School or Less	212	(16.4%)		18	(16.1%)		194	(16.4%)		19.23	0.001
Some College	526	(41.2%)		68	(57.9%)		458	(39.4%)			
4-Years or More	551	(42.4%)		29	(26.03%)		522	(44.2%)			
Number. of Heart Attacks	1.47	(0.03)		1.51	(0.09)		1.47	(0.03)		0.17	0.684
Depressive Symptoms	5.78	(0.16)		8.60	(0.62)		5.51	(0.17)		8.72	0.003
Anxiety Sensitivity	16.27	(0.38)		26.90	(1.53)		15.23	(0.37)		21.57	<0.001
Sleep Duration	6.56	(0.05)		5.76	(0.18)		6.65	(0.05)		22.38	<0.001
Short Sleep Duration	586	(47.0%)		80	(68.8%)		506	(44.6%)		28.20	<0.001

Note. N is unweighted. Percentages are weighted. Short sleep duration refers to < 7 hours of sleep per night. White refers to Non-Hispanic Whites.

Weighted Logistic Regression Models of Association of Anxiety Sensitivity and Race with Short Sleep (<7 hours) (N=1,289)

Table 2

	Model 1 ¹	Model 2 ²	Model 3 ³	Model 4a ⁴	Model 4b ⁵
	OR 95% CI	OR 95% CI	OR 95% CI	OR 95% CI	OR 95% CI
Anxiety Sensitivity	1.04 (1.03–1.05)	1.04 (1.03–1.05)	1.01 (1.00–1.03)	1.04 (1.03–1.05)	1.04 (1.03–1.05)
Race					
Black	1.84 (1.13–3.00)	1.84 (1.13–3.02)	1.95 (1.19–3.20)	1.83 (1.11–3.00)	1.86 (1.14–3.04)
White	1.00	1.00	1.00	1.00	1.00

OR= Odds Ratios; 95% CI = 95% confidence interval.

¹Model 1 adjusted for age, gender, and education level²Model 2 adjusted for Model 1+ number of heart attacks³Model 3 adjusts for Model 2+ depressive symptoms⁴Model 4a adjusts for Model 3+ lifetime anxiety disorder⁵Model 4b adjusts for Model 3+ region of the country

Table 3

Weighted Linear Regression Models of Association of Anxiety Sensitivity and Race with Sleep Duration (Continuous) (N=1,289)

	Model 1 ¹		Model 2 ²		Model 3 ³		Model 4a ⁴		Model 4b ⁵	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Anxiety	-0.03	****	-0.03	****	-0.01	****	-0.03	****	-0.03	****
Sensitivity	0.00		0.00		0.01		0.00		0.00	
Race										
Black	-0.47	**	-0.47	**	-0.50	**	-0.48	**	-0.47	*
	0.18		0.18		0.18		0.18		0.18	
White	1.00		1.00		1.00		1.00		1.00	

Note:

**** p< 0.0001

*** p<0.001

** p<0.01

* p<0.05

¹Model 1 adjusted for age, gender, and education level

²Model 2 adjusted for Model 1+ number of heart attacks

³Model 3 adjusts for Model 2+ depressive symptoms

⁴Model 4a adjusts for Model 3+ lifetime anxiety disorder

⁵Model 4b adjusts for Model 3+ region of the country